

oral & maxillofacial trauma

fourth edition

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To Marilyn, the love of my life.

Ray

To Cam, Aubrey, and Katharine with love and admiration.

Michael P. Powers

To my loving wife, Claudia, who stands behind me when I need support; in front of me when I need a shield; and always beside me as my best friend, lover, soul-mate, and spiritual guide. TAUIR! To our children: Ryan, Holly, and Kyle, who survived my absences as I dedicated myself to surgery. Thank you. And to R.V. Walker, who mentored and guided Ray Fonseca and others, who in turn, helped me and many others become surgeons in the truest sense of the word. Gig 'Em R.V. You are missed.

David E. Frost

I would like to dedicate this book to my wife, Kymberly; my children, Taylor, David, and Noah; and to my parents, Jesse and Hargrow Barber.

H. Dexter Barber

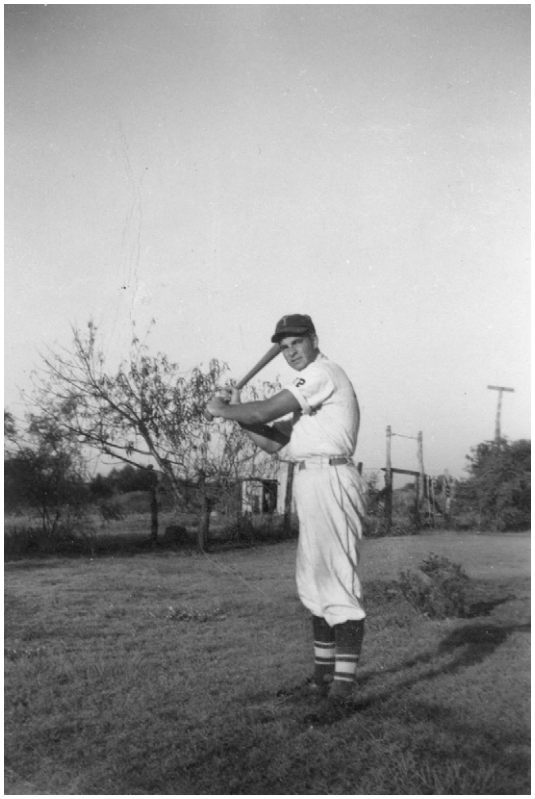
In Memoriam

Dr. Walker, fondly referred to as "R.V.," dedicated his long career to the discipline of oral and maxillofacial surgery and the promotion of the specialty through research, education, administration and service to patients. Robert V. Walker, "Bob" Walker, R.V.Walker, was born September 21, 1924 in Satin, Texas. Son of a hard-working farmer father and an education-oriented mother, R.V. learned skills and attributes that took him off the farm and well beyond. He started college at Texas A&M in 1941, and his athletic abilities earned him a place on the baseball team and a letter.

He won the Best-Drilled Cadet award due to motivation and dedication to the task at hand. When World War II started, R.V., as a member of the Corps of Cadets, was inducted into the military, went through basic training and was sent back to Texas A&M until the army assigned him to Baylor College of Dentistry in 1944. He graduated from dental school in 1947, the same year he married Emily Berger. One year of minor league baseball for the Tulsa Oilers convinced him that dentistry was a more reliable form of employment, so after dental school at Baylor College of Dentistry, he started a general practice in Waco, Texas. Being called to serve in the military once more in the Korean War, he was assigned to Brook Army Medical Center in San Antonio, mostly for his baseball skills. While at San Antonio, he was introduced to oral surgery and facial trauma in a major way and developed his lifelong connection to trauma as a facet of oral and maxillofacial surgery. Dr. Walker completed an oral surgery residency in 1956 at Parkland Memorial Hospital. He joined the UT Southwestern faculty that same year, and 2 years later was named as professor of surgery and chairman of oral and maxillofacial surgery, a position he held until 1984. He remained on the full-time faculty until 1997, when he was named professor emeritus.

He developed one of the top training programs in the country. While shaping a solid curriculum during his early years as chairman, he also worked diligently at the national level. He helped establish essentials for the education and training of oral surgeons across the country and helped create an accreditation system through the Council on Dental Education of the American Dental Association (ADA), the American Board of Oral and Maxillofacial Surgery (ABOMS), and the American Association of Oral and Maxillofacial Surgeons (AAOMS).

His early work with these associations eventually led to the creation of a seat for the ADA on the Board of Commissioners of the Joint Commission on Accreditation of Hospitals. He worked on the committee that helped launch the First International Conference on Oral and Maxillofacial Surgery, which was held at the Royal College of Surgeons in London in 1962. This led to the formation of the International Association of Oral and Maxillofacial Surgeons, of which Dr. Walker was a founding fellow. He served as president of that organization for many years. He also served as president of the American Trauma Society, the AAOMS, the ABOMS, the Texas Division of the American Cancer Society, and the Southwest Society of Oral and Maxillofacial Surgeons. He was chairman of the Oral and Maxillofacial Surgery Foundation, which established the Robert V. Walker Society in his honor in 1997.



Dr. Walker received many awards, including distinguished service awards from the Texas Dental Association (2003), American Trauma Society (1992), and the AAOMS (1981); the William J. Gies Foundation Award in Oral and Maxillofacial Surgery (1976); and the Robert V. Walker Chair in Oral and Maxillofacial Surgery at the University of Texas Southwestern Medical School at Dallas (1992). The annual meeting of the AAOMS was dedicated to him in 1987, and he was elected to the Baylor College of Dentistry Hall of Fame in 1999. He was appointed professor emeritus of oral and maxillofacial surgery at the University of Texas Southwestern Medical School at Dallas in 1997.

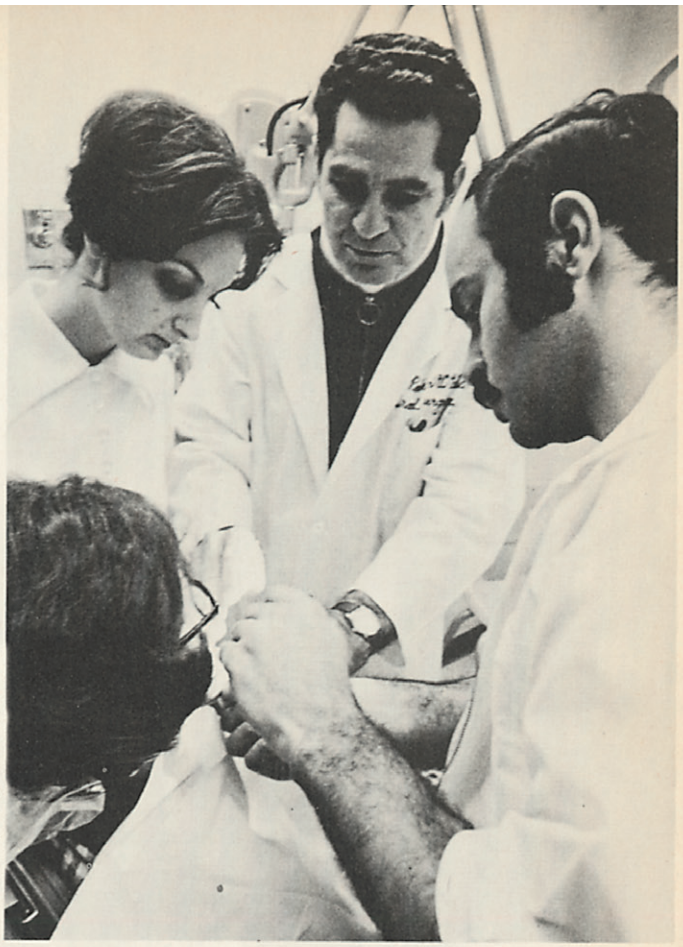
Dr. Walker has received nearly three dozen awards and honors from his peers in dentistry and oral surgery worldwide. Most notably, he was made a fellow of the Royal College of Surgeons of Ireland in 1973 and a fellow of the Royal College of Surgeons of England in 1984, where he gave the prestigious Charles Tomes Lecture. He also served on the Baylor College of Dentistry Oral Health Foundation board of trustees. In September 2012, Dr. Walker was posthumously named Distinguished Alumni of Texas A&M University.

"There are 217 proud alumni of the program, of whom 29 have been or are deans, chairs or program directors in the United States and beyond. Many of our graduates refer to him as the reason for their success, and RV knew each and every one of them by name—where they lived, who they were married to, and what their children were doing. That was the kind of man he was. His contributions to our program and to the field of oral and maxillofacial surgery are immeasurable," said Dr. John Zuniga, current Chair of the program R.V. made internationally recognized.

His leadership, mentorship, and friendship have shaped and guided generations of Oral and Maxillofacial Surgeons and have helped us to treat patients and educate peers throughout the world.



Robert V. Walker
1924–2011



Patient in oral surgery clinic is examined by Dr. Robert Walker, center, and Raymond Fonseca, right, a resident in Dr. Walker's oral surgery program, assisted by staff nurse Gretchen Brown.

The Walker program for advanced education in oral surgery

Robert E. Dunbar, MSJ, Chicago

Oral surgery, as a dental specialty, has become increasingly important in dentistry and in dentistry's role in total patient care. This is reflected in the emergence of several outstanding advanced education programs in oral surgery that have brought national and international preeminence to the profession.

One of the most progressive and innovative programs is directed by Dr. Robert V. Walker, professor of surgery and chairman of the division of oral surgery at the University of Texas Southwestern Medical School in Dallas. He is also president of the American Society of Oral Surgeons.

Dr. Walker has contributed several major innovations to the specialty, including maxillary orthognathic surgery, which was performed for the first time in this country at Parkland Memorial Hospital, the base hospital in Dr. Walker's program. He also produced the first article on this procedure in this country, published in 1963 in the *Journal of Oral Surgery*.

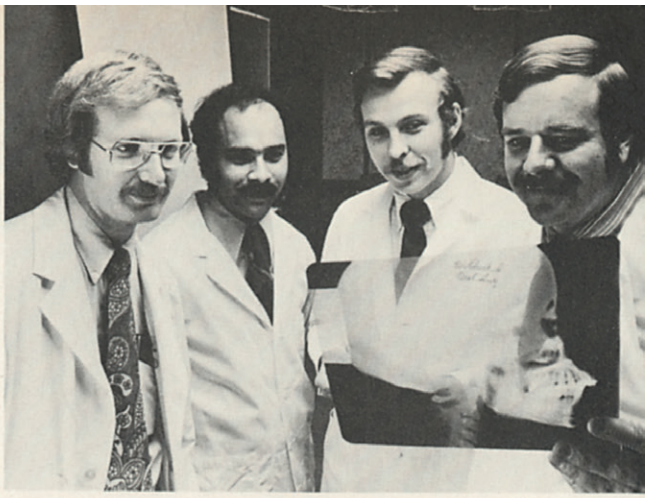
He has earned a reputation for his work in arthroplasty for the ankylosed temporomandibular joint; this procedure has been performed on more than 70 arthritic patients at Parkland thus far. The procedure includes extensive physiotherapy as a follow-up to make certain that the remade joint is functioning well.

Only three or four students are accepted into Dr. Walker's residency program each year, although the number of applicants is usually well over 100. It is a four-year program, and the number of residents at any one time will number up to 16. This past year he accepted four students: two were dental school graduates, and two were senior dental students—Raymond Fonseca of the University of Connecticut and John Dann of Harvard.

Because the final year is completely elective at both schools, both Mr. Fonseca and Mr. Dann are completing their senior year of dental school and the first year of Dr. Walker's residency at the same time. Additionally, Mr. Fonseca is now completing his fifth and final year under the American Fund for Dental Education's



Dr. Robert V. Walker, chairman of the division of oral surgery, University of Texas Southwestern Medical School, Dallas.



Inspecting radiograph are, left to right, Dr. D. Gary Wolford, who heads the oral surgery program at the Veterans Administration Hospital in Dallas, residents Raymond Fonseca and John Dann, and Dr. Bruce N. Epker, director of the oral surgery program at John Peter Smith Hospital, Fort Worth.

Scholarship Program for Disadvantaged Minority Students.

Dr. Walker considered many factors before he made new appointments to his program last year. His standards, which are extremely high, are the central reason why so many students and graduates apply to his program each year.

My philosophy has always been to pick out a guy that I think really has potential to be something else, something different. He doesn't have to be the top man in his class, but if he has demonstrated the intellectual capacity that is needed and has the statesmanlike qualities that are essential for high-level competition in a cauldron of activity in a major health complex, I'll go with that guy.

We didn't take Fonseca and Dann simply to be innovative. They were evaluated on exactly the same basis that I was evaluating people who had graduated from dental school or who had been in practice or in some branch of the armed services. I also looked into their dental school curriculums to see if they were progressive and if they gave them the strengths in basic science education which I think are essential to this program.

Two residents tell of surgery and research

Both Mr. Fonseca and Mr. Dann were exposed to clinical oral surgery soon after their arrival in Dallas last summer. After six weeks of intensive experience in the operating rooms and clinics at Parkland Memorial Hospital, Mr. Dann was assigned a research project. Mr. Fonseca continued his surgical experience until the first of this year. He spent his first three months at John Peter Smith Hospital in nearby Fort Worth and the remaining three months at Parkland.

■ *Fonseca's experience in surgery:* One of the highlights of Mr. Fonseca's experience was a case involving orthognathic surgery on which he was the surgeon. A 16-year-old boy came to the oral surgery clinic complaining of a sore tooth. Mr. Fonseca examined the patient and saw immediately that he had maxillary and mandibular protrusion as well as retrogenia. He recommended reconstructive surgery and was assigned the case.

During an interview in December immediately after the operation was performed, Mr. Fonseca described the procedures involved.

I took cephalometric and periapical radiographs as well as a panoramic radiograph. I then analyzed his case and decided what had to be done. I cut before-and-after models to mimic the surgical procedure and presented them to Dr. William H. Bell, who coordinates our orthognathic surgery at Parkland.

We decided to make mandibular and maxillary extractions of the first premolars on the left side and the second premolars on the right side. We would then move the mandible and maxilla back and the chin forward. The procedures are maxillary-mandibular

ostectomies and osteotomies and a genioplasty. So basically that's what we did. We put him into maxillary fixation with an occlusal wafer.

We'll keep him in the hospital three days and then release him. We'll have him back quite often to check on his progress. We'll also be keeping records, including radiographs and photographs, to follow up on the case and also for future use in research projects.

Mr. Fonseca will follow this case, among many others, during the four years he will be in Dr. Walker's program. He will see the patient more frequently at first to make certain the healing process is successful and also to make sure that the fixation is stable.

Reflecting on the excitement of the moment, Mr. Fonseca made these comments.

I am really thrilled about reconstructive surgery. It's a chance to see a change right before your eyes. A man like Dr. Bell has documented the stability of these procedures, many of them, and will be doing more, and I will be doing research with him along with John Dann.

We have pictures of people needing this surgical correction to show how they appear before surgery, and they look very disheveled. They have buck teeth or their chins are way back, and it affects their personalities. But when they come back after two or three months they're all nicely dressed.

When we show people the before-and-after photos, they ask us if we told the patients to dress up for the "after" photo. We never tell them anything. They don't know their pictures are going to be taken. But their personalities have changed. It's incredible, and I just get a big kick out of this.

■ *Fonseca's research projects:* Since the first of the year, Mr. Fonseca has been working on several research projects with Dr. Bell. One is a sagittal split study based on surgery on rhesus monkeys. They are documenting angiographic studies on the healing of monkeys after surgery and the redistribution of the blood supply after a sagittal split, as well as other variables. About 20 sagittal splits are performed each year throughout the three services at Parkland, John Peter Smith, and the Veterans Administration Hospital in Dallas. Mr. Fonseca commented that they will also be doing a clinical sagittal split study of their patients.

■ *Dann's research:* Mr. Dann, who is now back on the surgical service, broke fresh ground with his initial research project, which he completed earlier this year. The project's title is "Long-Term Analysis of Stability of Segmental Alveolar Surgery and Genioplasty and Analysis of the Associated Soft Tissue Chambers." He has used this research project as the basis for a thesis he has submitted to Harvard.



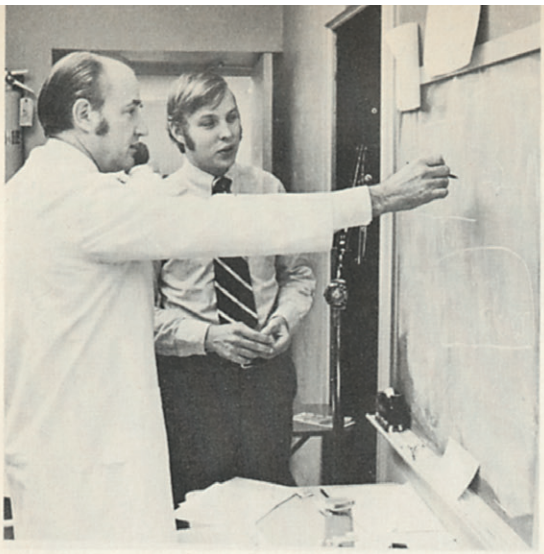
Close-up of Raymond Fonseca in reconstructive surgery at Parkland Memorial Hospital, Dallas. Mr. Fonseca was one of the first recipients of AFDE's dental scholarships for undergraduate disadvantaged minority students.

In this project I have tried to come up with an indicator for the surgeon so he can operate with greater accuracy in this type of surgery. It is hoped that this study will tell the surgeon such things as: If you want to move the teeth in an inch, that means the upper part of the chin is going to come in this far. Or, if you want to build a chin for an individual, you slide the jaw up this far, and this is where the chin will be.

All of my data have been computerized to make the study as accurate as possible.

Orthognathic surgery, which is what this is, is relatively new in this country, and this is one of the centers where they are doing a sufficient number of cases so you can make a fairly extensive study of the results.

There's always a big question when you start doing a new surgical technique. The results may look fine initially, but you may not be sure exactly what you have done then or what the results will look like five years later. In other words, how stable has the procedure been? This is the question I have tried to answer in my research project.



Dr. William H. Bell and John Dann discuss problem in quantitatively analyzing the stability and soft tissue changes associated with maxillary surgery.

■ *Dann's surgery experience:* One of the most memorable surgical experiences Mr. Dann has had thus far involved a 40-year-old man who had been in a serious automobile accident. He had bilateral Le Fort I, II, and III fractures, and his mandible was fractured in five places. Mr. Dann discussed the case.

His whole facial skeleton was disattached from his skull. We got called in on a Sunday evening, and the procedure lasted 16 hours. The problem with this type of case, especially with the mandible in so many pieces, is that you have very few guidelines as to how to put the patient's face back together. It was almost like sewing up the sides of a basketball.

But we were very pleased with the results. We've seen this man several times since and he looks good. He has practically no scars except where part of his chin was ripped off his face.

Structure of oral surgery program

When Dr. Walker came to Southwestern in 1954 as a resident in oral surgery, there was no full-time head of the program or even a division of oral surgery.

Through service in the Korean War, he had become highly competent in managing maxillofacial injuries, and this soon came to the attention of the chief of surgery at the time, Dr. Ben Wilson. Before Dr. Walker completed his residency, Dr. Wilson invited him to join the full-time faculty at the medical school and create a full division of oral surgery within the department of surgery. This was in 1956.

Dr. Walker recalled his initial reaction: "I was absolutely staggered. I really wondered what the place of oral surgery was in a medical school. But I decided to give it a whirl."

In the beginning he relied heavily on Thoma's textbooks on oral surgery, which he referred to constantly whenever a problem arose. Dr. Walker said, "It's surprising what vision and what a wide scope this man had. The program was really designed and arranged around what this man was doing or had done. That's how we started."

He also patterned his program after existing



Resident John Dann and David Mishelevich, director of the medical computer resources center, review computer needs for research project on stability of segmental alveolar surgery and genioplasty.



Raymond Fonseca and Dr. Walker examine patient's nail beds for clinical signs of excessive blood loss.

programs in the medical school. "We had research. We had our very busy clinic. And we had our surgical schedule. At first we only had half a day each week in surgery; now we have three full days a week elective time. That had to come slowly, but we've been very aggressive in terms of scheduling patients. We are accepted fully as an integral part of the hospital surgical staff, and our relations are absolutely superb here."

His initial objective was to produce highly competent oral surgeons to help fill a void that existed then in many cities in the Southwest. Now his overall objective is twofold: to produce top-flight oral surgeons and men who can eventually head oral surgery programs in major institutions throughout the country. To accomplish these objectives, Dr. Walker has evolved a philosophy that is firmly tied to his program.

■ *Importance of staff:* First and foremost, Dr. Walker believes in the importance of a full-time staff. He has four full-time people now. Dr. Bell directs the research activities of the program. Dr. Bruce N. Epker is full-time directorof the program at John Peter Smith Hospital. Dr. D. Gary Wolford, who trained under Dr. Walker, heads the oral surgery program at the Veterans Administration Hospital in Dallas. The fourth full-time staff member is Dr. Jose P. Ampil, a maxillofacial prosthodontist.

Commenting on his staff, Dr. Walker said, "I think the key item in our program is full-time people who have only one purpose for their presence and that's the teaching of advanced education in oral surgery."

In fulfilling this commitment, the staff gives trainees in the program as much responsibility as they can handle. This is also basic to Dr. Walker's philosophy. He said:

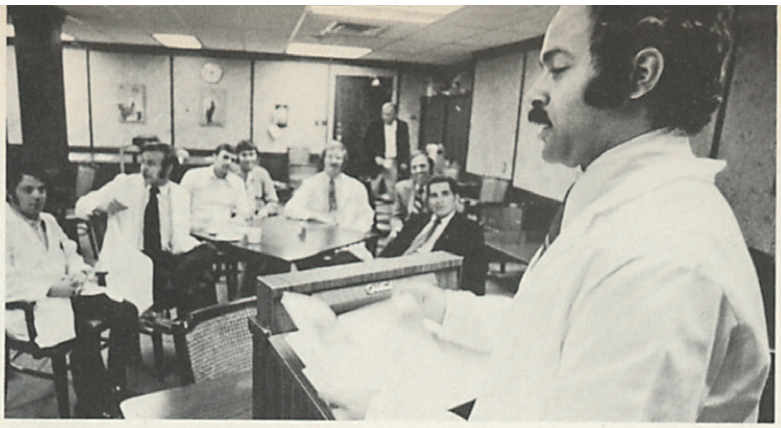
> When a trainee has had enormous responsibilities in a lot of different areas in the scope of oral surgery —decision-making, scheduling, performing very sophisticated surgery—he is less apt to narrow his professional scope when he leaves the program than he would be if he were oversupervised. It also builds confidence. Responsibilities are given to them for that reason, more than they like sometimes, but we feel it is absolutely essential for our trainees."

Effective communication, both verbal and written, is also essential to the program's objectives. Dr. Walker commented, "One of the biggest deficiencies we find in our people is that they are not forensically able, nor can they write well. This is a reflection of education far earlier than we can provide it."

Dr. Walker's trainees are encouraged to prepare formal papers suitable for publication, either individually or in collaboration with other members of the program. In this way they can begin to learn the art of scientific writing and also make contributions to the scope of knowledge in oral surgery.

■ *Conferences and literature reviews for residents:* The program includes a series of Wednesday and Saturday conferences throughout the year. Six major areas are covered: physical diagnosis, maxillofacial trauma, maxillofacial orthopedic reconstruction, surgical pathology, essentials of patient care, and myofascial and odontogenic considerations. Guest lecturers are often invited to the conferences.

A Journal Club program gives every trainee frequent opportunity to review the current oral



Bone graft and continuity defects were discussed by Raymond Fonseca at a recent session of the Journal Club.

surgery literature and to discuss it at the weekly meetings. Mr. Fonseca described one particular study in which he was involved.

One of my Journal studies was on bone graft and continuity defects. I looked through more than 80 articles on bone grafting in recent and pertinent literature before the meeting. From these 80 articles I selected 20 that were the most relevant and the best done and presented them at the meeting.

I criticized them constructively, agreeing with some things and disagreeing with others. Then everybody at the meeting, including Dr. Walker, his staff, and the other residents, joined in the discussion. These are very informal meetings, really. That's where we pick up what's being done in oral surgery, at these Journal Club meetings and the weekly conferences.

■ *Research comes early:* Dr. Walker believes in beginning the program with research as well as surgical experience; therefore, in the first two years of the program, his residents complete one year of research and one year on the surgical service.

The advantage of beginning with research is that the boy still has the carrot before him. He can fulfill his research requirements knowing that he still has two full years of surgery to come. This helps him to settle down in a more disciplined way in the laboratory. You can't mix the two together very well if you have a busy clinical service. You can't do part clinical and part research at the same time, so we keep these experiences separated.

Extent of surgical experience

Dr. Walker's primary objective is to produce a man who will be competent in managing the many problems attendant to the practice of oral surgery. Dr. Walker describes him as "an unflappable person who will be able to manage the most excruciating problems under almost any circumstances in a hospital or in an office." Because this man should also be able to direct a program in an institution, he is exposed to research disciplines as well as the demands of a clinical program. Dr. Walker described the extent of surgical training that a man will have at the end of his residency.

"He will be able to take care of the usual dental-alveolar surgery, which includes extractions, soft tissue surgery within the oral cavity, removal of impactions, preparation of ridges for prosthetics, and the usual intraoral procedures attendant to oral surgery," Dr. Walker said. "In addition, he's going to be able to take care of almost any type of maxillofacial injury in terms of bone injury, from the frontal bone down to the mandible."

Since 1956 the division of oral surgery has been responsible for the care of maxillofacial injuries, and it still has primary responsibility for the management of these injuries.

A resident in Dr. Walker's program will be able to take care of the usual odontogenic lesions that occur about the maxilla or the mandible, the benign tumors that occur in and about the oral cavity. He's a participant in the care of cancer patients—especially the reconstructive phase of it. Dr. Walker remarked, "This is one facet of the program that he's directly involved in at John Peter Smith Hospital. Under the direction of Dr. Bruce Epker he will have primary responsibility for caring for many of the malignancies that occur about the oral cavity. This is not to say that we are teaching this at all. But at least the young man has exposure to it."

The resident will be quite capable in orthognathic surgery. According to Dr. Walker, during the past 12 years there has been an enormous thrust forward in the care of patients with dental-alveolar plus skeletal deformities. These patients have severe orthodontic problems. The resident will be able to diagnose precisely where the deformity lies—whether it's within the dental al-



Dr. Stuart N. Kline, professor of oral surgery, University of Miami, Fla, School of Medicine, lectured on the use of bone grafts in managing deformities of the mandible at a recent Saturday morning conference.

veola, or in the skeletal area where the bone has to be manipulated to produce a better appearance.

"He will have a great capability in the care of many temporomandibular joint problems," Dr. Walker commented, "including intricate surgery in and about the temporomandibular joint and the usual infections of odontogenic origin. The young man will have great competence in the incision and drainage of the facial planes in and about the floor of the mouth. He will be able to manage a wide range of lesions that originate in the salivary gland and in the maxillary gland area."

He will also have extensive experience in preprosthetic surgery, involving the extensions of the vestibules, the lowering of the floor of the mouth, and skin grafting in the oral cavity to produce better ridges for the prosthodontist to work with in producing dentures.

Dr. Walker added, "He'll be quite capable in reconstruction from the standpoint of bone grafting, where there are defects of the jaws, either of congenital or acquired origin, for example, as a result of tumor surgery or secondary to infection. There are many defects of the face and jaws that require bone grafting."

Rotations in allied areas

■ *Required rotations:* Dr. Walker's program includes three required rotations. All trainees participate in the general anesthesia service at Parkland for four months. They spend three months on the internal medicine service at John Peter Smith and two months on pathology at the Dallas Veterans Administration Hospital. All of these rotations include daily afternoon lectures.

We want our trainees to be well-disciplined in the specialty of anesthesia. We give them full responsibility, and in this way they learn an enormous amount of cardio-pulmonary physiology, pharmacology, and resuscitation.

How in heaven's name do you resuscitate a patient who's had a cardiac arrest or get a tube into a patient if his airway is obstructed after an accident? Those are some of the reasons I want our trainees to have a thorough exposure to anesthesia, because these are some of the things they are going to have to do."

The internal medicine rotation is taken at John Peter Smith Hospital, which is oriented to the general practitioner in medicine. Here the resident becomes familiar with patients who have metabolic and medical diseases. The service is so structured that Dr. Walker's residents are given full responsibility for care. Dr. Walker said, "It's actually like a medical residency. They learn a great deal in this tenure in terms of taking care of sick patients."

During the rotation in pathology the resident learns the histology of lesions of the oral cavity. He learns how to recognize lesions from a clinical, radiographic, and histopathologic standpoint, especially lesions that are unique to the oral cavity. The resident not only becomes familiar with odontogenic lesions but with lesions of salivary gland or mucosal origin.

■ *Elective rotations:* In addition, residents also have an elective rotation that they can take in their last year in the program. They may elect to participate in the rotation on otolaryngology or on plastic surgery to learn some of the dis-

ciplines that are part of those particular services. Or they may opt for the rotation in the infectious disease service at Parkland, which is one of the busiest services, so they can learn more about the microbiology of disease and antibiotics. Some residents have elected the cardiology service at Methodist Hospital, an affiliate of the medical school. Whatever their interests, opportunities for intensive learning experiences are available in almost every medical specialty.

Dr. Walker's program includes 12 months of research, 12 months on required and elective rotations, and 24 months of clinical experience at the three affiliated teaching hospitals. His residents also participate in pediatric oral surgery at Children's Medical Center which is adjacent to Parkland. In all, his residents participate in approximately 1,500 elective and emergency surgical cases each year at all four hospitals.

Responsibilities of the senior resident

In each year of the program three or four senior residents spend at least three months as chief residents at all three oral surgery services. This is the program's final "maturing process" for residents. Each chief resident is administratively in charge of that particular service. Dr. Walker explained that the chief residents are directly responsible for the performance of the first- and second-year men. "If a trainee is not performing well, the chief resident has got to tell me why. He's my direct line of communication to the program. It doesn't mean that we're not counseling and talking to every man in the program, but the one who has to face the buck when things are not going well is the chief resident."

The chief resident in Dr. Walker's program decides which trainee is going to handle emergency trauma cases. He also has responsibility for the monthly schedule of call so that the oral surgery service is covered day and night at all three hospitals.

"There are almost 200,000 emergency admissions at Parkland alone each year," Dr. Walker remarked. "There's hardly a night when our people are not involved with patients. It may be someone with an abscessed tooth that needs to be incised and drained. It may be a patient with severe facial injuries from an auto accident. It could be any number of things."

Each trainee is provided with a booklet that describes the protocol of the program and each resident's responsibilities. But the assignments of the year for the Journal Club, the weekly conferences, the medical rotations, and the clinical service are made by third-year residents who will be the chief residents during the next year. Their decisions are reviewed and discussed with Dr. Walker and his staff before final approval is given.

Results of the program

Reflecting on the results of the program, its philosophy, and its major objectives, Dr. Walker believes that his goals are being accomplished. He referred to a report he prepared during the past year for an evaluation by the ADA Council on Dental Education. Of the nine men who completed their training during the past three years, four are in private practice and five are in institutions. This includes Dr. Wolford who heads the oral surgery program at Dallas Veterans Administration Hospital, and full-time staff members at the University of Connecticut in Hartford, the University of Florida in Gainesville, the University of Washington in Seattle, and University Hospitals in Jacksonville, Florida. More than half elected to go into institutions rather than private practice.

Dr. Walker's approach to advanced education in oral surgery is innovative and progressive in many respects. His emphasis on developing highly competent oral surgeons who will have the maturity and ability to head oral surgery programs of their own represents a major contribution to the specialty.

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Preface

This text represents the fourth edition of *Oral and Maxillofacial Trauma*. Our goal is still to provide the reader with a comprehensive text on the subject of oral and maxillofacial trauma. This field is evolving, and a fourth edition is required to update those areas where the body of knowledge has changed or where new areas are needed to be included. We realize that there will always be some area that will not be addressed as thoroughly as some readers would like, but we have tried to be as comprehensive as possible. We apologize in advance for any areas that we did not cover as thoroughly as our readers would have liked.

Our over-arching concept is that the best care of the traumatically injured patient is through the well-informed and educated trauma team. Many patients present with injuries to various organs. It is incumbent on the individual who is managing these patients to understand, recognize, and triage their injuries appropriately. Hopefully, our fourth edition continues to build our efforts in this regard.

Our chapter contributors have again done a remarkable job. Their expertise is exhibited by the excellent coverage of their assigned topics. Authors new to the fourth edition have written half of the chapters. This adds a new perspective to the material covered. Much of what made the first three editions successful has been retained in the fourth edition. The text remains clinically relevant and useful for both the resident and the practicing clinician.

The text maintains the basic format of the first three editions. We start with **Part I:** *Basic Principles in the Management of Traumatic Injury*. In this section the metabolic response to trauma, surgical nutrition, and healing of the traumatic wound are discussed. This is an imperative pre-requisite to understanding how to care for the traumatically injured patient.

Part II: *Systematic Evaluation of the Traumatized Patient* follows the ATLS principles of evaluating the trauma victim. There are chapters on emergency and intensive care, airway management, non-penetrating chest trauma, and shock.

Part III: *Management of Head and Neck Injuries* is written by authors who are unquestionably the most knowledgeable in their field. The chapter on applied surgical anatomy remains unchanged. We feel that this chapter is "state of the art." The next three chapters are essential reading to develop expertise in clinical and radiographic evaluation of traumatic head and neck injuries. The remainder of this part covers traumatic injuries to specific areas of the head and neck. Three chapters on the diagnosis and treatment of dentoalveolar, mandibular, zygomatic complex, midface, orbital, frontal sinus, and nasal fractures. The management of soft tissue injuries, including human and animal bites and salivary gland and nerve trauma is included in this part.

Part IV: *Special Consideration in the Management of Traumatic Injuries* covers topics that are essential if one is to have an understanding of the comprehensive management of the traumatized patient. Topics such as firearm and burn injuries are discussed in detail. The management of the growing and geriatric patient and the specific considerations for these patients are presented.

The book has numerous new color illustrations and clinical photographs. The artwork is excellent and helps the reader grasp the anatomical and surgical details.

Lastly, this edition is dedicated to the outstanding career and contributions of Dr. Robert V. Walker.

Acknowledgments

The fourth edition of *Oral and Maxillofacial Trauma* represents the work of many dedicated individuals. The contributors to each chapter have donated their expertise and time to create a comprehensive text. This endeavor was truly the result of teamwork and it demonstrates what can be accomplished when people work together. We cannot begin to thank all of the expert contributors for their efforts. Perhaps the greatest reward is the possibility that through this text we have improved the quality of the traumatically injured victim.

We also would like to acknowledge the work of previous contributors to the first three editions of the text, for much of their work was the foundation for many chapters: Ramin Bahram, Hans Bosker, Robert J.I. Bosker, Mark A. Cesta, Kelly R. Cottrell, Robert S. Glickman, Richard Haug, Vasiliki Karlis, Faisal A. Quereshy, Cyrus A. Ramsey, Fonda G. Robinson, Keith E. Silverstein, and Michael D. Turner.

Additionally, many residents have contributed to this edition as they did in the previous editions. They have stimulated us to revise and update this text and have provided us with inspiration and friendship. For this and much more we extend our thanks.

Our thanks are also extended to the individuals at Elsevier who worked closely with us to help us accomplish our goal. Executive Content Strategist, John Dolan, Senior Content Development Strategist, Brian Loehr, and Senior Project Manager, Marquita Parker, are equally contributors to this text and without them this book would not have been written.

PART ONE

Principles in the Management of Traumatic Injuries

CHAPTER 1

Metabolic Response to Trauma

Thomas A. Stark | Harry L. Anderson, III

OUTLINE

Physiologic Response
Mediators of the Response
Neuroendocrine Response
Lipid-Derived Mediators
Cytokines
Polymorphonuclear Neutrophils
Clinical Implications
Modulation of the Response
Adult Respiratory Distress Syndrome
Nutrition As Therapy
Deep Vein Thrombosis Prophylaxis
Stress Gastritis

Injury produces profound systemic effects. Hormones, the autonomic nervous system, and cytokines all produce a series of responses that are teleologically designed to help defend the body against the insult of trauma and promote healing. Classically, these responses have been described as the *stress response*, a term coined by the Scottish chemist Cuthbertson in 1932.[1] However, some of these responses may be counterproductive. The cascade of interactions is orchestrated in the severely traumatized patient to produce a host of responses that follow a recognizable pattern, but the depth and duration of these changes are variable, usually proportional to the extent of the injury and the presence of ongoing stimulation. Stresses other than major trauma produce alterations in the metabolic responses; examples are burns, sepsis, and starvation. Each results in marked variations in the metabolic response, and this variability persists during the later chronic and recovery phases of the original injury (Fig. 1-1).

The body's initial response to insult (the acute phase) is directed at maintaining adequate substrate delivery to the vital organs, in particular oxygen and energy. Cuthbertson's pioneering work recognized the increases in basal temperature, energy expenditure, and oxygen consumption, and also the loss of potassium and nitrogen.[2,3] The term *systemic inflammatory response syndrome* (SIRS) is used to describe the body's response to infectious and noninfectious causes and consists of two or more of the following—hyperthermia or hypothermia, leukocytosis or leukopenia, tachycardia, and tachypnea. When the inflammatory response impairs function of organs or organ systems, the term *multiple organ dysfunction syndrome* (MODS) is used. As greater sophistication in the care of the multiply injured patient has permitted careful observation and analysis of the metabolic changes that accompany trauma; similar advances in the field of molecular biology have allowed the identification and measurement of the precise hormones and inflammatory mediators involved in the body's response to trauma. This chapter reviews the mechanisms and consequences of the metabolic response to traumatic injury and some common approaches to the problems produced by these metabolic derangements.

PHYSIOLOGIC RESPONSE

Tissue damage produces an inflammatory reaction that causes local effects, such as tissue edema, vasoconstriction, and thrombosis. Other mediators released into the systemic circulation act at sites removed from the injury. For example, they stimulate the autonomic nervous system, with concomitant production of hormones, cytokines, and arachidonic acid metabolites. The orchestrated response seen with severe injury has been described as having two phases that overlap, the ebb phase, which occurs immediately and may last as long as 24 hours after injury, and the flow phase, which may last for weeks.

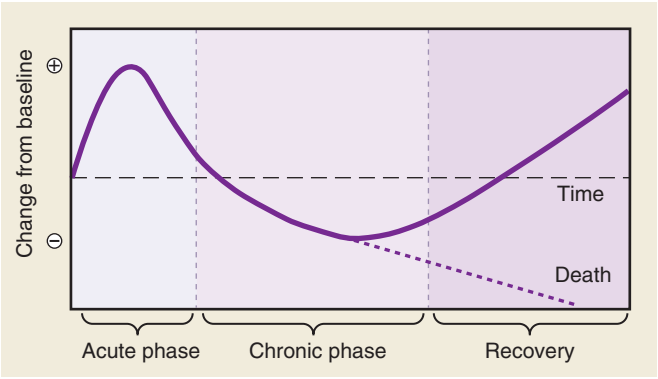


FIGURE 1-1 Phases of metabolism after critical illness and injury. (Adapted from Mongardon N, Singer M: The evolutionary role of nutrition and metabolic support in critical illness. *Crit Care Clin* 26:443–450, 2010.)

The first, or ebb, phase is characterized by the release of catecholamines and vasoactive hormones. Cardiac output is elevated by increases in heart rate and cardiac contractility. Peripheral and splanchnic vasoconstriction occurs and extravascular fluids are mobilized to maintain blood volume. These hemodynamic consequences act to preserve blood volume. The respiratory rate is also increased and this increase, in concert with the hemodynamic changes, drives maximal oxygen delivery.

The blood glucose level is also elevated through a complex and tissue-specific insulin resistance pathway.[4] The maintenance of blood glucose levels supports the obligate glucose-metabolizing tissues—brain, bone marrow, erythrocytes, granulation tissue, and the immune system. Free fatty acids are mobilized by the catabolic hormones and become the main source of energy for peripheral tissue. Stored hepatic glycogen is limited and may be depleted within 1 day.

As the catabolic or flow phase is established, the metabolic emphasis is on the provision of substrate for healing. The basal metabolic rate is elevated, and thereafter the patient catabolizes muscle to make glucose. Birkhahn and colleagues found an almost 80% rise in catabolism after skeletal trauma, and Kien and associates have reported elevations as high as 103% in burn patients.[5,6] The total body nitrogen balance becomes negative. Some of the earlier changes of the ebb phase may be reversed; for example, a diuresis may occur, or the heart rate may slow.

It may be difficult to separate the effects of the metabolic response to trauma from the effects of shock or incomplete resuscitation, but clearly such a distinction is necessary. The initial phase of the trauma response is complicated by an ongoing deficiency in tissue perfusion. The clinician must respond to dynamic changes in physiology to make time-sensitive interventions. The pulmonary artery or Swan-Ganz catheter offers a continuous window into the minute to minute mechanics of the severely injured patient. More recently, the safety and usefulness of Swan-Ganz catheters in trauma resuscitation has been scrutinized and their routine use has declined.[7] Less invasive modalities such as the central venous catheter, near-infrared spectroscopy (NIRS), and continuous cardiac output monitoring by arterial waveform analysis are as effective at guiding therapy.[8-10]

The measured serum lactate level may help guide therapy and additional resuscitation. During shock states, tissue perfusion is decreased and increasing numbers of cells depend on anaerobic metabolism for energy. Normalization of serum lactate levels can indicate a return to global aerobic metabolic states and serve as an end point for resuscitation. Partly because of the reversible nature of hypoperfusion in the trauma setting, initial serum lactate levels are not themselves independent predictors of morbidity and mortality.[11]

Similar injuries to different body regions can produce dramatic differences in metabolic requirement (Fig. 1-2). Two injuries in particular are worth special mention because of their notable metabolic consequences. The first is injury caused by head trauma. When brain injury is superimposed on multisystem trauma, the risk of death more than doubles.[12] Increased mortality may not be directly caused by neurologic injury per se, but rather by the systemic consequences of deranged metabolism.[13] In addition, many methods whereby brain-injured patients are managed after injury have themselves profound effects on metabolism. Examples of these interventions include steroids, systemic dehydration, muscle paralysis, hyperventilation, hypothermia, hyperbaric oxygen therapy, and barbiturate coma. The effectiveness of these treatments has been challenged but they continue to remain in use.[14-16]

The second injury is the clinical entity of classic fat embolism syndrome, seen particularly after long bone fractures. Fatty acid and triglyceride-rich fat globules enter venous sinusoids at the fracture site. These fat droplets migrate to the lung, where they are converted by pulmonary lipase to free fatty acids, which can damage the pulmonary microvasculature. In addition, platelets are activated, aggregate, and attach to the fat droplet, a process that may lead to a consumptive coagulopathy.

MEDIATORS OF THE RESPONSE

NEUROENDOCRINE RESPONSE

An early response of the neuroendocrine system is the upregulation of the sympathoadrenal axis, which generates high plasma concentrations of epinephrine, norepinephrine, vasopressin, and dopamine. The peak and duration of the response parallel the severity of the trauma.[17] These substances directly affect blood glucose levels and also produce inhibition of glucose uptake by tissue, which stimulates glucagon secretion. Sympathetic activity promotes lipolysis within adipose tissue, which begins to provide an energy source for gluconeogenesis.[18] Gluconeogenesis in the liver is stimulated by glucagon.

The hypothalamic-pituitary axis is stimulated at the same time as the sympathetic nervous system after trauma.[19] The pituitary releases adrenocorticotropic hormone (corticotropin, or ACTH), which stimulates the adrenal cortex to secrete glucocorticoid hormones such as cortisol and aldosterone. Epinephrine and

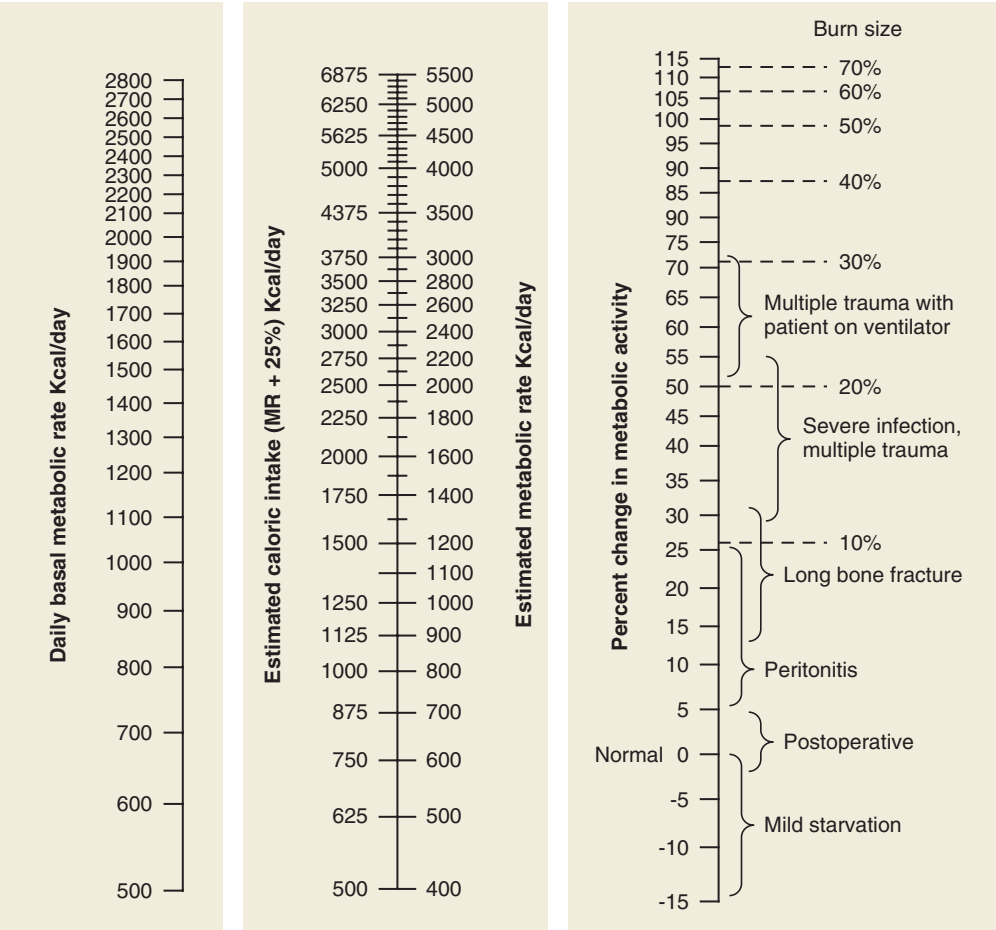


FIGURE 1-2 Changes in resting energy expenditure associated with trauma, burns, and other common clinical conditions. (Adapted From Wilmore DW: *The metabolic management of the critically ill*, New York, 1977, Plenum Press.)

cortisol promote muscle breakdown, protein catabolism, and amino acid release.

The effects of the flow phase of the metabolic response to trauma are partly attributable to hormones such as glucagon and cortisol, but not entirely, because the catabolic consequences extend beyond measurable elevated levels of these hormones.[20,21] This finding has implicated other factors such as cytokines or the suppression of other hormonal axes such as those of somatostatin and growth hormone.

LIPID-DERIVED MEDIATORS

Cyclooxygenase products of arachidonic acid metabolism are present in increased amounts in human studies of injury. Thromboxane A2 accentuates neutrophil aggregation and, with prostacyclin, has potent and opposing vascular effects that may have a role in pulmonary hypoxic vasoconstriction and systemic vasodilation. Lipoxygenase products are also released in large quantities and affect the permeability of the pulmonary vascular bed.

Platelet-activating factor (PAF) is a phospholipid metabolite released by a number of cells, including neutrophils. The response to PAF at the endothelial surface results in enhanced superoxide production, enhanced platelet aggregation, altered pulmonary vascular reactivity, and changes in endothelial permeability.

CYTOKINES

Protein mediators, collectively called cytokines, are produced at the site of injury and by diverse circulating immune cells. Monocytes, lymphocytes, macrophages, and other cells release cytokines. They can act locally as paracrines by way of direct cell to cell communication or systemically when produced in excess by way of endocrine mechanisms. The most important cytokines in trauma are tumor necrosis factor (TNF), the interleukins (IL-1, IL-2, IL-6, and IL-8), the interferons, and various growth factors such as granulocyte-macrophage colony-stimulating factor (GM-CSF), and platelet-derived growth factors (PDGFs). They enhance immune cell function and are responsible for the systemic effects of inflammation and sepsis, such as fever, leukocytosis, hypotension, delayed gastric emptying, and malaise.

Thought to be the most proximal mediator of the inflammatory response, TNF was originally described as the catabolic factor cachectin.[22] At least two forms of TNF exist.[23,24] TNF influences cellular attraction as part of the local inflammatory response, leukocyte migration, and systemic hypotension.[25,26] It also promotes muscle

catabolism, free fatty acid release, and hepatic synthesis of acute-phase reactants. TNF free receptor is a glycosylated protein found in membrane-bound and free-floating forms. In contrast to IL-1, TNF appears to act peripherally and has no direct effect on lymphocyte activation.[27]

The interleukins are polypeptides released from lymphocytes; each is numbered according to the amino acid sequence that elicits its action.[28] Circulating free receptors are known for IL-1 and IL-6. Free receptors may exist for all cytokines. They appear to function in the regulation of cytokine activity. IL-1, which can be detected in the circulation within a few hours after injury, has multiple biologic effects, including the activation of resting T lymphocytes and macrophages, induction of hematopoietic growth factors, stimulation of chemotaxis of neutrophils, and synthesis of collagen and collagenases. More profound systemic effects include fever and changes in protein metabolism. Originally described as pyrexin, IL-1 was shown in subsequent studies to act via the production of prostaglandins in the hypothalamus and to alter the set point of the thermoregulator in the hypothalamus.[29,30] It is in this way that antiprostaglandin agents, such as aspirin, can block the fever effect of IL-1.[31] A byproduct of IL-1 metabolism can increase proteolysis of human muscle and induce hepatic protein synthesis.[32,33] Identified as a B cell differentiation factor, IL-6 is now recognized as the final common mediator in a cascade of cytokine activity that alters hepatocyte protein synthesis.[34,35]

The interactions of cytokines with one another and the stress response hormones have been increasingly studied. Cytokines are potent stimulators of the release of other mediators (e.g., IL-1 acting on the hypothalamus). Evidence exists for convoluted positive-feedback relationships organized within the cytokine cascade. Release of early mediators, for example TNF, triggers release of the complete aggregate of cytokines, which then combine to elicit the host response.

The cytokine receptors and cytokine agonists may have several roles in trauma patients. Both are released in a sequential manner, paralleling the release of cytokines, and they modulate the body's response to trauma.[36] Figure 1-3 demonstrates the fluctuating levels of cytokine receptors over time. Quantitative serum cytokine levels, particularly IL-6, correlate with injury severity and serve as a predictor of adverse outcomes and mortality.[37,38] Although highly sensitive, lack of specificity has limited their use in clinical settings.

POLYMORPHONUCLEAR NEUTROPHILS

Catecholamines and glucocorticoids marginalize peripheral polymorphonuclear neutrophils (PMNs) and recruit them from the bone marrow.[39,40] Lipids and cytokines (e.g., IL-1, TNF, PAF) then prime these cells for enhanced superoxide anion release and sequestration in end-organs.[41] Capillary endothelial integrity is disrupted, leading to the formation of edema, defects in oxygen delivery, hypoxic cellular injury, and other adverse consequences for cellular homeostasis. This derangement leads to the clinical entity of multiple-organ failure. Human studies have supported this sequence and also suggested that additional stress results in a rerelease of mediators of the stress response that multiplies the effect of the already enhanced PMNs.[42,43]

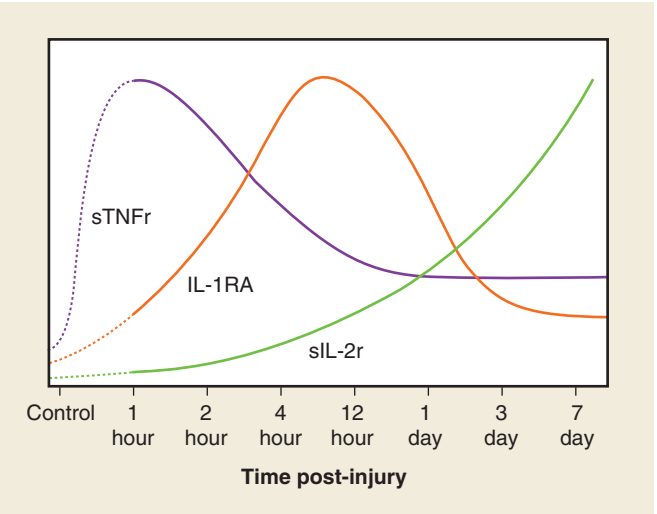


FIGURE 1-3 The sequential release of cytokine receptors and receptor agonists after injury. *IL-1RA,* interleukin-1 receptor antagonist; *sIL-2r,* soluble interleukin-2 receptor; *sTNFr,* soluble TNF receptor. (Adapted from Cinat M, Waxman K, Vaziri ND, et al: Soluble cytokine receptors and receptor antagonists are sequentially released after trauma. *J Trauma* 39:112–118,1995.)

CLINICAL IMPLICATIONS

The successful management of the metabolic changes that accompany severe trauma influences and may prevent some of the major complications of trauma—namely impaired immune function, multiple organ failure, and sepsis.[44]

MODULATION OF THE RESPONSE

Researchers have tested novel therapeutic strategies and options aimed at selectively inhibiting the undesirable actions of cytokines while allowing the appropriate responses to be expressed. Some effects of cytokines on target tissue have been successfully blocked by the use of anticytokine antibodies and specific cytokine receptor antagonists. Animal studies, in particular, have suggested significant efficacy of these agents, but the results in humans have been largely disappointing, particularly when they have been used in septic patients.[45] Most of the work, however, has focused on multicenter trials of patients who have sepsis from many causes, rather than solely the multiply injured. Caution must nonetheless be exercised because of the risk that these agents may neutralize the beneficial, survival-enhancing effects of the cytokines and other elements of inflammation. Another problem with these therapies is that cytokines are rapidly released after injury, and the administration of antibodies, by necessity, occurs later—after the initiation of the cytokine cascade.

Pharmacologic manipulation of the end-organ response to stress is also accomplished with some drug classes that act on specific mediators of the response. For example, cyclooxygenase inhibitors such as ibuprofen

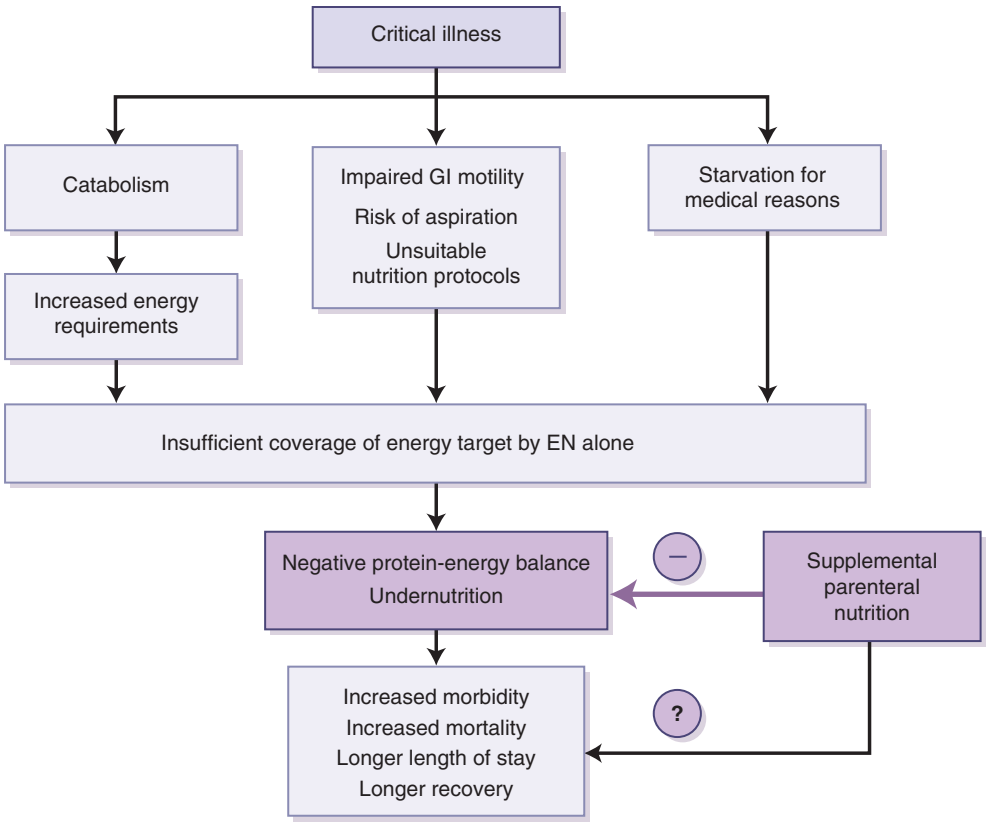


FIGURE 1-4 Factors whereby enteral nutrition may result in undernutrition of critically ill and injured patients—the potential role of supplemental parenteral nutrition. (Adapted from Thibault R, Pichard C: Parenteral nutrition in critical illness: Can it safely improve outcomes? *Crit Care Clin* 26:467–480, 2010.)

inhibit the production of eicosanoids and may thus blunt the physiologic response to cytokines, such as fever, associated with TNF, IL-1, and IL-6. In patients with sepsis, ibuprofen has shown some improvement in clinical parameters, but has not been proven to decrease the duration of shock or improve mortality.[46]

Control of hyperglycemia in critically ill surgical patients has been shown in a large, prospective, randomized trial to decrease morbidity and mortality. Intensive insulin therapy (IIT) requires maintenance of blood glucose levels below 110 mg/dL.[47] Subsequent analysis found that increased mortality from hypoglycemic events negates the benefits of IIT in clinical practice. Trauma patients, however, were a subset found to having benefited the most from IIT.[48] Further investigation is necessary to determine safe and effective mechanisms for glycemic control in trauma patients.

The role of glucocorticoids in modulating the stress response remains unclear. In severe cases of injury, sepsis, and critical illness, the adrenal system is unable to supply the overwhelming demand for glucocorticoids, and a relative adrenal insufficiency ensues.[49] Pharmacologic factors such as even a single dose of etomidate have also proven to increase rates of adrenal insufficiency and mortality in the critically ill.[50] Multiple trials have failed to identify a definite improvement in mortality, although low-dose corticosteroid therapy may decrease the duration of shock states and improve short-term survival.[51-53] In trauma patients, there is some evidence that hydrocortisone therapy attenuates the stress response and decreases the likelihood of hospital-acquired pneumonia.[54] Further research is needed to establish practical therapeutic strategies, particularly in traumatic brain injury, in which high-dose steroids have been associated with an increase in mortality.[55]

Human activated protein C (drotrecogin alfa [activated]) was one of the first approved recombinant agents targeting the procoagulant and generalized inflammatory response that occurs during sepsis. It had been initially found to reduce death rates in patients with severe sepsis.[56] Ongoing surveillance proved that there was no survival benefit in patients with severe sepsis when compared with placebo, and the drug has since been withdrawn from the market.[57]

Pharmacologic manipulation of the response to traumatic injury has been met with limited success. Research continues to attempt to identify agents that protect the patient from the deleterious effects of the host response. Knowing which patient may benefit from a particular medication may be a function of that individual's unique DNA. Current studies have identified specific genetic polymorphisms that are predictors of adverse outcomes in severe trauma and sepsis.[58] Future investigation may help develop individually tailored treatments.

ADULT RESPIRATORY DISTRESS SYNDROME

The adult respiratory distress syndrome (ARDS) is an acute illness characterized by noncardiogenic pulmonary

edema. This refractory hypoxemia arises in part as a consequence of lung inflammation secondary to the mediators of the acute response to trauma. Damage to the alveolar-capillary interface results in intrapulmonary shunting of blood, raised pulmonary vascular pressures, and surfactant depletion.

The syndrome is primarily treated by mechanical ventilation, and the National Institutes of Health Acute Respiratory Distress Syndrome Network has identified that low tidal volume ventilation (6 mL/kg predicted body weight) was superior to using traditional tidal volumes (12 mL/kg of predicted body weight) in treating hypoxemia.[59] When therapy fails to keep pace with progressive lung dysfunction, alternative therapies—such as high-frequency oscillatory ventilation, prone positioning, and extracorporeal life support (ECLS) or extracorporeal membrane oxygenation (ECMO)—may be indicated.[60-63]

NUTRITION AS THERAPY

The advantages of enteral nutrition over parenteral nutrition have been clearly demonstrated, and the gastrointestinal tract should be used whenever possible. Recently, a role for supplemental parenteral nutrition has been advocated (Fig. 1-4). The traditional preference is to feed patients by the enteral route for reasons that include a reduction of the number of enteric organisms that may be responsible for bacterial translocation. Stimulation of the enterocyte brush border and gut-associated lymphoid tissue is an important protective mechanism against the proliferation of the offending organisms.[64] The route of feeding may also have an impact on the production of cytokines after injury; thus, use of the enteral route may confer an additional advantage.[65]

Considerable attention has focused on nutrients that attenuate the metabolic response to injury. Nutrients that appear to enhance the immune system include arginine, glutamine, and nucleic acids. The immune system may be enhanced by altering the relative amounts of omega-6 versus omega-3 unsaturated fatty acids.[66,67] Other nutrients may act as oxidants, preventing damage by free radicals, such as the common antioxidants vitamins A, C, E, and the trace element selenium.

There has been lukewarm interest in the concept of "immunonutrition"to ameliorate the end-organ damage from critical illness and sepsis, which may later result in acute renal failure and ARDS. A study of supplementation with an enteral diet of omega-3 fatty acid, gamma-linolenic acid, and antioxidants versus an isocaloric enteral formulation was reported in 2011. These nutrients are typically thought to modulate the systemic inflammatory response.[68] The study randomized 272 adults who had developed acute lung injury and required mechanical ventilation. Enteral nutrition was provided to both patient groups using a standard protocol, and the study supplement was provided twice daily to the study cohort of patients. The study was halted early because of futility. The ventilator-free and intensive care unit-free days were lower in the omega-3 group and, although not significant, hospital and 60-day mortality were higher in the omega-3 group.

A recent study comparing a special enteral formulation of eicosapentaenoic acid, gamma-linolenic acid, and antioxidants versus a standard formulation to patients during the early stages of sepsis (without organ failure) yielded different results.[69] The study, funded in part by the product manufacturer, revealed no significant difference in mortality between the two groups. A significant reduction in the appearance of cardiac and respiratory failure occurred in the study population given the special enteral formulation versus those given the standard formula control. Subjects in the test arm also experienced a benefit of fewer days on mechanical ventilation, fewer days in the intensive care unit, and shorter length of hospital stay. The concept of immunonutrition continues to evolve and, particularly within the last 5 years, the approach to the modulation of nutrition by timing to feed, amounts, route of administration,and composition of the nutritional product have yielded new information regarding how to optimally feed injured and critically ill patients.

DEEP VEIN THROMBOSIS PROPHYLAXIS

The hypercoagulable state exists immediately following severe traumatic injury, and an even more severe injury may be followed by increases in the hypercoagulable state.[70] When this condition exists in combination with patient immobility and direct venous injury, Virchow's triad for venous thrombosis is complete. Tissue injury may be responsible for the release of tissue thromboplastin, which initiates the conversion of factor VII to enzyme factor VIIa. Therefore, it is important to provide deep venous thrombosis (DVT) prophylaxis with subcutaneous mixed or low-molecular-weight heparins when possible, except in cases in which specific contraindications exist, such as intracranial hemorrhage, known peptic ulcer, solid organ laceration, and hematoma. An alternative is the placement of a sequential compression device on the limbs. The overall efficacy of DVT prophylaxis is well established; it is important that prophylaxis be maintained for the duration of the hospital stay or at least until the patient is fully mobile.[71]

Traumatic brain injury with intracranial hemorrhage prohibits the use of chemoprophylaxis. Recent data have demonstrated a three- to fourfold increased risk of DVT in brain-injured patients. This patient population requires early application of appropriate nonpharmacologic measures and an early decision on the placement of inferior vena cava filters (removable, if possible) for pulmonary embolism prophylaxis.[72]

STRESS GASTRITIS

Stress gastritis is common to the multiply injured intensive care unit population, and patients left untreated may have clinically significant gastrointestinal bleeding. The principal risk factors for stress gastritis are head injury, mechanical ventilation, and abnormal coagulation profiles. Prophylaxis using histamine-2 receptor antagonists or proton pump inhibitors is very effective.[73]

SUMMARY

Injury produces a series of physiologic changes mediated by local and systemic agents and systemic effects, mainly

cytokines, hormones, and activation of the sympathetic nervous system. The metabolic response aims to promote substrate delivery to the injured organs and promote healing. However, in the setting of severe trauma, these responses can result in organ injury, particularly to the lungs. These consequences can produce significant morbidity and mortality. An appreciation of nuances of the metabolic response allows the clinician to support the patient through the physiologic changes associated with the stress response caused by injury. Future research offers the promise of directly tailoring treatment and modulating the metabolic response to minimize the impact of major trauma.

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CHAPTER

2 Wound Healing

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OUTLINE

General Concepts of Wound Healing
- *Normal Soft Tissue Healing (Repair)*
- *Abnormal Soft Tissue Healing (Repair): Keloids and Hypertrophic Scars*

Wound Repair in Other Tissues of the Head and Neck
- *Normal Bone Healing (Repair)*
- *Complications in Bone Healing*
- *Normal Cartilage Healing (Repair)*
- *Normal Nerve Healing (Repair)*
- *Skin Grafting*

Factors in Suboptimal Wound Healing
- *Aging*
- *Infection*
- *Diabetes*
- *Smoking*
- *Malnutrition*
- *Immunosuppression*
- *Radiation and Chemotherapy*

Wound Management
- *Healing by Primary Intention or Primary Wound Closure*
- *Delayed Primary Closure or Wound Repair*
- *Healing by Secondary Intention*
- *Dressings and Topical Agents*

The capacity for self-repair is crucial for the survival of any organism, because without it the organism would likely perish after minimal injury. A wound is a disruption in the normal anatomic structure and function of tissue and is accompanied by cellular damage. Wound healing is an intricately coordinated series of processes that involve cellular and subcellular responses to tissue injury, leading to the release of cytokines and growth factors, cell activation, and resultant tissue regeneration.[1,2] The large variation in repair capacity of different tissue types is intriguing. For example, hepatic tissue has a high capacity to regenerate, whereas nerve tissue has an exceptionally low repair potential, given its inability to replicate. A solid understanding of the repair process is essential for optimizing patients' perioperative healing and is the basis for minimizing iatrogenic injury. It is especially important for surgeons treating maxillofacial injuries to possess a thorough knowledge of the wound-healing process, because nowhere else in the body are the effects of poor healing more noticeable and potentially disfiguring. To optimize the restoration of function and esthetic harmony after facial trauma, the surgeon must also be cognizant of patient-specific comorbidities and understand how health status influences the healing process.[3,4]

The challenge to optimize healing has placed wound physiology at the forefront of clinical and laboratory research.[5] The understanding of the remarkable cascade of events involved in wound repair and healing is advancing exponentially with the ongoing discoveries of the roles of growth factors and signaling pathways. There is growing interest in stem cell research, regenerative medicine applications, and bioactive wound healing products.

Wounds are classified as acute or chronic. Acute wounds have surgical, traumatic, pathologic, or ischemic causes. Surgical wounds, intentionally created in the operating room environment, vary in their degree of contamination, depending on their anatomic location and presence of local microbial flora. Subsequent healing is affected by the level of cleanliness or contamination of the wound. Traumatic injuries caused by blunt or penetrating trauma result in tissue laceration, abrasion, or even tissue avulsion. Other mechanisms of traumatic injury include tissue exposure to extremes of temperature, radiation, or caustic chemicals that cause injury by altering tissue pH, denaturing proteins, and causing local ischemia.[1,2] Pathologic processes such as neoplasms and nonhealing ulcers also cause tissue disruption. Skin breakdown or ulceration secondary to ischemia is related to impaired blood flow to an area by vascular occlusion, compression, stasis or pressure. Traumatic handling of tissues during treatment, including crush injuries and desiccation, can add further insult to the initial injury.

In the state of health, wound healing occurs in three distinct but overlapping phases—inflammation, proliferation, and remodeling (Table 2-1).[1-4,6] When the tissue's normal healing process experiences a disruption or delay, a chronic wound forms. Delays usually arrest healing at the inflammatory phase and result in excessive collagen deposition and scarring. Local factors impairing wound healing include the presence of foreign bodies or necrotic tissue within the wound, a high microbial burden, ischemia secondary to venous or arterial insufficiency, and tissue hypoxia secondary to radiation fibrosis. Some systemic factors that reduce healing capacity include aging, malnutrition, vitamin deficiencies, diabetes, immunocompromised states, atherosclerosis,

TABLE 2-1 Phases of Repair in Soft Tissue Wounds

Phase	Function
I. Inflammation	Vascular response Cellular infiltrate Polymorphonuclear leukocytes Macrophages Neovascularization Synthesis of granulation tissue
II. Proliferation	Cellular proliferation Macrophages Fibroblasts Collagen synthesis Endothelial cell proliferation Mature formation of granulation tissue Increase in mechanical strength
III. Maturation	Collagen remodeling Increase in wound strength Decrease in vascularity Macrophages Fibroblasts Formation of scar tissue

peripheral artery disease, collagen vascular disease, and chemotherapy.[7]

GENERAL CONCEPTS OF WOUND HEALING

NORMAL SOFT TISSUE HEALING (REPAIR)

Normal wound healing results in tissue regeneration and takes place in three separate but overlapping phases—inflammation, proliferation and remodeling (Fig. 2-1).[1-4,8-10]

The inflammatory phase of wound healing begins at the time of injury and lasts for 3 to 5 days. Vasoconstriction initiates the process, because catecholamines and prostaglandins (epinephrine and thromboxane) cause small blood vessels to constrict for initial hemostasis. Hemostasis via vasoconstriction is short-lived but is soon followed by the formation of a blood clot. The clotting cascade, prompted by vessel disruption, is initiated by platelets. Platelets, fragments of megakaryocytes that circulate in the blood for 9 to 11 days, contain glycogen, dense granules, and alpha granules (Fig. 2-2). When platelets adhere to the exposed subendothelial collagen of injured vessels with the aid of von Willebrand factor, they degranulate, releasing adenosine triphosphate, serotonin, prostaglandins, and thromboxane A2.[3,4] Serotonin, prostaglandins, and kinins increase vascular permeability. Platelets also release interleukins (ILs) and growth factors (transforming growth factor β [TGF-β], platelet-derived growth factor [PGDF] and vascular endothelial growth factor [VEGF]), which further potentiate platelet activation and aggregation. Insulin-like growth factor 1 (IGF-1), TGF-α, TGF-β, and PGDF attract leukocytes and fibroblasts to the wound. These factors also serve as chemoattractants when released into the bloodstream, recruiting neutrophils and monocytes to the site of injury. As platelets continue to aggregate, they form a platelet plug within the vessel. The complement, kinin, plasminogen, and clotting cascades are activated. The clotting cascade results in the deposition of fibrin, a key component that strengthens the platelet plug and acts as a scaffold for wound healing.

Vasodilation and inflammation follow hemostasis. This process is mediated by a variety of cytokines and factors, including histamine from mast cells (Fig. 2-3; Table 2-2), prostaglandins PGI_2 and PGE_2, prostacyclin, platelet-activating factor (PAF), bradykinin, leukotrienes, and nitric oxide. These chemicals cause leukocytes and plasma proteins to permeate into the wound. The endothelial cells of the small venules and capillaries transiently change shape and become rounded, creating gaps within the vessel wall that allow for leaking of plasma and fibrinogen.[3,4] Leukocytes migrate to the extravascular space via diapedesis (Fig. 2-4). Histamine, produced by mast cells and basophils, increases vascular permeability by inducing endothelial cell contraction and exposing the endothelial basement membrane. Consequently, histamine receptor blockers can prevent early changes in vascular permeability. Increased vascular permeability leads to the clinical findings of calor, rubor, dolor and tumor.[1] Wound heat and redness are a result of vasodilation and the presence of blood-borne cells. Pain, a nerve response caused by vasoactive amines or pressure from edema, protects the site from further tissue injury. Finally, wound swelling is caused by increased volume at the site of injury, secondary to edema from vascular permeability.

Some of the first cells to be recruited to the site of injury are neutrophils, which move across the endothelium via diapedesis within minutes of injury (Fig. 2-5). The neutrophil concentration within the wound peaks by 24 hours. They autolyse to release intracellular contents into the wound, including lysosomic proteases for degradation of nonviable tissue, debris, and bacteria (Table 2-3). Collagenases, elastases, cathepsin, and bactericidal cationic proteins are also released from neutrophil granules. Collagenases and cathepsin G activate complement and aid in the conversion of kininogens to kinins. Monocytes follow and concentrate at the site of inflammation within 2 to 3 days. These cells are paramount in directing wound healing because they transform into macrophages that continue wound débridement via the secretion of hydrolytic enzymes into the extracellular space. Collagenases, elastases, and cathepsins catalyze the conversion of plasminogen to plasmin to begin clot breakdown. Macrophages engage in microbial phagocytosis, a process enhanced by infiltrating opsonins. Macrophages release additional chemoattractant substances and growth factors that recruit fibroblasts and stimulate collagen production. Growth factors necessary for the formation of granulation tissue include TGF-α, TGF-β1, PDGF, epidermal growth factor (EGF), fibroblast growth factor (FGF), IGFs, TNF-α and IL-1. Neutrophils and macrophages continue to release cytokines that will initiate the proliferative phase of healing.

Continuing after the inflammatory phase, the proliferative phase (also known as the fibroblastic stage) is established by the fourth or fifth day and lasts 2 to 3

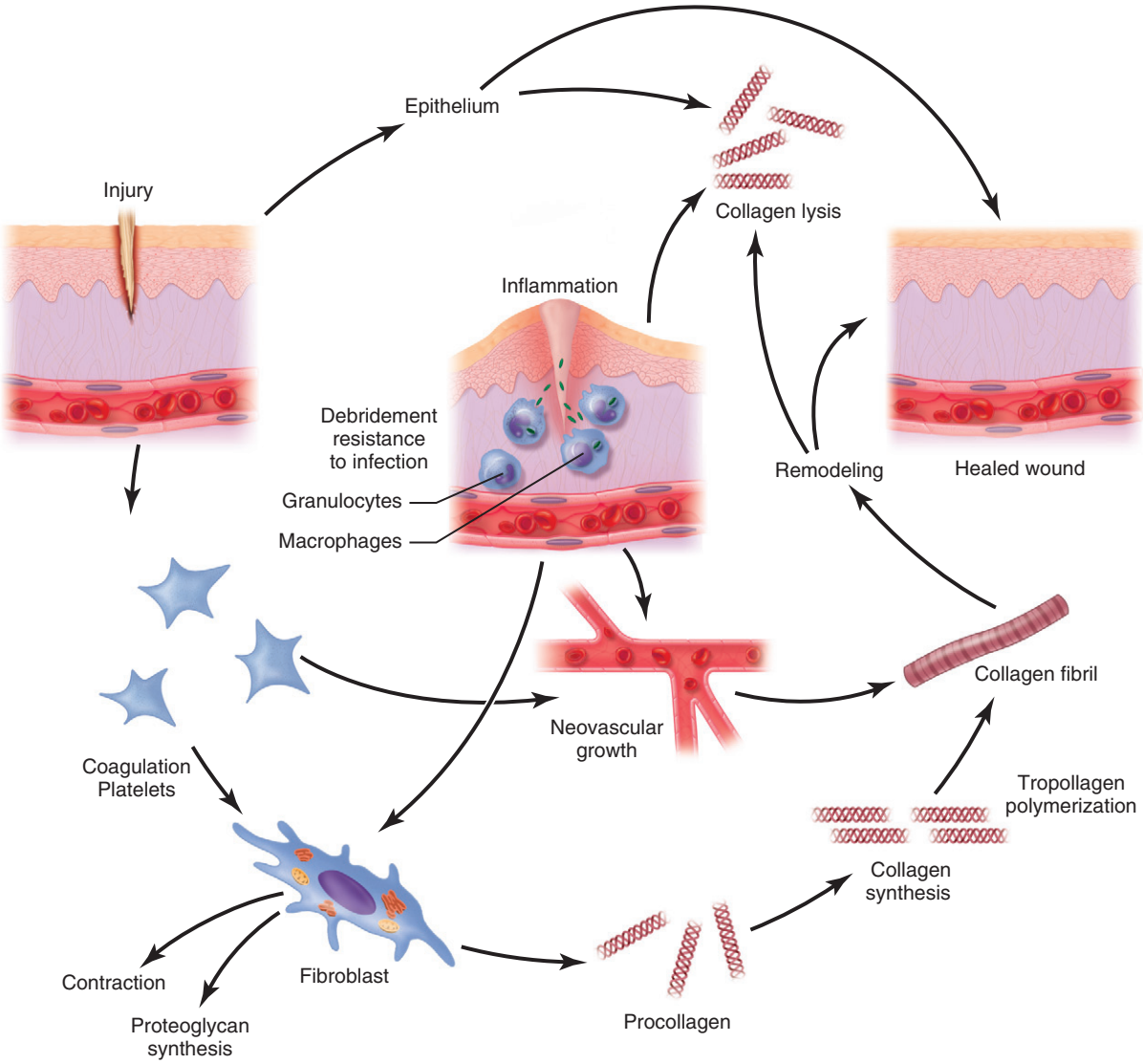


FIGURE 2-1 Overview of the process of soft tissue healing.

weeks. It is characterized by the ingrowth and proliferation of granulation tissue within the wound. Granulation tissue, a loose connective tissue matrix formed by collagen-secreting fibroblasts, supports neovasculature and inflammatory cells (Fig. 2-6). Fibroplasia, angiogenesis, and subsequent epithelialization further typify the proliferative phase. Responding to the release of PDGF and TGF-β, fibroblasts arrive at the wound on the third day and peak in concentration within 1 week. They actively produce proteoglycans and collagen, with force, stress, strain, and motion directing the collagen and proteoglycan alignment. Fibroblasts synthesize mostly type III collagen for approximately 3 weeks, until equilibrium is reached between the production and breakdown of collagen (Table 2-4). Budding vessels closely follow fibroblast activity. Neovascularization is enhanced by local factors such as hypoxia, elevated tissue lactate levels, and cytokines, such as FGF, VEGF, and PDGF.[1-4] Angiogenesis is crucial because new vasculature is required for the influx of oxygen and nutrients and the removal of metabolic waste products. The granulation tissue contains inflammatory cells and fibroblasts in a matrix of collagen and new vasculature. Epithelialization is promoted by EGF, TGF-α, and keratinocyte growth factor, and is itself composed of three phases—epithelial migration, proliferation, and differentiation. The dermal layer is reepithelialized and the contractile forces exerted by fibroblasts and myofibroblasts aid in reapproximating wound margins. Skin-grafting open soft tissue wounds can limit the amount of granulation tissue produced, thereby reducing scarring and tissue contracture. In the head and neck region, reepithelialization occurs faster in

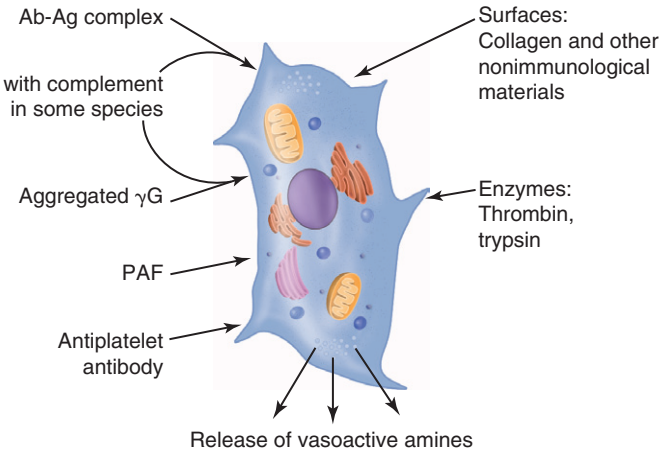


FIGURE 2-2 Stimuli that can induce release of vasoactive amines from platelets.

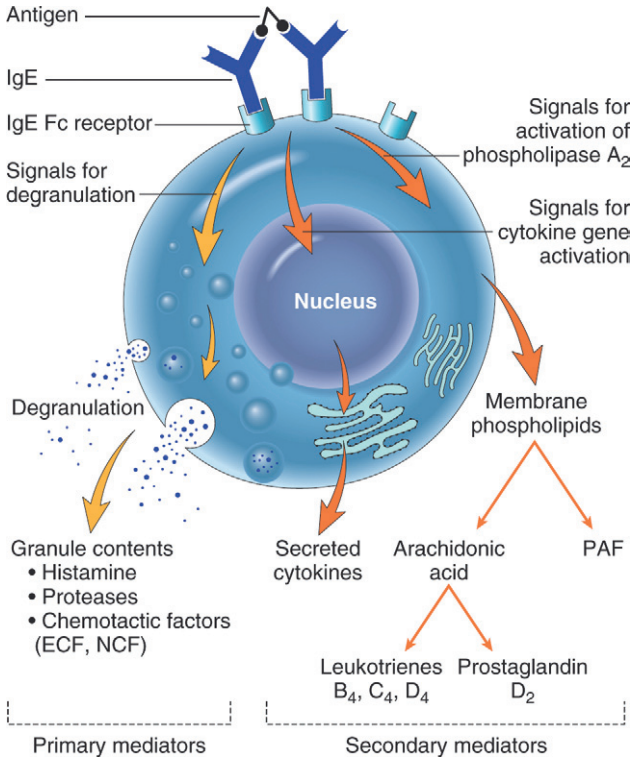


FIGURE 2-3 Stimuli that can induce release of vasoactive amines and other material from mast cells.

mucosa than in skin. Mucosal reepithelialization occurs over a moist surface whereas reepithelialization in skin occurs under a dry scab. The mechanical strength of the wound increases through the proliferative phase.

Tissue remodeling finalizes the wound healing process. Also known as maturation, the remodeling phase is characterized by an increase in wound tensile strength related to increased collagen production and breakdown. The remodeling process begins after the third week and usually lasts 6 to 12 months. Initial scar tissue becomes stronger as type I collagen replaces type III collagen. The augmented collagen deposition and subsequent collagen cross-linking increase the scar's tensile strength to 75%

TABLE 2-2 Chemical Mediators Derived from the Mast Cell

Preformed	Unstored
Histamine	Slow-reacting substance of anaphylaxis (SRS-A)
Heparin	Platelet-activating factors (PAFs)
Eosinophil chemotactic factor of anaphylaxis (ECF-A)	Lipid chemotactic factor (LCF)
Neutrophil chemotactic factor (NCF)	
Chymase	
N-Acetyl-beta-glucosaminidase	

From Yurt RW: Role of the mast cell in trauma. In Dineen P, Hildrick-Smith G, editors: *The surgical wound*, Philadelphia, 1981, Lea & Febiger, pp 37–62.

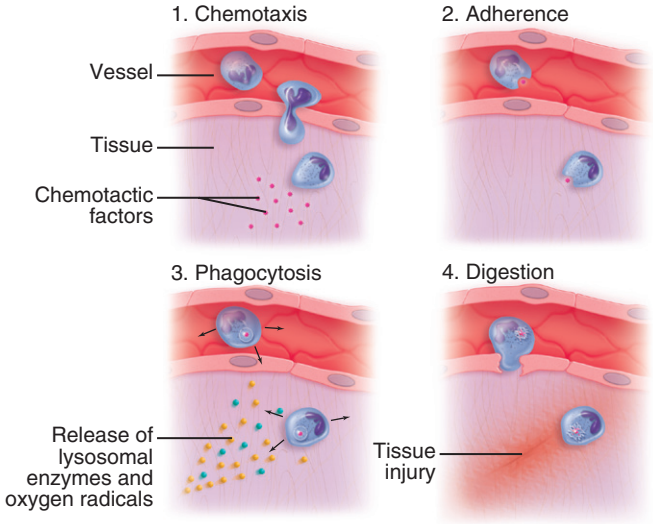


FIGURE 2-4 Sequence of reactions leading to tissue injury associated with influx of polymorphonuclear leukocytes. Note that in addition to chemotaxis, adherence, phagocytosis, and the digestion process, which normally result in particle inactivation, the release of neutrophilic constituents (lysosomal enzymes) may also result in tissue injury.

to 80% of the preinjury tensile strength. Tensile strength is defined as the load per cross-sectional area that can be supported by a wound; the increase in tensile strength is proportional to the rate of collagen synthesis. Over time, a decrease in the number of fibroblasts and macrophages is seen, with scar vascularity also decreasing as tissue proliferation declines.[1,2] These changes are clinically correlated to a less erythematous, flatter, softer scar.

ABNORMAL SOFT TISSUE HEALING (REPAIR): KELOIDS AND HYPERTROPHIC SCARS

Keloids and hypertrophic scars are aberrant forms of wound healing that result in proliferative scarring.[11] Although clinically similar, they differ in their formative timeline and boundaries. Keloids are benign growths of fibrous tissue that grow beyond wound boundaries. They more commonly occur over the sternum, ear lobes, back, trunk, and extremities. Keloids are firm and rubbery and

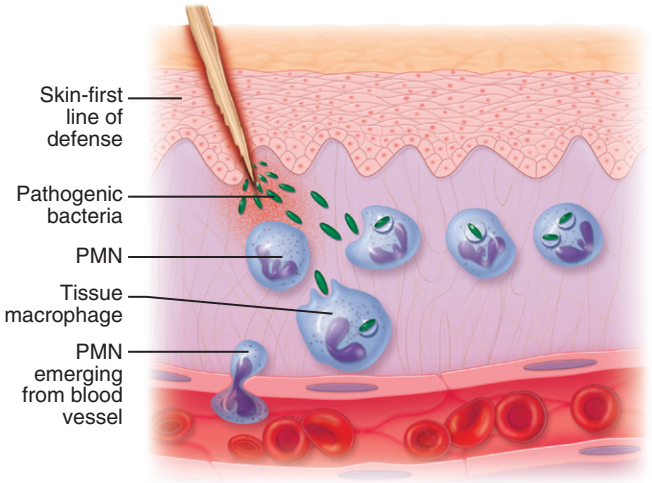


FIGURE 2-5 Phagocytosis by polymorphonuclear leukocytes (PMNs, neutrophils) and tissue macrophages following penetration of the skin and introduction of pathogenic bacteria into deeper tissue. The PMNs are more efficient in phagocytosis than the macrophages. Note that the PMNs are mobilized into tissue from blood vessels during the inflammatory process.

TABLE 2-3 Injurious Constituents of Neutrophils

Constituent	Activity
Collagenase, elastase, and cathepsin A	Hydrolysis of basement membranes, internal elastic laminae, cartilage and other connective tissue; generation of C5 fragments, angiotensin II
Basic proteins (three)	Increased vascular permeability
Basic protein (one)	Activation of mast cells, release of vasoactive amines
Leukotrienes C4 and D4	Increased vascular permeability, contraction of smooth muscle
Kininogenase	Hydrolysis of kininogen with release of vasoactive kinin
Procoagulant activity	Generation of fibrin, activation of platelets
Platelet activation factor (PAF)	Activation of platelets, increased vascular permeability, contraction of smooth muscles, activation of neutrophils
Leukotriene B4	Attraction of leukocytes
Lysosomal enzymes	Digestion of tissue constituents
Oxygen radicals	Damage to cells

From Bellanti JA: *Immunology*, ed 3, Philadelphia, 1985, WB Saunders, p 258.

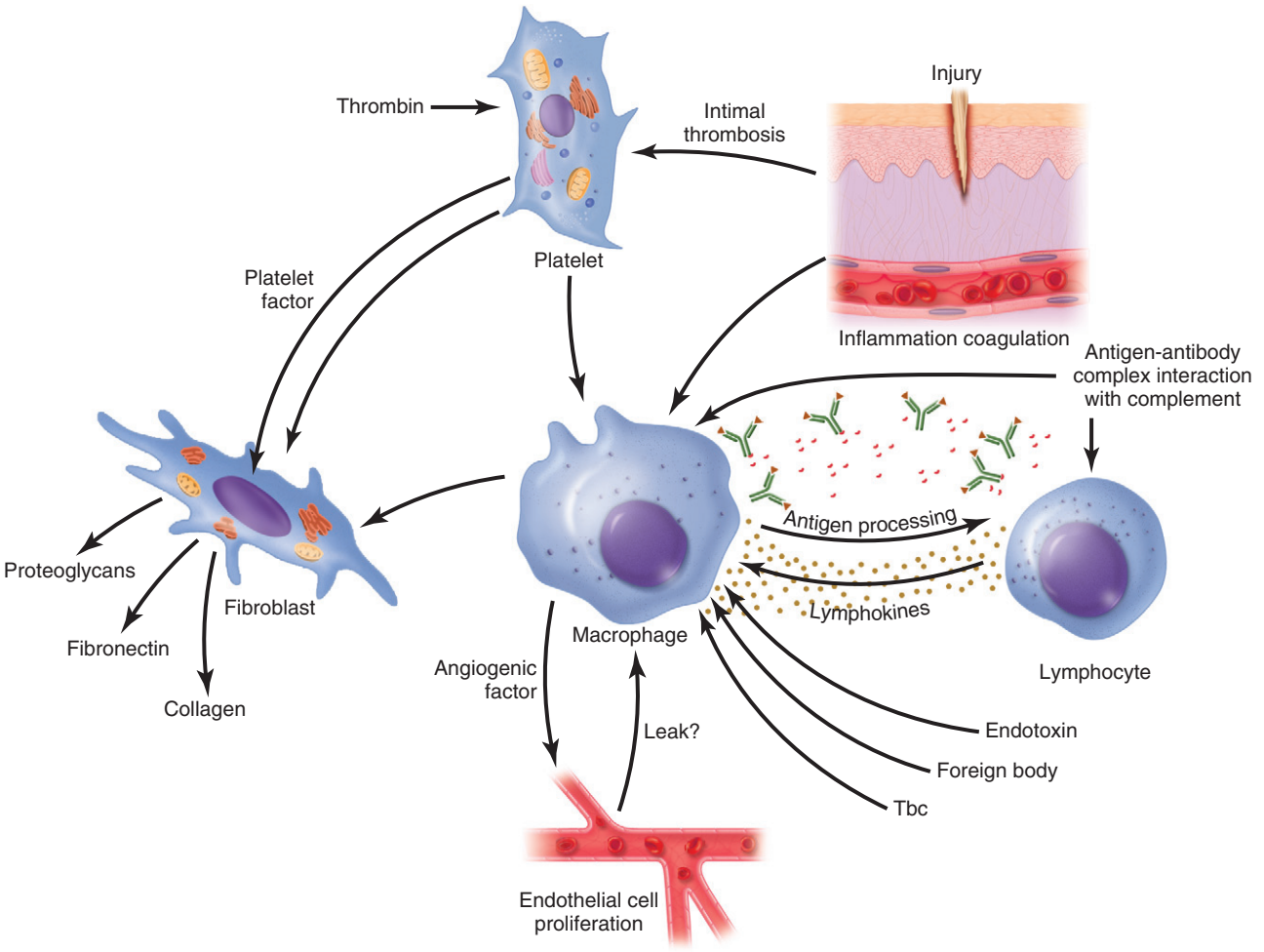


FIGURE 2-6 Cell interactions that lead to wound healing. The macrophage plays a central role that involves its activation by lymphokines, release of angiogenic factor, and collaborative roles with platelets, lymphocytes, and fibroblasts.

TABLE 2-4 Different Types of Collagens

Type	Tissue Distribution	Cells	Chemical Characteristics
I	Bone, tendon, skin, dentin, ligament, fascia, arteries, and uterus	Fibroblasts	Hybrid composed of two kinds of chains; low content of hydroxylysine and glycosylated hydroxylysine
II	Hyaline cartilage	Chondrocytes	Relatively high content of hydroxylysine and glycosylated hydroxylysine
III	Skin, arteries, and uterus	Fibroblasts Smooth muscle	High content of hydroxyproline and low hydroxylysine; contains interchain disulfide bonds
IV	Basement membranes	Epithelial cells	High content of hydroxyproline and glycosylated hydroxylysine; may contain large globular regions
A chain, B chain	Basement membranes	Uncertain	Similar to [alpha (IV)], but may contain larger globular domains

From Prockop DJ: Collagen biochemistry and the design of agents to inhibit excessive accumulation of collagen during wound repair. In Dineen P, Hildrick-Smith G, editors: *The surgical wound*. Philadelphia, 1981, Lea & Febiger, p 97.

can be erythematous, painful, or pruritic. They develop months after trauma, piercing, or surgical incisions and are caused by an overproduction of connective tissue, likely secondary to altered apoptosis or hyperproliferation of keloidal fibroblasts. Genetic causes have also been implicated in keloid formation. Keloids occur more commonly in certain ethnic populations. The incidence of keloid formation is correlated with increased skin pigmentation. Keloids rarely improve without treatment and a variety of treatment modalities have been described. First-line treatment is intralesional injection of corticosteroids into the scar to reduce fibroblastic production of collagen and extracellular matrix proteins. Localized pressure therapy, interferon, or fluorouracil can be used in combination with intralesional corticosteroid injections. Keloids can also be surgically excised or treated with radiation, cryosurgery, or topical imiquimod. The use of calcineurin inhibitors is currently under investigation. Nevertheless, regardless of treatment modality, keloids commonly recur to some degree after treatment.[12]

Hypertrophic scars have a similar appearance to keloids but differ from keloids in that they do not extend beyond the margins of the original wound. They also appear shortly after injury and may recede over time. Hypertrophic scars, characterized by prolonged inflammation and collagen deposition, are red, firm, and elevated. Hypertrophic scars are also treated with intralesional corticosteroid injections and are less likely than keloids to recur after treatment.[13]

WOUND REPAIR IN OTHER TISSUES OF THE HEAD AND NECK

NORMAL BONE HEALING (REPAIR)

Normal bone healing parallels soft tissue healing. Both tissue types undergo three phases of wound healing—inflammation, proliferation, and remodeling.[3,10,14,15] However, bone healing also undergoes calcification. As with soft tissue repair, the inflammatory stage in bone healing begins immediately after the injury and lasts up to 5 days. Inflammation is stimulated by vessel injury in the haversian canals and periosteum and by the presence of bony debris or necrotic material in the fracture site. Vasoconstriction allows a blood clot to form, and inflammatory cells phagocytize debris and bacteria. A hematoma is formed within the fractured bone. The necrotic bone edges are resorbed. The proliferative fibroplastic stage follows. Pleuripotential mesenchymal cells and fibroblasts enter the site of injury to lay down fibrous tissue, cartilage, and immature bone fibers (Table 2-5). These components permit the wound to gain some strength over the 2 to 3 weeks following injury. Granulation tissue forms as a matrix of fibrin, collagen, and neovasculature is laid down. If the fracture segments are not precisely reduced to the preinjury anatomic position, or if bone is avulsed, leaving a residual space between the two bony segments, the fracture will heal by secondary intention. Greater collagen deposition is then required to bridge the gap, resulting in callus formation at the surface and within the fractured bone. The soft cartilage callus calcifies into woven bone as osteoblast and osteoclast concentrations increase within the fracture site (Fig. 2-7). Osteoblasts continue to deposit osteoid on spicules of calcified cartilage and this osteoid is later calcified to immature bone (Fig. 2-8). The callus, much like a rudimentary splint, offers the fractured bone some minor stability against bending and torsion (Fig. 2-9). However, immobilization is required for healing to proceed; otherwise, a fibrous union will result.

In areas adjacent to endosteum, where the vascular supply and osteoprogenitor cells abound, no intermediate fibrocartilage is seen. Instead, the endosteal osteoblasts form a direct bony callus. Similarly, no cartilaginous callus is formed when minimally displaced fractures heal under immobilization or when acute fractures are anatomically reduced without a gap between the segments and immobilized with rigid fixation. This type of fracture healing is known as primary intention (Fig. 2-10).

As long as the fracture site remains immobilized, maintains an adequate blood supply, and remains free of infection, the remodeling stage will complete the fracture healing process,. The callus completely ossifies as

TABLE 2-5 Polypeptide Growth Factors Involved in Bone Healing

Stage of Bone Healing	Growth Factor	Function(s)
I. Vascular	Plasma fibronectin	Anchors cells in the ground substance; ingrowth required for collagen formation
	Endothelial cell–derived growth factor	Mitogen
II. Callus	Platelet-derived growth factor	Mitogen—fibroblasts, bone cell formation; activates monocytes; promotes bone resorption
	Epidermal cell growth factor	Mitogen—cartilage, bone; inhibits type I bone collagen synthesis
	Fibroblast growth factor	Mitogen—fibroblasts, chondrocytes
	Insulin-like growth factor	Chondrocyte proliferation; chondrocyte proteoglycan synthesis
	Nerve growth factor	Mitogen
III. Bone formation, remodeling phase	Epidermal growth factor	Promotes bone resorption
	Fibroblast growth factor	Promotes bone resorption in high doses
	Insulin	Synergistic effect with bone growth factors
	Interleukins (monocyte products)	IL-1: Fibroblast proliferation, collagenase production, prostaglandin production IL-2: T cell growth factor, stimulation of bone resorption by osteoclastic activation factor (OAF) production

From Simmons DJ: Fracture healing perspectives. *Clin Orthop Relat Res* 200:100, 1985.

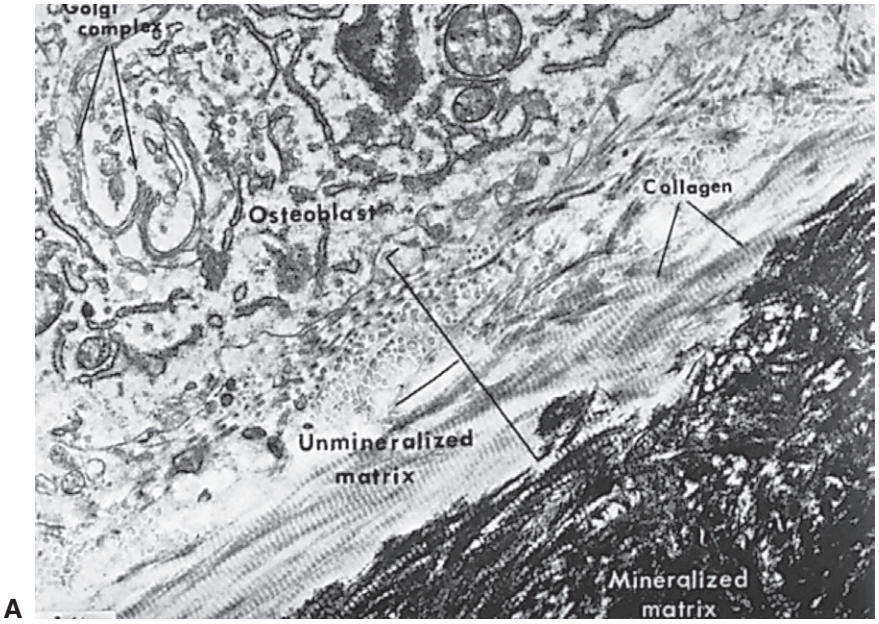
osteoclasts gradually resorb the immature bone and it becomes remodeled into lamellar bone. The gradual osteoclastic resorption of immature woven bone with osteoblastic bone formation and maturation to lamellar bone is known as creeping substitution.[3,10,14]

Dental extraction sites heal by secondary intention. The socket first fills with blood that quickly coagulates within the first 24 hours to form a blood clot that seals the alveolar socket from the oral cavity. Inflammation ensues soon after the injury of dental extraction. During the first week of healing, leukocytes débride the extraction site, phagocytose bacteria, and débride bony fragments. Osteoclasts resorb the marginal bone along the extraction socket (Fig. 2-11). Meanwhile, epithelial cells begin migrating along the socket wall to reepithelialize the socket surface. During the second week of healing, granulation tissue is generated while osteoid is deposited by osteoblasts to form woven bone. By the fourth week of healing, full epithelialization is achieved. The alveolar bone is remodeled over 4 to 6 months. During this time, the alveolar cortical bone and trabecular woven bone are resorbed and replaced by lamellar bone.[10,14]

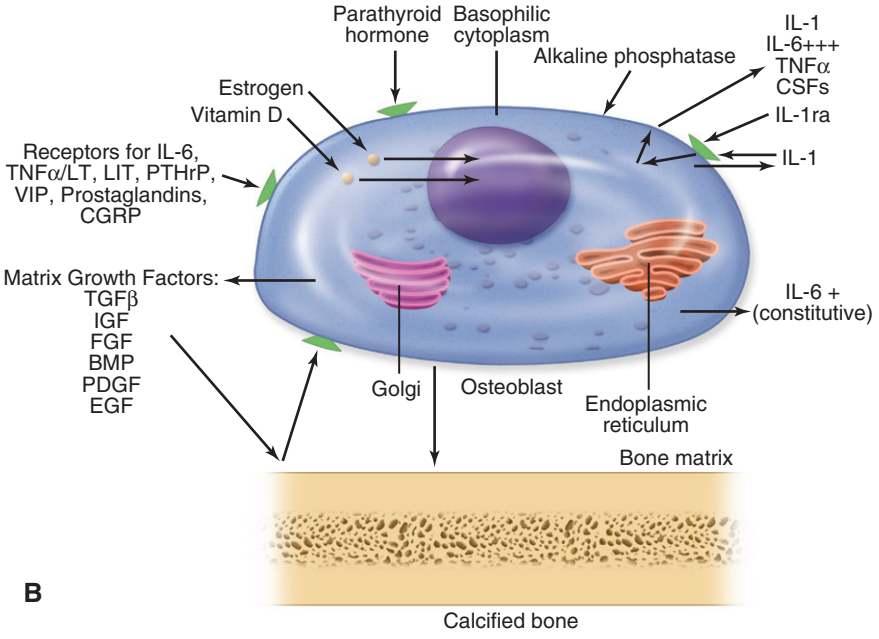
COMPLICATIONS IN BONE HEALING

Repeated trauma to an area undergoing bone healing can result in malunion or nonunion of a bone fracture (Fig. 2-12). A malunion results when the bony fracture segments heal in an incorrect or nonanatomic position, which can lead to a deformity.[16] For fractures of the jaws, malunion will create a malocclusion. A bony nonunion results when the fracture segments do not form bone to bone contact, but instead remain bridged by fibrous tissue. In the maxillofacial region, bony nonunion is most commonly seen in inadequately treated mandible fractures.[17] The mandible is a bone with high functional forces and repeated trauma can occur if the patient masticates before bone healing is complete.[18] Tobacco use and excessive alcohol intake increase the risk of nonunion.[19] Nicotine impairs bone healing by preventing vascular ingrowth and diminishing osteoblast function during the proliferative stage.[20,21] Use of certain anti-inflammatory or cytotoxic medications (e.g., nonsteroidal anti-inflammatory drugs [NSAIDs], glucocorticoids, chemotherapeutic agents, fluoroquinolone antibiotics) during the inflammatory phase of bone healing has also been implicated in an increased risk of nonunion,[22] but the actual clinical significance may be negligible. A nonunion of the mandible can lead to severe pain, instability of the mandibular fracture segments, and malocclusion. A nonunion requires open surgical treatment, with removal of the fibrous callus and adequate fixation of the fracture segments for stability. In cases in which the defect between the fracture segments are large, a bone graft may be required to promote proper bone healing and help bridge the gap between these fracture segments.

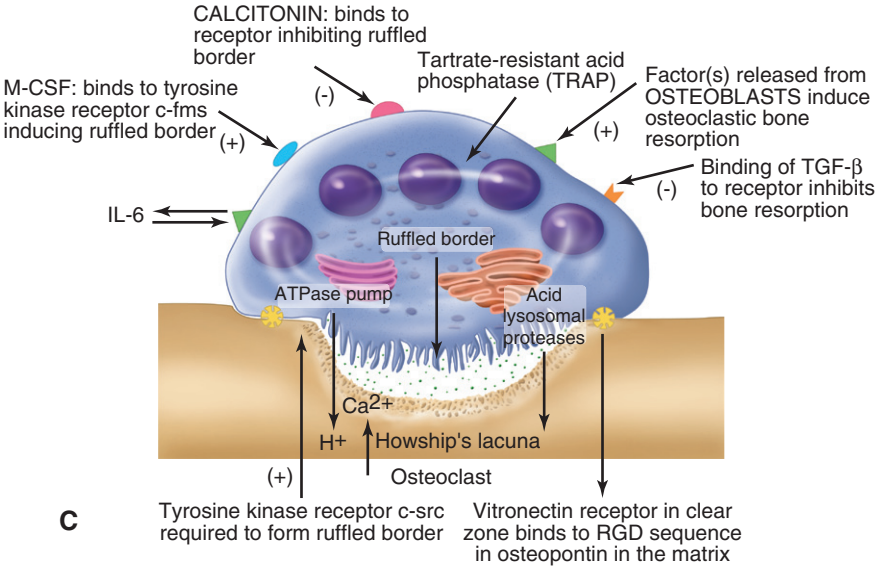
Open fractures, especially those in communication with oral or respiratory secretions or those exposed to exogenous debris from trauma, are at risk of infection. Contaminated open fractures are also at risk of developing osteomyelitis. Osteomyelitis is an infection of bone that extends to the medullary space (Fig. 2-13). Osteomyelitis can be painful and debilitating, and requires surgical débridement of the involved bones and long-term antibiotic therapy. Osteomyelitis can be post-traumatic or can be a result of the hematogenous spread of infectious organisms. In the jaws, osteomyelitis can occur secondary to chronic odontogenic infection. When osteomyelitis occurs post-traumatically, the bone fracture will not heal appropriately and a nonunion often occurs.



A



B



C

FIGURE 2-7 **A,** Osteoblast engaged in synthesis. The cytoplasm is indicative of a cell actively involved in the export of proteins. An elaborate Golgi complex and rough endoplasmic reticulum are evident. The unmineralized front containing collagen fibrils is adjacent to the mineralized matrix, which is the site of release of the internally produced matrix. **B,** An osteoblast secreting bone matrix proteins. Secretory products, including matrix proteins, cytokines, and membrane and intracellular receptors, are indicated. **C,** A mature osteoclast in a resorption pit. Binding at the clear (sealing) zone is indicated, together with specific receptors and factors involved in the formation of the ruffled border.

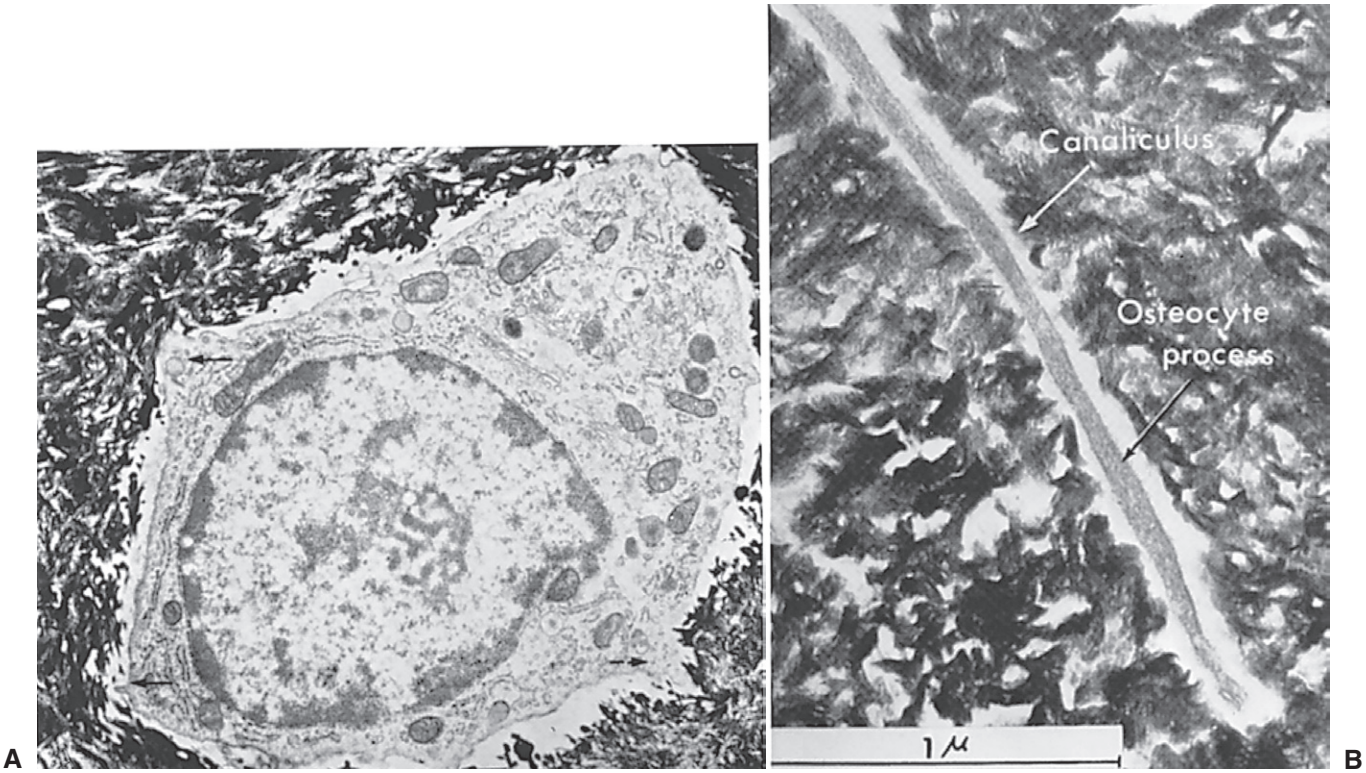


FIGURE 2-8 **A,** Photomicrograph of an osteocyte with its cytoplasmic processes visible on the periphery *(arrows)*. **B,** Longitudinal section of cytoplasmic process of osteocyte in **A**. Through this extensive canalicular system, osteocytes are able to transfer oxygen and metabolites (e.g., calcium) from blood to nourish the surrounding bone.

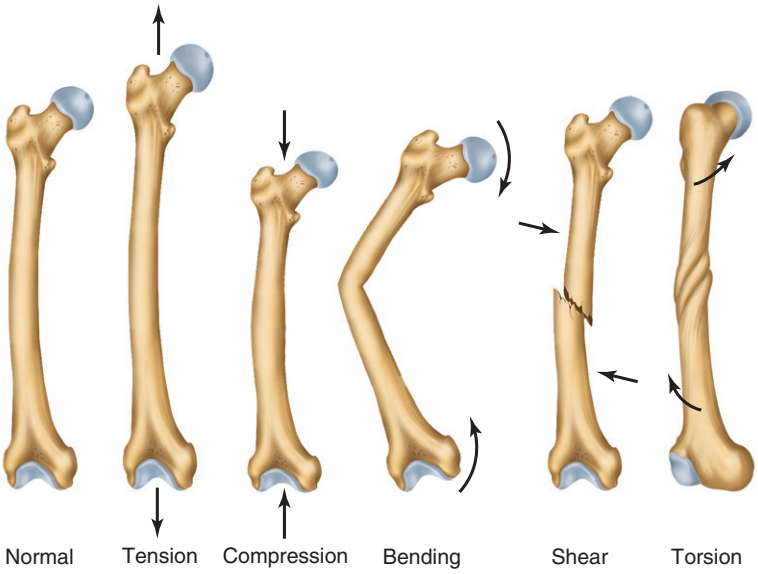


FIGURE 2-9 Various loading models (stress) seen on bone.

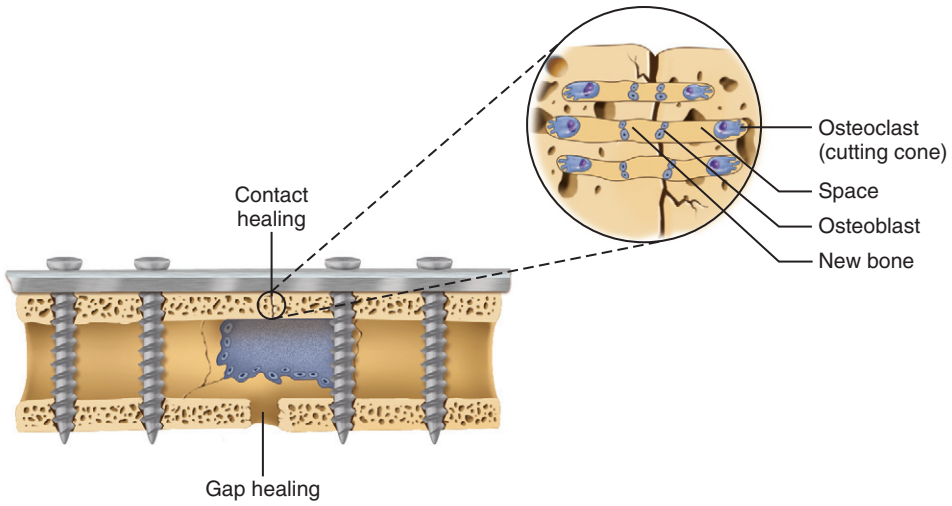


FIGURE 2-10 Gap healing has occurred opposite to the compression plate, with direct bone deposition between the cortical ends. Contact healing is taking place within the cortical bone in the area of compression. The osteoclastic cutting cones have produced cores into which osteoblasts lay down new bone *(inset)*.

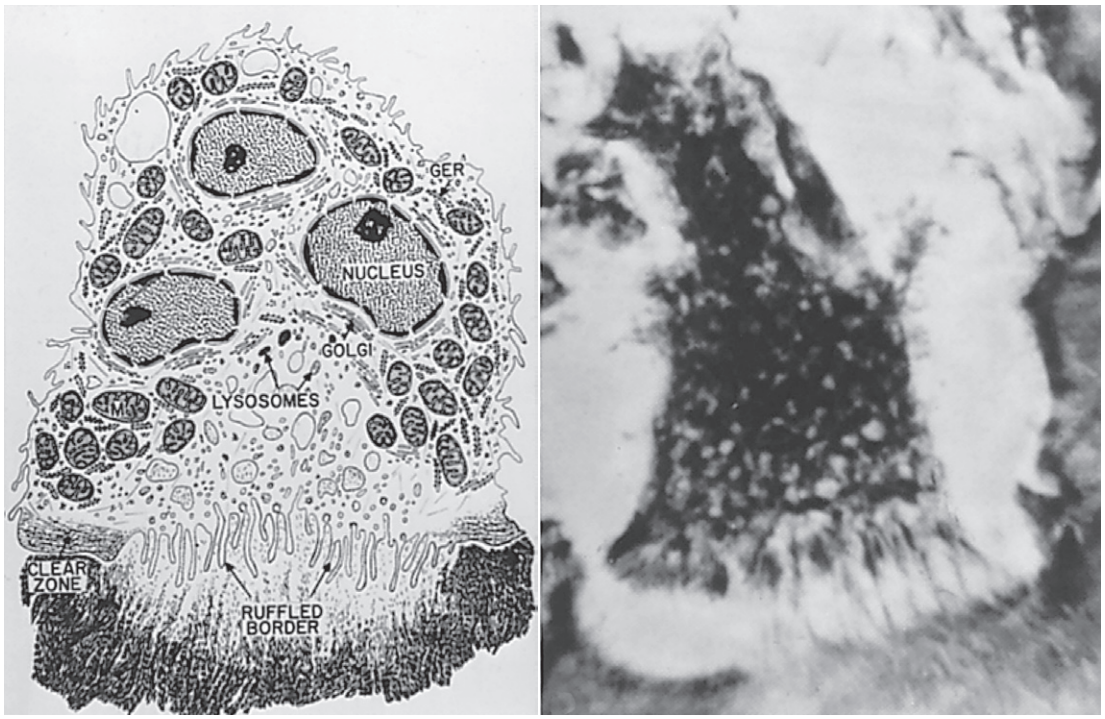


FIGURE 2-11 **A,** An osteoclast. Osteoclasts are relatively large multinucleated cells. Their cytoplasm is laden with lysosomal enzymes, a well-developed Golgi process, mitochondria (M), and rough endoplasmic reticulum (RER). The active process of bone resorption occurs at the convoluted membrane of the osteoclast (ruffled border). **B,** Photomicrograph of an osteoclast with a prominent ruffled border.

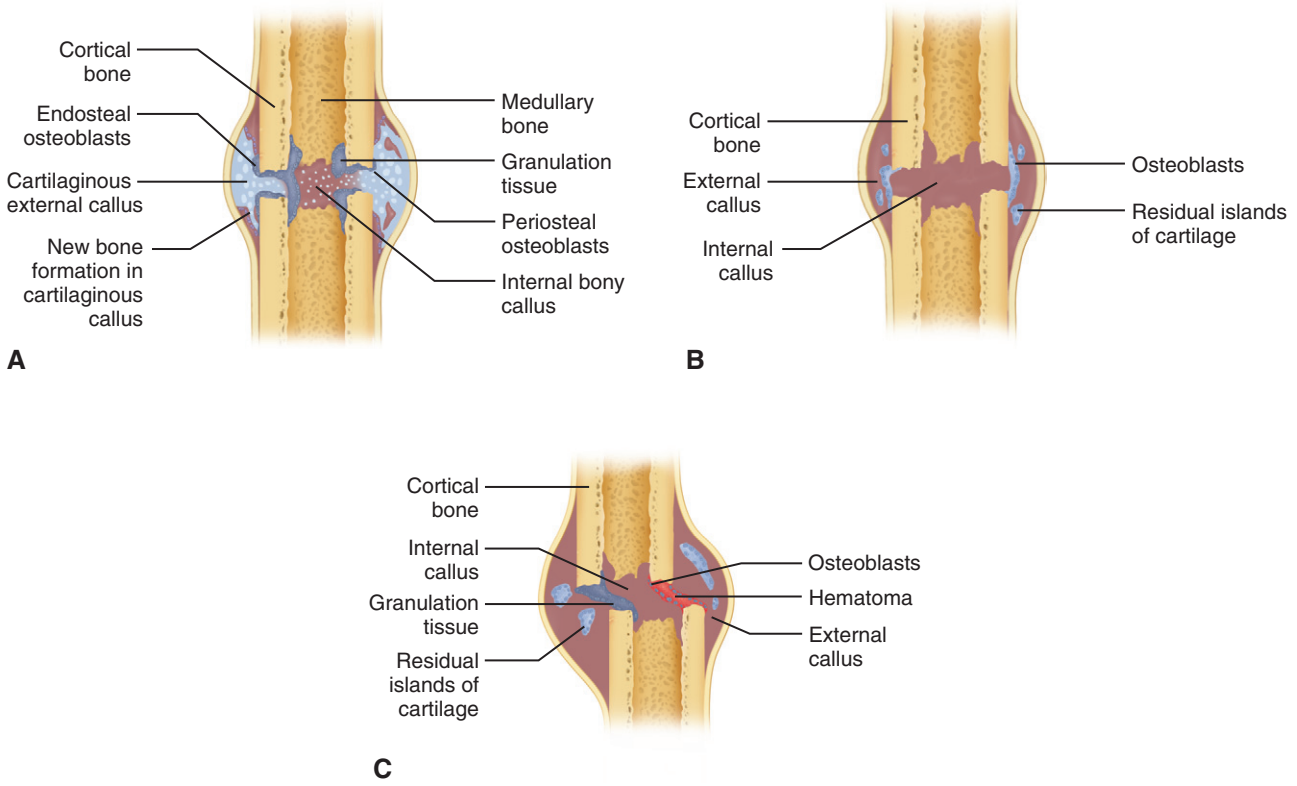


FIGURE 2-12 **A,** Late cartilaginous callus stage. The internal bony callus has bridged the defect with direct bone formation by endosteal osteoblasts. The cartilaginous callus has formed peripherally and is undergoing ossification. **B,** Bony callus stage. The internal bony callus is complete. Ossification of the cartilaginous callus has left small islands of residual cartilage. **C,** Bony callus stage—fracture with notable displacement illustrating adaptation of the callus.

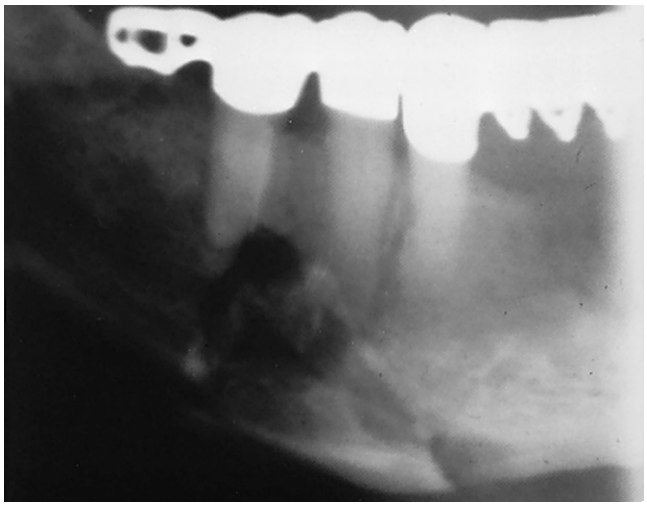


FIGURE 2-13 A comminuted mandibular body fracture that resulted from a high loading force. The force was dissipated at the point of impact, resulting in the comminution. This patient suffered no other mandibular fractures.

Osteomyelitis is more common in the mandible than in the maxilla because of the relatively poorer vascular supply. Mandibular osteomyelitis can be exquisitely painful and can be associated with anesthesia, hypoesthesia, or dysesthesia of the inferior alveolar nerve.[23-25]

NORMAL CARTILAGE HEALING (REPAIR)

In the maxillofacial region, cartilage is found in the articular surfaces of the temporomandibular joint (TMJ) and in the auricular and nasal cartilages. Facial cartilaginous defects are generally caused by traumatic injury, infection, pathology, or chronic degeneration. Cartilage repair is challenging, given its limited inherent capacity for self-repair secondary to its low cellular density and minimal vascular supply. Large defects in nasal and auricular cartilage usually require autologous cartilage grafting.[26,27]

Although cartilage is a metabolically active tissue, it has no inherent vascularity or lymphatic vasculature. A poor inflammatory response is elicited after cartilage injury because progenitor cells from the blood or bone marrow are unable to access the damaged cartilage. Therefore, cartilage healing and repair is significantly more limited than with other tissues of the oral and maxillofacial region.[27,28]

Damage to articular cartilage initiates a healing response in areas in which cartilage and bone are in direct contact. If the damage extends from the articular surface into the subchondral bone, bleeding from the bone into the cartilaginous defect occurs. This bleeding activates the normal bone-healing process.[26] A hematoma is formed and fibroblasts subsequently lay down collagen. The resultant fibrous tissue is not consistent with the structural and functional properties of articular

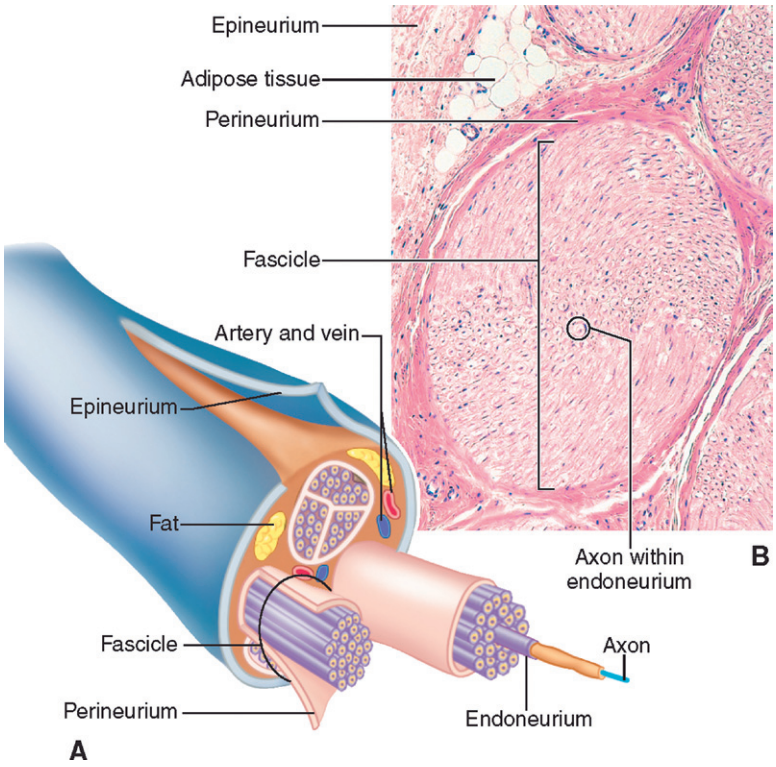


FIGURE 2-14 Structure of a myelinated peripheral nerve. (From Patton K, Thibodeau G: *Anatomy and Physiology*, ed 7, Mosby, St. Louis, 2010.)

cartilage. Alternatively, partial-thickness cartilage injuries do not reach the subchondral bone, so there is no access for blood or cells to the site of injury. Therefore, a partial-thickness cartilage injury will not undergo repair and the defect will remain. This significant challenge with cartilage healing has clinicians and researchers searching for techniques to regenerate damaged cartilage successfully.[14,29,30]

NORMAL NERVE HEALING (REPAIR)

Nerve injuries (Fig. 2-14) can be categorized by the Seddon or Sudderland classification. The Seddon classification describes three types of nerve injury—neuropraxia, axonotmesis, and neurotmesis (Fig. 2-15). Neuropraxia is a transient interruption in nerve conduction, sometimes described as nerve bruising. Axonal continuity is preserved in neuropraxia. Recovery is spontaneous but may take weeks to months. In axonotmesis, individual axons are damaged within the nerve, with the epineurium being preserved. Wallerian degeneration, the degradation of distal axons with concomitant loss of Schwann cells, occurs (Fig. 2-16). As long as the proximal nerve body survives, axonal regeneration may occur at a growth rate of 1 mm/day. Complete nerve transection without preservation of nerve continuity occurs in neurotmesis. Wallerian degeneration ensues after neurotmesis. Nerve transection injuries rarely recover spontaneously and therefore require surgical intervention for improved outcomes. After neurotmetic injuries, neuromas often form as the axons attempt to regenerate in a random fashion.[10]

Alternatively, nerve injuries can be described by the Sudderland classification. The original Sudderland classification encompassed five degrees of nerve injury. A sixth classification was later added by Mackinnon. A first-degree injury, similar to neuropraxia, involves a nerve conduction block. Second- to fourth-degree injuries are similar to axonotmesis injuries and wallerian degeneration occurs in these. In a second-degree injury, some nerve fibers are damaged, without injury to endoneurium. Endoneurium involvement without perineurium damage is seen in a third-degree injury. Perineurial damage within an intact epineurium is seen in fourth-degree injuries. Fifth-degree injuries are complete nerve transections, much like neurotmesis injuries. Surgical intervention is recommended for fourth- and fifth-degree injuries.

Examples of traumatic nerve injuries in oral and maxillofacial surgery include transection of the inferior alveolar nerve or infraorbital nerve with a fracture of the mandible or midface, respectively, transection of the lingual nerve during third molar removal, and transection of the facial nerve or trigeminal nerve branches with penetrating trauma to the face.[31-33]

SKIN GRAFTING

Skin grafts may be used to cover traumatic defects that cannot be adequately repaired with primary closure alone or defects that are otherwise not expected to resurface quickly.[34] Skin grafts can be classified as split-thickness or full-thickness, depending on the extent of dermis included in the graft. Full-thickness skin grafts (FTSGs) are composed of epidermis and the entire dermis. Split-thickness grafts, on the other hand, are composed of epidermis and a variable thickness of partial dermis. Split-thickness skin grafts (STSGs) are further subdivided based on the thickness of harvested dermis. Approximate measurements for split-thickness grafts are

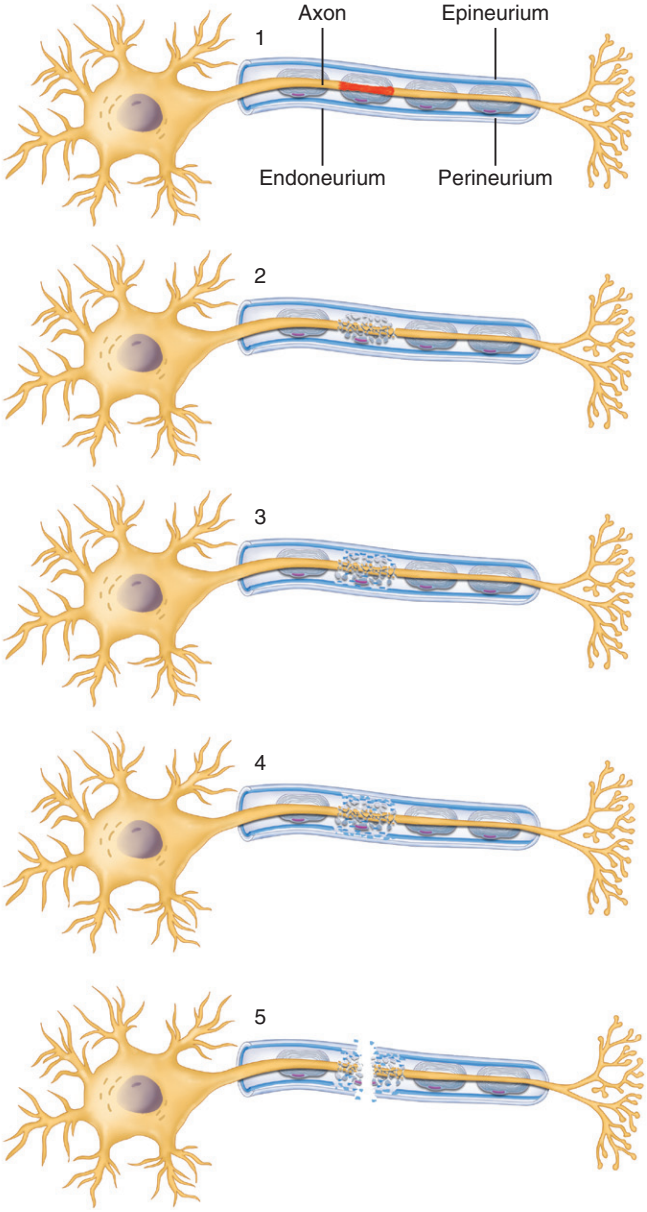


FIGURE 2-15 Sunderland classification system. Shown is a schematic representation of the first through fifth degrees of nerve injury.

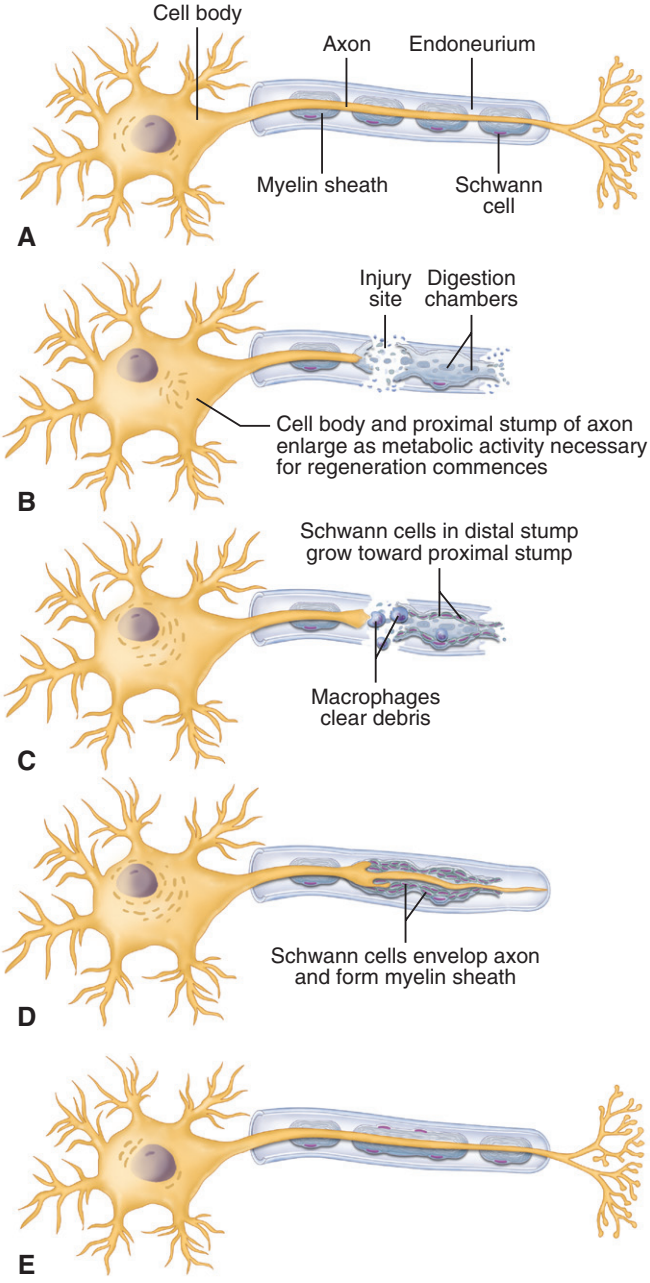


FIGURE 2-16 Stages of peripheral nerve healing. **A,** Normal nerve cell and axon. **B,** Early wallerian degeneration. **C,** Phagocytosis and Schwann cell proliferation. **D,** Axon growth. **E,** Repaired nerve fiber.

thin (0.2 to 0.3 mm), intermediate (0.3 to 0.5 mm) and thick (0.5 to 0.8 mm). Skin grafts differ in more than just thickness; full-thickness and split-thickness grafts have different properties that can be exploited for the patient's reconstructive needs. Full-thickness grafts undergo more initial primary contracture because of the presence of more elastic fibers in the dermal layer, but significantly less secondary contracture. Therefore, full-thickness skin grafts are placed in areas in which tissue contraction would otherwise cause a deformity, such as the face or hands, or over joints. These grafts also have the benefit of maintaining more normal skin characteristics, such as texture, pigmentation, and presence of dermal appendages (e.g., hair, sweat glands). Split-thickness grafts have less primary contracture because fewer elastic fibers are present in partial dermis, but they undergo more secondary contracture than full-thickness grafts. Thinner split-thickness skin grafts have better survival than thicker grafts because the epithelium is in closer contact with the underlying graft bed vasculature. Split-thickness grafts also have the advantage of ease of harvesting and grafts with a much larger surface area can be obtained.

Graft donor sites are selected with the type and appearance of the recipient site in mind.[35] Donor site morbidity, aesthetics, and ease of donor site closure are also considered. Usual donor sites for STSGs include the thigh, abdomen, buttocks, and inner arms. FTSG donor sites include the forehead, neck, groin, and periauricular or

supraclavicular skin. Skin grafts can be harvested free-hand with a scalpel or a variety of specialized blades, or with a powered dermatome. Free- hand skin grafts are of variable thickness and tend to have irregular borders. Those harvested with a powered dermatome are of more consistent thickness and have less irregular borders. Meshing of split-thickness grafts increases the graft area for resurfacing and allows fluid egression from the graft bed, thereby reducing the risk of hematoma or seroma formation under the graft. However, meshing increases graft contraction and can produce a less cosmetic or less functional result. In cosmetic areas or high-function sites (e.g., over joints), unmeshed sheet grafts should be used. The resultant skin graft is sutured to the recipient site and covered with a bolster dressing for gentle pressure to prevent fluid from collecting under the graft and to protect from inadvertent trauma to the grafted site.[36,37]

A skin graft initially adheres to the graft bed by fibrin. For the first 2 to 3 days, skin graft survival is dependent on the imbibition of nutrients from the underlying graft bed capillaries. The graft bed must therefore have an adequate blood supply to ensure its survival. Graft revascularization begins early. Capillaries of the skin graft and graft bed reanastomose in a process known as inosculation. Graft neovascularization also occurs, with random vascular ingrowth of new vessels. Reinnervation follows as nerve fibers subsequently grow into the graft. Initial return of skin sensation over a grafted site takes approximately 2 months, although grafted sites generally do not regain sensation equal to that of unaffected normal skin.

Full-thickness skin grafts are slower to revascularize than split-thickness grafts because of the presence of a thicker dermal component. For the same reason, imbibition occurs more slowly in full-thickness grafts. Full-thickness skin grafts therefore are at a greater risk of graft failure. Defatting the underside of a full-thickness graft improves imbibition and increases the rate of revascularization. Causes of skin graft failure include hematoma or seroma formation (separation of graft from graft bed), presence of high bacterial load, inadequate débridement prior to graft placement, and inadvertent trauma to the graft site. As discussed in the following section, the patient's health and nutritional status should be optimized to allow for proper healing and graft take. Grafted sites should be dressed with a bolster dressing that discourages hematoma or seroma formation, helps maintain contact between the graft and recipient site bed, and prevents micromotion. The split-thickness donor site can be dressed with occlusive or semiocclusive dressings, whereas the full-thickness donor site should be closed primarily.[36,37]

FACTORS IN SUBOPTIMAL WOUND HEALING

Chronic wounds arise when healing is compromised. These wounds generally have extended inflammatory phases with resultant fibroplasia that increases tissue scarring and wound contracture. Common risk factors for poor healing in the inpatient or trauma setting are advanced age, wound infection, diabetes, history of smoking, and malnutrition. These risk factors can be classified as local or systemic (Table 2-6). Local factors include infection or a high bacterial burden, presence of foreign bodies or necrotic tissue within a wound, ischemia secondary to vascular obstruction or pressure, venous or arterial insufficiency, and hypoxia related to radiation changes. Systemic factors include aging, diabetes mellitus, malnutrition or vitamin deficiencies, immunocompromised states, peripheral artery disease, and use of chemotherapeutic agents.[7]

TABLE 2-6 Impediments to Wound Healing

Local	Systemic
Foreign bodies	Smoking
Venous insufficiency	Diabetes
Pressure, trauma	Corticosteroids
Ischemia, hypoxia	Malnutrition
Radiation	Cytotoxic chemotherapy
Salivary contamination	Vitamin deficiency
Scarring	Chronic illness
Hematoma	

TABLE 2-7 Criteria for Bacterial Infection Based on Quantitative Wound Culture

Microorganism	Criterion for Infection
General flora	$>10^5$ bacteria/cm^3
Enterococcus	$>10^3$ bacteria/cm^3
Group B *streptococcus*	Any

AGING

As with most metabolic processes, the capacity for wound repair declines with age. Aging results in generalized tissue thinning caused by collagen loss, as well as vascular compromise and poor perfusion. These changes cause tissues to be friable and more easily damaged. Thus, injuries in older adults have compromised repair potential. In addition, older patients are more likely to have associated health conditions that adversely affect healing. To reduce the risk of poor wound healing associated with advanced age, patients should undergo preoperative optimization of comorbid conditions.[1,7]

INFECTION

Infection is a major cause of impaired wound healing.[38] Wounds are described as being sterile, contaminated, colonized, or infected, depending on the mechanism of injury (e.g., bite wound) and its location with respect to normal bacterial flora. If not properly addressed, contaminated wounds can become infected (Table 2-7). A high microbial burden in a wound increases the host's inflammatory response to the area. Bacteria release endotoxins and metalloproteases that destroy the extracellular matrix of healing tissue and cause cell lysis. Therefore, bacterial infection and increased inflammation can lead to further tissue damage. Bacteria also compromise wound healing by competing with the

healing tissue for nutrients and oxygen. Infected wounds should be addressed with adequate débridement, removal of foreign bodies and necrotic tissue, and irrigation. Such measures decrease bacterial burden and optimize host defenses. Infected soft tissues wounds are characterized by erythema, edema, warmth, and tenderness, and a patient with a wound infection may further demonstrate leukocytosis and fever. Fluid collections, abscesses, and hematomas should be drained to avoid bacterial growth. Topical or systemic antibiotics can be administered in conjunction with incision and drainage or débridement when wounds appear infected. Whenever possible, cultures should be obtained prior to initiating empirical antibiotic therapy. Targeted antibiotic therapy should be instituted based on the risk of developing antibiotic-resistant organisms. When appropriate, wound dressings should be used as part of the wound management regimen. Wet to dry (or wet to moist) dressings can be used as a form of wound débridement because a layer of the wound will be removed with each wound dressing change.

Adherence to surgical principles is also crucial for the prevention of postsurgical wound infections.[38] Skin should be sterilely prepped prior to repair of traumatic wounds and before creating surgical incisions. Whenever possible, skin incisions should be made on intact, noncompromised tissue and be closed primarily in layers in a tension-free manner to avoid dehiscence.[1]

DIABETES

Diabetic patients are at an increased risk for compromised wound healing, because poor glucose control brings about inadequate tissue perfusion secondary to microvascular disease. Microvascular disease adversely affects the blood supply of healing tissue, thereby delaying wound healing and rendering diabetics susceptible to wound infection. Release of oxygen to tissues is also reduced, because glycosylated hemoglobin has a higher affinity for oxygen than nonglycosylated hemoglobin. Hyperglycemia also adversely affects the immune system by impairing neutrophil and lymphocyte function, chemotaxis, and phagocytosis. In addition, an uncontrolled blood glucose level decreases red blood cell permeability and decreases blood flow through the small vessels of the wound surface. The combination of poor cell recruitment and wound ischemia creates a suboptimal healing environment.[39]

In the immediate post-traumatic period, a diabetic patient's blood glucose level may prove difficult to control. Those with peripheral neuropathy have decreased pain sensation and are more prone to develop pressure ulcers. Areas vulnerable to injury should be routinely inspected. In the diabetic population, failure to protect the patient from pressure injuries and inadequately addressing existing wounds may result in a devastating limb amputation. Tight serum glucose level control is recommended to improve the likelihood of wound healing.

SMOKING

Smoking tobacco causes tissue hypoperfusion and hypoxia. Tobacco releases chemicals such as nicotine, carbon monoxide, and nitrosamine. Nicotine reduces oxygen delivery to peripheral tissues secondary to vasoconstriction by epinephrine and norepinephrine. Hypoxia is exacerbated by carbon monoxide binding to hemoglobin. Nicotine also causes collagen deposition and prostacyclin formation. It increases platelet aggregation, causes neutrophil dysfunction, and increases blood viscosity, all of which adversely affect wound healing. Prior to elective surgery, patients should be advised to discontinue tobacco use for at least 2 weeks and should refrain from smoking until wound healing is complete.[1,7,20] However, these presurgical precautions are rarely feasible when treating patients with acute traumatic injuries.

MALNUTRITION

Nutritional status is an extremely important consideration in wound healing.[7,40] It is likely the greatest contributor to poor wound healing, especially in older adults. Healthy adults require 35 kcal/kg/day and 0.8 to 2.0 g protein/kg/day. These requirements are greater in injured patients, especially those with large wounds or burns. In hypermetabolic states, protein replacement is 2.5 to 3 g/kg/day in adults and 3 to 4 g/kg/day in children.[2,4] Inadequate protein stores lead to prolongation of the inflammatory phase of healing and promotes protein catabolism. Unless adequately addressed, hospitalized patients' nutritional status can easily drop, particularly in older patients. Patients suffering maxillofacial trauma are at a much greater risk because of their inability to chew and swallow normally. Such patients warrant a dietary evaluation. Modified consistency diets are indicated in patients with chewing or swallowing difficulties. Enteral feeding should be reinstated as soon as possible and the trauma surgeon should have a low threshold for placement of a feeding tube. Nutritional supplements, such as high-calorie or high-protein drinks can help patients with inadequate intake improve their nutritional status. Protein stores should be evaluated by measuring serum albumin and prealbumin levels. The normal serum albumin concentration is higher than 3.5 g/dL and the normal range for prealbumin is 17 to 45 g/dL. Serum prealbumin is a better indicator of short-term nutritional status and a better predictor of wound healing than serum albumin, given its half-life of 2 to 3 days, as compared with the albumin half-life of 20 days.[1]

Vitamin deficiencies are also common in older or malnourished patients and in patients with special dietary requirements, malabsorption syndrome, or chronic alcoholism. Vitamins are needed for normal metabolism and have important roles in many biologic processes.[41-43] For example, vitamin A (retinol and carotenoids) decreases oxidative damage and aids in healing by stimulating fibroplasia, collagen cross-linking, cellular differentiation, and epithelialization. Vitamin B complex aids in antibody formation and leukocytic function, making wounds less susceptible to infection.[44,45] Vitamin C (ascorbic acid) is another antioxidant that aids in wound healing and in boosting the immune system. It is required for appropriate collagen synthesis. Deficiency (scurvy) of vitamin C is associated with the formation of fragile capillaries and a reduced rate of collagen synthesis. Vitamin

D is required for calcium absorption and is crucial for bone repair. Vitamin D deficiency causes rickets in children and osteomalacia and osteoporosis in adults, contributing to an increased risk of fractures. Vitamin E constitutes a family of compounds that includes the tocopherols; it is a potent antioxidant that protects from cell membrane oxidation. Deficiency leads to poor immune response and has been associated with the development of myopathies or neuropathies. Vitamin E (α-tocopherol) has been used in conjunction with pentoxifylline in the management of osteoradionecrosis and bisphosphonate-associated osteonecrosis of the jaws. Vitamin K is vital in the synthesis of prothrombin (factor II) and factors VII, IX, and X of the coagulation cascade. Vitamin K deficiency may lead to increased bleeding and hematoma formation. Deficiency also impairs healing and predisposes to infection. Patients with liver disease or fat malabsorption diseases may require vitamin K supplementation.

A comprehensive review of nonprescription vitamin and herbal supplements is beyond the scope of this chapter. However, surgeons are encouraged to review the patient's medication list, including vitamins, herbal supplements, and alternative medications. Medication reconciliation before embarking in surgery is important because certain vitamins and nutritional supplements may have side effects that can lead to undesired perioperative events, such as increased bleeding and altered metabolism of other medications.[40,46-50]

IMMUNOSUPPRESSION

Patients can be immunosuppressed for a variety of reasons. They may have a medical condition causing immunosuppression or may take medications that induce immunosuppression. Immunosuppression is the hallmark of HIV and AIDS and may be seen in cancer patients and poorly controlled diabetics. Immunosuppressed states can also be encountered in older and malnourished patients and, to a lesser extent, in pregnancy and situations of extreme stress. Medication-induced immunosuppression is seen in transplant recipients and in patients being treated for autoimmune and collagen vascular diseases such as rheumatoid arthritis, systemic lupus erythematosus, Crohn's disease, and ulcerative colitis.[51] Patients with asthma or severe allergic reactions may be taking glucocorticosteroids as anti-inflammatory immunosuppressant drugs. Use of glucocorticosteroids can cause a variety of adverse side effects and complications, such as delayed wound healing, osteoporosis, hypertension, and susceptibility to infection. Glucocorticoids reduce the normal inflammatory response and adversely affect wound healing by suppressing protein synthesis and cell proliferation.[52]

RADIATION AND CHEMOTHERAPY

Radiation therapy induces many deleterious effects in tissue, including hypocellularity, hypovascularity, and hypoxemia. The adverse effects of radiation are dose-dependent. Radiation therapy can have acute and chronic effects. Acute radiation changes in the oral region include mucositis, tissue erythema, and desquamation. Chronic radiation changes are irreversible and are seen in vessel walls and connective tissues of skin and mucosa. Tissues exposed to radiation may be injured permanently because radiation causes irreversible tissue fibrosis and obliteration of small vessels. Postradiation trauma patients are more likely to have wound breakdown; they may require soft tissue flaps to bring blood vessels to inadequately perfused areas to allow for adequate healing to occur.[53,54]

Chemotherapeutic drugs inhibit wound repair.[55] Chemotherapy causes bone marrow suppression, with decreased production of inflammatory cells increasing the risk for infection. Some chemotherapeutic agents target VEGF, an important mediator of angiogenesis. Given its role in tumor angiogenesis and neovascularization, suppressing VEGF is a good target for combating neoplasia. However, this will also have detrimental effects on wound healing.[56]

WOUND MANAGEMENT

Wound care begins by optimizing the healing environment.[1,55,57] Wounds must be properly addressed to optimize healing and avoid complications such as infection, excessive scarring and contracture, tissue maceration, and wound dehiscence. Wounds should be débrided and closed when appropriate. Adherence to surgical principles and observance of wound care standards are crucial. Tissues should be handled with care to avoid additional injury and avoid compromising vascular perfusion. Wounds should not be dessicated or exposed to caustic chemicals. When a wound is closed, it should be done in a tension-free manner. Wounds should be closed in layers, with appropriate suture selection. All dead space should be obliterated and vital structures covered with well-vascularized tissues.[58] Bone fractures should be reduced and adequately immobilized. A patient's medical comorbidities, as well as volume and nutritional status, should be optimized.

Wounds should be initially addressed with adequate débridement.[57] Necrotic tissue and foreign bodies should be removed to decrease the bacterial burden. Hematomas and abscesses should be drained. Infections can be treated with systemic or topical antibiotics or by débridement of infected tissue.[59-61]

Débridement involves the removal of nonvital tissue, foreign bodies, and biofilm.[57] This can be done surgically with sharp excision until viable tissue is reached or hydrodynamically via low-pressure irrigation. Wound irrigation helps decrease bacterial load and washes out foreign bodies and debris. Débridement can also be accomplished with wet to dry (wet to moist) dressings. Other methods include mechanical or chemical débridement by application of topical agents such as silver sulfadiazine, cadexomer iodine, or topical collagenase.[61]

Surgical wounds are described as clean, clean-contaminated, contaminated, or dirty. The type of surgical wound closure performed is dependent on the level of contamination of the wound. For example, clean and clean-contaminated wounds are usually closed primarily, whereas closure of contaminated and dirty wounds is often delayed until the wound has been decontaminated through appropriate débridement or packing. Open

wounds, on the other hand, heal by secondary intention. There are three types of wound healing or wound closure—primary intention, delayed primary closure and secondary intention.

HEALING BY PRIMARY INTENTION OR PRIMARY WOUND CLOSURE

Healing by primary intention occurs in wounds with minimal tissue loss and occurs when the edges of an acute surgical or traumatic wound are approximated. Surgical closure can be done successfully shortly after appropriate wound management. Healing by primary intention results in rapid healing and minimal scarring. Primary wound closure is not recommended in grossly contaminated wounds that cannot be adequately débrided.

DELAYED PRIMARY CLOSURE OR WOUND REPAIR

Delayed primary closure is recommended for wounds that require more extensive decontamination or débridement. The wound edges are apposed only after a period of wound management to optimize healing. A delay in closure may also allow for host defenses to control contamination. As with primary wound closure, the wound edges should be undermined to obtain a tension free closure. Tissue grafts may also be used for wound closure.

HEALING BY SECONDARY INTENTION

Secondary intention is healing by the body's natural mechanisms, without surgical intervention. This is done in large wounds with tissue loss or avulsion, so that wound edges are widely separated and cannot be apposed. Healing occurs by clot formation, granulation, deposition of collagen, and eventual epithelialization. Wound contracture brings the wound margins together. Healing by secondary intention results in very slow healing and notable scarring.

DRESSINGS AND TOPICAL AGENTS

Wound Management: Dressings

Dressings are an important aspect of wound care.[55,62] Dressings are used to maintain a moist environment that encourages faster wound resurfacing by allowing migration of epithelial cells over the moist surface and by preserving growth factors exuded at the wound surface. Dressings can aid in the delivery of topical antimicrobials or recombinant growth factors to the wound bed, protect the wound from friction or shearing trauma, and collect exudate or drainage. Alternatively, they can be used as a form of débridement with frequent dressing changes, especially when used in conjunction with topical agents. Dry dressings are often not recommended because they allow tissues to desiccate easily and form dry scabs. Dressings can be categorized as open (e.g., no dressing with scab formation), semiopen, occlusive, semiocclusive, and biologic. Examples of dressing types include wet to moist gauze, Xeroform or petroleum impregnated gauze (semiopen), Duoderm and hydrocolloid dressings (occlusive), Op-Site or Tegaderm (semiocclusive), and autologous or cadaveric skin grafts (biologic dressings). Skin equivalents, another form of biologic dressings, are also available. AlloDerm, an acellular cadaveric dermal matrix, and Integra, a bovine collagen dermal matrix, can be used when the patient has inadequate or insufficient skin graft donor sites.[55]

In regenerative medicine and tissue engineering applications, living cells and growth factors are added to a scaffold to produce tissue for a bioactive wound dressing.[55,63-68] The cells used can be of autologous or allogeneic origin. The matrix can vary in consistency, depending on the material used—blood, cartilage, or bone. Apligraf is an engineered, bioactive, composite wound healing product that contains epidermal and dermal components. It is made up of living allogenic keratinocytes and fibroblasts, suspended in a bovine collagen matrix, and is approved by the U.S. Food and Drug Administration (FDA) for the treatment of diabetic foot ulcers and venous leg ulcers. Other bioactive wound healing products, such as the Oral LCC or living cellular construct, is currently under FDA study for oral mucosa applications. Human skin equivalents, grown from cultured human infant foreskin, are dermal matrix dressings that contain a layer of live allogenic fibroblasts, covered by a second outer layer of live allogenic keratinocytes. Cultured all-human bilayered bioengineered skin is also under development. VCT01 is an example of an all-human bioengineered skin product that has de novo dermal matrix generated from human dermal fibroblasts.

Negative-Pressure Wound Therapy

Negative-pressure dressings apply subatmospheric pressure to a wound.[55,69-71] The negative- pressure dressing system is comprised of several elements—an open-pore polyurethane foam sponge that can be cut to the size of the open wound, a semiocclusive dressing that adheres to and covers the sponge, tubing that connects the sponge to the vacuum system, and a vacuum pump with a fluid collection canister. A silver-containing sponge can also be used with the polyurethane foam sponge. The negative-pressure dressing should be changed every 24 to 36 hours.

A negative-pressure dressing promotes wound healing by creating a moist wound environment. This is achieved by a pressure gradient that allows for fluid egression from the wound, with excess fluid collected in a vacuum pump canister. This also permits wound exudates, which contain growth factors and cytokines, to travel through the wound bed. In addition to promoting a moist environment, negative-pressure wound therapy increases blood flow into the wound and decreases the wound's inflammatory response.

Topical Agents

Topical agents may be beneficial as an adjunctive wound care treatment.[55,59-61] Some commonly used topical antimicrobials are Bacitracin zinc ointment, cadexomer iodine, and silver sulfadiazine. They help reduce bacterial load within a wound and keep the wound moist. Silver sulfadiazine has broad gram-negative and gram-positive bacterial coverage, whereas Bacitracin zinc ointment has gram-positive coverage. Silver is also toxic to bacteria. These topical applications may be used in conjunction with dressings for gentle wound

TABLE 2-8 Growth Factors—Origins and Actions

Factor	Cells of Origin	Action
PDGF (AA, AB, BB)	Platelets, macrophages, endothelial cells, fibroblasts	Mitogenic for fibroblasts, smooth muscle cells, and endothelial cells; chemotactic for fibroblasts and macrophages
FGF (acidic, FGF1; basic, FGF2)	Fibroblasts	Mitogenic and chemotactic for endothelial cells and keratinocytes; angiogenic
EGF	Multiple	Chemotactic for epithelial cells and fibroblasts. Mitogenic for fibroblasts and epithelial and endothelial cells
VEGF	Epithelial cells, macrophages	Angiogenic
TGF-α	Macrophages, keratinocytes, platelets	Chemotactic for epithelial cells and fibroblasts; mitogenic for fibroblasts and epithelial and endothelial cells
TGF-β	Platelets, macrophages	Chemotactic and mitogenic for fibroblasts; stimulates collagen, fibronectin, and proteoglycan synthesis; angiogenic; and wound contraction
IGF-1, IGF-2	Plasma, fibroblasts, keratinocytes, macrophages	Mitogenic for fibroblasts, keratinocytes, endothelial cells, and smooth muscle cells

Adapted from Lawrence WT: Wound healing biology and its application to wound management. In O'Leary JP, editor: *Physiologic basis of surgery*, ed 3, Baltimore, 2002, Williams & Wilkins.

débridement during dressing changes. Nonadherent silver-impregnated dressings are also available and may be useful in the granulation stage of healing. Sulfamylon, or 10% mafenide acetate, is used for burn wounds and care of exposed cartilage. Topical collagenases, such as Santyl, can be used to débride necrotic wounds with dressing changes enzymatically.

Growth Factors

Wound healing is orchestrated by intercellular communication via chemical signaling within a wound. Growth factors are signaling peptides that are found in wound exudates. They act through specific cell receptors and can cause cellular differentiation, cellular proliferation, and cellular migration. Some of the more well-known growth factors involved in healing are PDGF, TGF-β, EGF, VEGF, basic FGF (bFGF), insulin-like growth factors (IGF-1 and IGF-2), and TNF-α. Growth factors continue to be extensively studied for their potential to accelerate the healing process. Clinically, the use of recombinant growth factors is likely to become an increasingly common practice for improving healing in chronic wounds (Table 2-8).[14]

PDGF is known to play a role in all phases of wound healing. Many cells secrete PDGF, including fibroblasts, endothelial cells, smooth muscle cells, platelets, and inflammatory cells. PDGF is a chemoattractant for neutrophils and macrophages. It promotes chemotaxis and stimulates mitogenesis in fibroblasts and smooth muscle cells. It induces the synthesis of collagen, fibronectin, and hyaluronan. It also increases collagenase activity for the breakdown of necrotic tissue, but has no direct effect on epithelial or endothelial cell function.[72,73] In animal studies, PDGF has even been shown to induce bone regeneration in calvarial defects when implanted on a poly(l-lactide) scaffold.[74] In humans, recombinant PDGF is used to decrease the size of pressure ulcers. Becaplermin, commercially known as Regranex, is a PDGF gel used for the treatment of diabetic foot ulcers. It aids in wound healing by promoting cellular proliferation and angiogenesis.[75] Recombinant PDGF has also been shown to reduce periodontal defects. GEM 21S is an example of a growth factor-enhanced product used to stimulate periodontal wound healing and alveolar bone regeneration. It contains recombinant human PDGF (rhPDGF-BB) within an osteoconductive matrix (beta-tricalcium phosphate [β-TCP]).[76]

bFGF is a potent angiogenic stimulator that is produced by fibroblasts, vascular smooth muscle cells, adrenocortical cells, chondrocytes, and osteoblasts. It aids in tissue repair by stimulating cellular differentiation and proliferation. It promotes neovascularization and mitogenesis and stimulates epithelialization and collagen synthesis. Its role in alveolar bone repair, as well as mandibular and long bone fractures, is well established. Recent animal studies[77] and other reports[78,79] have shown preliminary success of bFGF in the repair of large osteochondral defects.

IGF-1 and IGF-2 are synthesized by various organs, including the liver, heart, lung, kidney, pancreas, cartilage, brain, and muscle. IGFs are mitogens for osteoblasts and osteoblast precursors, thus stimulating bone formation. They also stimulate mitosis in fibroblasts, osteocytes, and chondrocytes. IGFs work synergistically with PDGF in the regeneration of dermal connective tissue and epithelium. IGF-1, when used in combination with TGF-β, has been shown to improve bone healing in healthy and diabetic animals. Dental implants coated with IGF-1 and TGF-β demonstrate an increased bone to implant contact.[80-82]

TGF-β has a role in embryonic development and has also been shown to regulate tissue repair after injury. TGF-β is found in platelet alpha granules that are released at the site of injury on platelet degranulation. TGF-β has chemotactic and mitogenic properties. It promotes osteoblast differentiation and inhibits osteoclastic bone resorption. It has bone-specific properties, but is not as potent as bone morphogenetic protein-2 (BMP-2), a

TABLE 2-9 Growth Factors Found in the Fracture Callus

Growth Factor	Source	Matrix Location	Responding Cells
TGF-β	Platelets, inflammatory cells (monocytes, macrophages, osteoblasts, chondrocytes)	Bone is the most abundant source of TFG-β in the receptors body	Most cells have TFG-β
BMPs	Chondrocytes, urinary bladder, epithelium, brain	BMPs originally identified in bone, but distributed throughout body	Unknown
Fibroblast growth factors	Inflammatory cells, osteoblasts, chondrocytes	Bonds bone and cartilage matrix	Most cells of mesodermal or neuroectodermal origin
Platelet-derived growth factors	Platelets, monocytes, activated macrophages, endothelial cells	Interactions unknown	Most cells of mesodermal origin

TGF-β, Transforming growth factor-β; *BMP*, bone morphogenetic protein.
From Bolander ME: Regulation of fraction repair and synthesis of matrix macromolecules. In Hollinger J, Seyfer AE, editors: *Portland Bone Symposium*, Portland, Oregon, Oregon Health Sciences University, 1995, p 165.

member of the larger TGF-β superfamily. TGF-β also stimulates the synthesis of collagen and extracellular matrix.[83,84]

Growth factors play a crucial role in bone healing (Table 2-9).[14] BMP-2, bFGF, PDGF, TGF-β, and VEGF have all shown positive effects in promoting fracture healing. The BMPs make up a large portion of the TGF-β super family. BMPs induce the expression of osteoblast markers and stimulate bone formation. They also regulate key steps in the differentiation, proliferation, and morphogenic processes of bone and cartilage.[85-87] BMPs are among the most potent of the known regulators of osteoblast differentiation, and BMP-2, 4, 6, and 7 all have osteoinductive properties in vivo.[88-90] BMP-2 released from various carriers has been shown to regenerate calvarial defects completely in the rodent model.[91,92] In humans, BMP-2 recombinant protein can regenerate mandibular continuity defects and alveolar cleft defects and augment maxillary sinuses with results comparable to those of autogenous particulate bone and marrow. Infuse, a commercially available rhBMP-2 used with an absorbable collagen sponge, is FDA-approved for spinal fusion and nonunion of tibia fractures, as well as for maxillary sinus augmentation and grafting of localized alveolar ridge defects.[93-95]

VEGF is the most potent and widely used regulator of vascularization.[56] It has a role in tumor angiogenesis and neovascularization and its concentration increases under hypoxic conditions. It is released from endothelial cells, platelets, megakaryocytes, T lymphocytes, and plasma cells. VEGF has important effects on osteoblasts and osteoclasts during bone repair.[96] Animal studies have shown that VEGF enhances the effects of BMP-2 in the repair of critical sized defects.[97]To improve bone regeneration, other combinations of growth factors have also been investigated to mimic natural temporal and spatial expression more closely.[98] Although PDGF alone is known to increase bone formation, when delayed-release VEGF is added several days later, both new vessel formation and bone regeneration are increased.[99] It is also known that the sequential release of factors such as VEGF and PDGF is more effective for promoting angiogenesis than simultaneous release.[14,100]

SUMMARY

When treating facial injuries, knowledge of the wound healing process is crucial to maximize healing and minimize adverse outcomes such as infection, malunion, and disfiguring scarring. In the trauma arena, oral and maxillofacial surgeons must not only treat acute traumatic wounds appropriately, but must also do everything possible to optimize the wound healing conditions. Knowledge of the technologic advances in wound care, regenerative medicine, and tissue engineering will allow the surgeon treating maxillofacial trauma to achieve the best possible outcome in these potentially devastating facial injuries.

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CHAPTER

3

Nutrition for the Oral and Maxillofacial Surgery Patient

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OUTLINE

Fasting- and Starvation-Induced Malnutrition Physiology
Trauma-, Stress-, and Sepsis-Induced Malnutrition Physiology
Substrate Depletion and Requirements
Protein Requirements
Amino Acids
Electrolyte Requirements
Glucose and Insulin
Assessment Tools for Diagnosis of Nutritional Failure
Clinical Assessment
Laboratory Assessment
Nutritional Support Methods for Patients with Functioning Gastrointestinal Systems
Indications for the Use of Nutritional Therapy
Oral Methods of Nutritional Therapy
Nonoral Methods of Nutritional Therapy
Nasogastric Feeding Tubes
Transcutaneous Enteral Feeding Tubes
Mechanics of Enteral Feeding
Enteral Formula Delivery
Enteral Diets
Polymeric Enteral Formulas
Oligomeric Enteral Formulas
Monomeric Enteral Formulas
Osmolarity
Energy Sources in Enteral Formulas
Complications of Enteral Nutritional Therapy
Parenteral Nutritional Therapy
Peripheral Parenteral Nutrition and Total Parenteral Nutrition
Initiating Nutritional Support in the Critically Ill Patient
Intermaxillary Fixation

Among the many functions the oral cavity provides, one of the most important is the entry of nutrients into the gastrointestinal (GI) tract. The ability of a patient to ingest a normal diet by mouth can be altered by many things, including neoplasia, infection, congenital deformities, and injury. Oral and maxillofacial surgeons provide form- and function-altering surgical procedures to correct these problems, and these procedures themselves may limit function of the oral cavity.

Most patients are well nourished before their visit. However, those patients with chronic illness, alcoholism, or anorexia and those who are older, institutionalized, or homeless may be in various stages of malnutrition. The mortality and morbidity of these malnourished patients is clearly higher than well-nourished patients who have sustained maxillofacial or multisystem trauma, or are undergoing the same operation. Evaluation and correction of malnutrition are time-consuming and not dramatic, yet its correct recognition and application can reduce postoperative complications. This chapter will compare the physiology of slow compensated starvation to the all-consuming hypermetabolism of the critically injured trauma victim. As is true in many clinical areas, the literature can be contradictory and can offer opposing opinions. Some believe that early enteral feeding in trauma patients results in decreased morbidity and complications.[1] Others conclude that nutritional support is currently overused, improperly used, and has failed to show an improvement in clinical outcome.[2]

FASTING- AND STARVATION-INDUCED MALNUTRITION PHYSIOLOGY

A healthy 75-kg man normally stores 200 to 300 g of carbohydrate, equal to 800 to 1200 kcal (4 kcal/g), mostly as glycogen.[3] Fat is normally 15% to 30% (11 to 22 kg) and protein 14% to 20% (10 to 15 kg) of body weight. The average total caloric reservoir is therefore approximately 200,000 kcal, of which 75% is fat. In the complete absence of nutritional intake, an otherwise healthy person could catabolize 1 to 2 g/kg of protein and 2 to 3 g/kg of fat/day. Theoretically, this caloric reserve could sustain life for 3 to 5 months. Realistically, death would occur after burning about 140,000 kcal (75% body fat and 50% body protein).[4]

Starvation involves a cascade of substitution of energy substrates as the body attempts to conserve energy resources and cellular functions. With the onset of starvation, glycogenolysis provides most of the necessary blood glucose. The available glycogen stores are rapidly depleted, and amino acids become the prime source of carbon for hepatic gluconeogenesis. The amino acid sources are muscle, connective tissue, and visceral proteins. As starvation continues, the rate of gluconeogenesis diminishes, coincident with a decrease in metabolic rate and increase in ketone use as fuel by the central nervous system. The early loss of body mass is slowed and accompanied by a decrease in urinary nitrogen excretion. In a healthy patient, nitrogen intake equals

urinary excretion, about 10 to 30 g/day. In fasting starvation, urinary nitrogen excretion falls to 3 to 4 g/day, reflecting protein and therefore amino acid conservation.[4] Because hepatic and GI function are more acutely affected by decreased protein intake, compromised function of both will rapidly develop. One kg of muscle equals roughly 800 kcal, and 1 kg fat equals 7000 kcal, resulting in greater weight loss when muscle is used as an energy source. This is the basis for the correlation of anthropometric measurements with malnutrition (see later).[5]

The brain normally uses glucose as its sole energy source. During starvation, the central nervous system (CNS) converts to the use of fat. Fatty acids cannot cross the blood-brain barrier for use as fuel, but ketones and ketoacids provided by hepatic metabolism of fatty acids can. As starvation continues, the glucose requirement of the CNS falls about 70% and the requirement for gluconeogenesis by the body falls about 50%. This results in significant sparing of lean body mass.

TRAUMA-, STRESS-, AND SEPSIS-INDUCED MALNUTRITION PHYSIOLOGY

Malnutrition can be thought of as a spectrum. One end is the conservative rationing of body energy resources in fasting starvation. The other end is hypermetabolism induced by stress, such as trauma or sepsis. This hypercatabolic state is a neuroendocrine-mediated response to dead tissue, injured tissue, severe perfusion deficits, and invading microorganisms.[6] The components of the response are the CNS, macroendocrine hormones, and microendocrine cytokines.[7] The macroendocrine mediators are the anti-insulin, counterregulatory stress hormones—cortisol, glucagon, and catecholamines. Together, their actions are considered synergistic in this response.[8] Especially in large wounds, such as cutaneous burns, the microendocrine response is mediated on a cellular level by interleukin-1, tumor necrosis factor, and interleukin-6.[9] However, the total degree and severity of the hypermetabolic response cannot be predicted based only on the severity of the injury.

The manifestations of the neuroendocrine response are increased basal metabolic rate, increased core temperature, increased oxygen consumption, hyperdynamic circulation, loss of lean body mass, reduced insulin release, marked lipolysis with reduced hepatic ketogenesis, increased gluconeogenesis unresponsive to glucose administration, increase in lactate and pyruvate production and release, increased amino acid oxidation and efflux from peripheral stores, increased hepatic protein synthesis, and ureagenesis.[7,10] These hypermetabolic responses can be duplicated in normal patients who are given levels of epinephrine, cortisol, and glucagon in concentrations seen in stressed patients.[11] After activation, the hypermetabolic response peaks at 48 to 72 hours. It then subsides over another 3 to 4 days. Loss of important vitamins, trace elements, and minerals occurs very rapidly. Malnutrition will result within days. Complications such as infection will prevent the hypermetabolic response from subsiding. A vicious circle may develop because hypermetabolic patients have increased susceptibility to infection and poor wound healing. The response will once again subside if these complications can be controlled. With repeated episodes or persistent hypermetabolic state, organ failure will result.[7] End-stage organ failure is manifested by liver failure, triglyceride intolerance, rapid rise in lactate levels, reduced amino acid clearance, reduced hepatic protein synthesis, and marked increase in catabolism, with ureagenesis with prerenal azotemia. Once this occurs, it is unresponsive to any nutritional supportive efforts.[12] Table 3-1 is a summary of fasting starvation compared with hypermetabolic catabolism.

TABLE 3-1 Physiologic Summary of Starvation to Hypermetabolism

Characteristic	Fasting Starvation	Hypermetabolic Catabolism
Neuroendocrine activation	–	+
Resting energy expenditure	–	++
Protein catabolism	+	+++
Protein anabolism		
Total body	–	–
Hepatic	+	+++
Amino acid oxidation	+	+++
Ureagenesis	+	+++
Gluconeogenesis	+	+++
Ketone production	++++	+
Malnutrition development rate	+	+++
Simple mathematical total	7	21

(From Cerra FB: Nutrition in trauma, stress and sepsis. In Shoemaker WC, Ayres S, Grenvik O, et al, editors: Society of Critical Care Medicine: Textbook of critical care, ed 2, Philadelphia, 1989, WB Saunders.)

SUBSTRATE DEPLETION AND REQUIREMENTS

Given the spectrum from fasting starvation to hypermetabolic catabolism, the clinician must understand the difference between nutritional and metabolic support, as described by Cerra.[7] In both, the patient's energy and substrate requirements must be met. The proper nutritional support, however, is dependent on how the patient's neuroendocrine axis was activated. In fasting starvation, the hormonal response is activated not by starvation, but by the reintroduction of nutritional support. In stress the hormonal response is activated by the stress insult and subsequent nutritional support. Nutritional support therapy of the stressed hypermetabolic patient is more appropriately termed *metabolic support* because the substrate doses are adjusted to accommodate the metabolic response to injury. Table 3-2[13,14] compares substrate use in starvation to hypermetabolism.

TABLE 3-2 Substrate Use in Starvation Compared with Hypermetabolism

Substrate	Fasting Starvation (Nutritional Support)	Hypermetabolism (Metabolic Support)
Carbohydrate	Maximum usable glucose is 5 mg/kg/min 20 nonprotein kcal/kg/day effective 150:1 nonprotein cal/g nitrogen or greater in diabetics; fat can replace glucose	Resting energy expenditure and oxidation Glucose production is increased, with normal peripheral uptake Glucose entrance into the Krebs cycle is reduced Reduced fraction of total caloric production is from glucose Decreased ability of exogenous glucose to reduce the rate of gluconeogenesis In stress states, the wound itself has an extreme requirement for glucose
Fat	Major long-term energy source in starvation; 10% of energy requirement should be fat Necessary to prevent fatty acid deficiency secondary to insulin inhibition of lipolysis from high glucose infusions 2-3 g/kg/day of fat supplementation can cause hepatic steatosis[7]	75%-90% of energy supplied is by oxidation of fat Plasma changes diagnostic of essential fatty deficiency present from onset of stress Fatty acid intolerance develops in higher levels of stress; important to monitor triglyceride clearance
Protein	Body attempts to spare the amino acid reserve by using other sources of carbon for energy	Decreased total body protein synthesis Increased hepatic protein synthesis Increased catabolism, increased amino acid oxidation (up to 30% of caloric expenditure in high-level stress) Exogenous amino acids ineffective in reducing rate of catabolism Branched-chain amino acids constitute most of the amino acid oxidation Amount of muscle catabolism related to brain's need for glucose, rather than the wound Estimated 40 mg/kg/day nitrogen required to avoid negative balance Resultant lowering of nonprotein calorie-to-nitrogen ratio to 100:1

Adapted from references 7, 13, and 14.

PROTEIN REQUIREMENTS

In an uninjured healthy patient, daily protein losses approximate 20 to 30 g/day. In a critically ill patient in a hypermetabolic state, the protein losses are greater. The net losses can be 1% of the total body protein each day. Therefore, daily protein supplementation should be 1.5 to 2.0 g/kg of ideal body weight. There have been no studies showing that there is benefit to the patient beyond 2 g/kg, no matter the degree of injury and insensible protein losses.

AMINO ACIDS

Glutamine and arginine are semiessential amino acids that are important in times of stress and injury. Glutamine is significantly upregulated and serves as a nitrogen donor important in the formation of ammonia and in the control of acid-base balance by the kidney, as well as other anabolic processes. Glutamine is also an important amino acid for cellular energy—most significantly, for various immunologic cells and enterocytes lining the GI tract. In a hypermetabolic state, glutamine supplementation becomes essential for preventing muscle catabolism and muscle glutamine depletion. Arginine plays an important role in cell division, the healing of wounds, ammonia excretion, immune function, and the release of hormones. Branched-chain amino acids (e.g., leucine, isoleucine, valine) have been shown to have anticatabolic effects in muscle, but there is no evidence that supplementation after injury is beneficial.[15]

ELECTROLYTE REQUIREMENTS

The phosphorus-dependent metabolic pathways are numerous. Significant shifts in phosphorus can critically affect normal body function. The diaphragm is dependent on the energy that phosphorus stores provide; thus, hypophosphatemia can lead to respiratory failure. Total body stores of intracellular electrolytes are significantly depleted in the malnourished state. The clinician should be aware that refeeding will cause shifts of the already depleted electrolytes (phosphorus, magnesium, and potassium) intracellularly, and thus must be monitored closely to avoid cardiovascular complications.

GLUCOSE AND INSULIN

There is no argument that hyperglycemia is detrimental to wound healing and immunologic status. Insulin resistance is common in the critically ill,[16] and tight control of the blood glucose level has been shown to reduce mortality in intensive care unit (ICU) patients.[17]

ASSESSMENT TOOLS FOR DIAGNOSIS OF NUTRITIONAL FAILURE

CLINICAL ASSESSMENT

As in many areas of clinical diagnosis, there are no reliable absolute methods for the measurement of nutritional failure. Most of the methods available are too difficult or tedious to use in the clinical arena. Most produce results no better than a well-considered clinical judgment based on history and physical examination.[18,19] Table 3-3 summarizes the physical findings of malnutrition.[20]

Ideal Body Weight

A medical history and review of systems revealing nausea, anorexia, diarrhea, or weight loss is suggestive of nutritional abnormality. Change in body weight, intentional or unintentional, is important. Normal ideal body weight (IBW) for patients can be calculated by the formulas[21]:

- Healthy males: 106 lb for initial 5 feet, plus 6 lb for every inch over 5 feet, plus 10% if over 50 years old.
- Healthy females: 100 lb for initial 5 feet, plus 5 lb for every inch over 5 feet, plus 10% if older than 50 years.

A decreasing ratio of actual to normal weight reflects increasing malnutrition. Greater than 80% to 90% is mild malnutrition, 70% to 85% is moderate malnutrition, and less than 75% is severe malnutrition.[4] As an alternative, the U.S. National Center for Health Statistics has compiled normal tables of weight as a function of height for men and women (Table 3-4).

Body Mass Index. In 1998, the National Institutes of Health published new guidelines for the determination of healthy weight. These guidelines for defining the point at which someone is overweight or obese are stricter than previous guidelines. The guidelines are based on the body mass index (BMI). The BMI uses a person's height and weight to establish a value that suggests a statistical health risk based on weight alone. The following is used to determine the BMI value:

$$\text{BMI} = \text{body weight (in kg)/height (in m}^2\text{)}$$

A BMI of 26 to 27 is thought to carry moderate health risk and a BMI of 30 increases the risk of death from any cause by 50% to 150%, according to some estimates. The average American woman has a BMI of 26; fashion models typically have BMIs of 18. Conversely, a BMI of less than 18.5 has been proposed as an indication to screen for malnutrition; a BMI less than 15 is also associated with increased mortality.[22]

Anthropometric Measurements. Anthropometric means the study of human body measurements on a comparative basis. When applied to nutrition, anthropometric measurements estimate stores of subcutaneous fat and lean skeletal muscle mass, which are assumed to reflect protein and caloric intake.[23] Because the comparative control data are based on healthy subjects, clinical data may not apply to trauma patients. Subscapular skin fold, triceps skin fold, and midarm muscle circumference are some of the measurements used for nutritional estimates. Some anthropometric measurements have been combined together, or with laboratory data, to create predictive indices. The prognostic nutritional index and the creatinine height index have been correlated with postoperative complication rates.[24]

Energy Expenditure

Estimation of energy expenditure and requirements is the first step in determining the nutritional needs of a patient. The balance between energy intake and expenditure determines the daily energy requirements (calories).[16] Underestimation, and subsequent underfeeding, is associated with delayed wound healing, organ failure, and infection.[25] Overfeeding can produce hyperglycemia, increased carbon dioxide production, respiratory failure, and hepatic steatosis.[26] Energy requirements are dependent on a number of variables, such as body surface area, age, gender, and clinical disease state. Energy expenditure is the body's need for adenosine triphosphate (ATP) to meet metabolic needs. Total energy expenditure (TEE) is the sum of basal energy expenditure (BEE), the thermic effect of food, physical activity, and the disease process itself. BEE is further defined as the energy expended by a patient in the resting, nonstressed state after an overnight fast and is commonly referred to as resting energy expenditure (REE) in hospitalized patients.[27] In situations in which measured energy expenditure is not readily attainable, an estimated caloric requirement may be used (104.67 to 146.54kJ/kg of ideal body weight); however, more sophisticated approaches to measuring requirements exist.[16]

Indirect Calorimetry. The gold standard of assessment of energy expenditure is indirect calorimetry. This technique is expensive, requires trained personnel, and has significant error at higher inspired oxygen concentrations.[28] Indirect calorimetry calculates a patient's energy expenditure by measuring oxygen consumption and carbon dioxide production. It can be performed in spontaneously breathing and mechanically ventilated patients; however, inaccuracy occurs in patients with an inspired oxygen concentration higher than 60%.[27] This method measures REE and reflects the clinical situation at the time the measurement is made; it may not reflect true energy expenditure over a 24-hour period.[7,19]

$$\text{Energy expenditure} = (\text{cardiac output} \times \text{V}_{\text{O}_2}) + (1.11)(\text{V}_{\text{CO}_2})$$

Estimating energy requirements is necessary in clinical practice when indirect calorimetry is impractical.[29] Several standardized prediction equations to measure energy expenditure have been developed, with the intent to estimate energy requirements accurately in the absence of indirect calorimetry. Each has its limitations, and none have been shown to correlate statistically with indirect calorimetry on a consistent basis.

Harris-Benedict Equation. This equation was published 85 years ago and was developed through the analysis of indirect calorimetry obtained from healthy, young, lean body mass male and female volunteers. This equation,

TABLE 3-3 Nutrition-Focused Physical Examination

System	Normal Findings	Abnormal Findings	Possible Nutritional or Metabolic Associations	Non-Nutritional Examples
general survey	weight for height appropriate, well-nourished, alert, and cooperative	loss of excess weight, muscle mass and fat stores, growth retardation	protein-calorie deficiency	endocrine disorders, osteogenic disorders, menopausal disorders secondary to estrogen depletion
		excess fat stores	excess calorie intake	
		fatigue, anemia	iron deficiency	
skin	pink, soft, moist, turgor with instant recoil, smooth appearance	poor wound healing, ulcers	protein, vitamin C, or zinc deficiency	diabetes, steroids
		dry with fine lines and shedding, scaly (xerosis)	essential fat or vitamin A deficiency	environmental or hygiene factors
		spinelike plaques around hair follicles on buttocks, thighs, or knees (follicular hyperkeratosis)	vitamin A or essential fat deficiency	
		pellagrous dermatitis (hyperpigmentation of skin exposed to sunlight)	niacin or tryptophan deficiency	thermal, sun, or chemical burns; Addison's disease
		pallor	iron or folic acid deficiency	skin pigmentation disorders, hemorrhage
		yellow pigmentation	carotene excess	jaundice
		poor skin turgor	fluid loss	
		petechiae, ecchymoses	vitamin K or C deficiency	aspirin overdose, liver disease, or trauma
nails	smooth, translucent, slightly curved nail surface and firmly attached to nail bed; nail beds with brisk capillary refill	spoon-shaped (koilonychia)	iron deficiency	COPD, heart disease, aortic stenosis
		dull, lackluster	protein or iron deficiency	chemical effects
		pale, mottled	vitamin A or C deficiency	infection, chemical effects
scalp	pink, no lesions, tenderness; fontanels without softening, bulging	softening of craniotabes	vitamin D deficiency	
		open anterior fontanel (usually closes by 18 months of age)	vitamin D deficiency	hydrocephalus
hair	natural shine, consistency in color and quantity, fine to coarse texture	lack of shine and luster, thin, sparse	protein, zinc, biotin, or linoleic acid deficiency	hypothyroidism, chemotherapy, psoriasis, color treatment
		easily plucked	protein, zinc, or biotin deficiency	hypothyroidism, chemotherapy, psoriasis, color treatment
		color change	zinc deficiency	chemically processed or bleached hair
		brittle hair	biotin deficiency	
		hair loss	protein, B_{12}, or folate deficiency	
face	skin warm, smooth and dry, soft moist with instant recoil	diffuse depigmentation, swollen	protein deficiency	steroids and other medications
		pallor	iron, folate, or B_{12} deficiency	low perfusion, low volume states
		moon face	protein-calorie deficiency	Cushing's disease
		bilateral temporal wasting	protein-calorie deficiency	neuromuscular disorders

TABLE 3-3 Nutrition-Focused Physical Examination—cont'd

System	Normal Findings	Abnormal Findings	Possible Nutritional or Metabolic Associations	Non-Nutritional Examples
eyes	evenly distributed brows, lids, lashes, conjunctiva pink without discharge, sclerae without spots, cornea clear, skin without cracks or lesions	pale conjunctiva	iron, folate, or B_{12} deficiency	
		night blindness	vitamin A deficiency	
		dry, grayish, yellow, or white foamy spots on whites of eyes (Bitot's spots)	vitamin A deficiency	pterygium, Gaucher's disease
		dull, milky, or opaque cornea (corneal xerosis)	vitamin A deficiency	
		dull, dry rough appearance to whites of eyes and inner lids (conjunctival xerosis)	vitamin A deficiency	chemical, environmental
		softening of cornea (keratomalacia)	vitamin A deficiency	
		cracked and reddened corners of eyes (angular palpebritis)	riboflavin or niacin deficiency	infection, foreign objects
nose	uniform shape, septum slightly to left of midline, nares patent bilaterally, mucosa pink and moist, able to identify smells	scaly, greasy, with gray or yellowish material around nares (nasolabial seborrhea)	riboflavin or niacin, pyridoxine deficiency	
		inflammation, redness of sinus tract, discharge, obstruction or polyps		need to reconsider if placing feeding tube
lips, mouth	pink in color, symmetrical, smooth, intact	bilateral cracks, redness of lips (angular stomatitis)	riboflavin, niacin, pyridoxine, iron, or protein deficiency	poor fitting dentures, herpes, syphilis
		vertical cracks of lips (cheilosis)	riboflavin, niacin, iron, or protein deficiency	AIDS (Kaposi's sarcoma), environmental exposure
tongue	pink color, moist, midline, symmetrical with rough texture	magenta in color	riboflavin deficiency	
		smooth, slick, loss of papillae (atrophic filiform papillae)	folate, niacin, riboflavin, iron, or B_{12} deficiency	
		beefy red color, atrophied taste buds, and mucosa red and swollen	niacin, folate, riboflavin, iron, B_{12}, or pyridoxine deficiency	Crohn's disease, infection
		decreased taste (hypogeusia)	zinc deficiency	cancer therapy
gums	pink, moist without sponginess	spongy, bleeding, receding	vitamin C deficiency	dilantin and other medication, poor hygiene, lymphoma, polycythemia thrombocytopenia
teeth	repaired, no loose teeth, color may be various shades of white	missing, poor repair, caries, loose	excess sugar	trauma, syphilis, aging, poor dental hygiene, radiation therapy
		white or brownish patches (mottled)	excess fluoride	enamel hypoplasia, erosion
parotid gland	located anterior to earlobe, no enlargement	bilateral enlargement	protein deficiency	bulimia, cysts, tumors, hyperparathyroidism
neck nodules	trachea midline, freely movable without enlargement or nodules	enlarged thyroid	iodine deficiency	cancer, allergy, cold, infection

Continued

TABLE 3-3 Nutrition-Focused Physical Examination—cont'd

System	Normal Findings	Abnormal Findings	Possible Nutritional or Metabolic Associations	Non-Nutritional Examples
chest, lungs	anterior and posterior thorax: adequate muscle and fat stores, respirations even and unlabored, symmetrical rise and fall of chest during inspiration and expiration, lung sounds clear	somatic muscle and fat wasting, labored respirations, adventitious breath sounds such as crackles, rhonchi, and wheezing; evaluate for fluid status vs. tenacious secretions that may labor breathing and increase energy expenditure. Also consider increased rate and depth, decreased rate and depth	protein-calorie deficiency, metabolic acidosis, metabolic alkalosis	respiratory disease (e.g., COPD)
heart	rhythm regular and rate within normal range; S1 and S2 heart sounds heard	irregular rhythm	potassium deficiency or excess, calcium deficiency, magnesium deficiency/excess, or phosphorus deficiency	cardiopulmonary disease states
		pounding pulse	fluid overload (hypervolemia)	cardiopulmonary disease
		small, weak pulse	fluid deficiency (hypovolemia)	
		palpitations	hypoglycemia	
		tachycardia	thiamine deficiency	
		enlarged heart	thiamine deficiency associated with anemia and beri-beri	
abdomen	soft, nondistended, symmetrical, bilateral without masses, umbilicus in midline, no ascites, bowel sounds present and normoactive; tympanic on percussion; feeding device intact without redness, swelling	generalized symmetric distention	obesity	enlarged organs, fluid, or gas
		protruding, everted umbilicus, tight glistening appearance (ascites)	influence protein, fluid, sodium concerns of feeding	
		scaphoid appearance	protein-calorie deficiency	
		increased bowel sounds	influences nutrition of gastroenteritis (normal if hunger pains)	
		high-pitched tinkling	influences nutrition if intestinal fluid and air present indicating early obstruction	
		decreased bowel sounds	influences nutrition if peritonitis, or paralytic, ileus present	
kidney, ureter, bladder	urine golden yellow (range from pale yellow to deep gold), clear without cloudiness, adequate output	decreased output, extremely dark, concentrated	dehydration	

TABLE 3-3 Nutrition-Focused Physical Examination—cont'd

System	Normal Findings	Abnormal Findings	Possible Nutritional or Metabolic Associations	Non-Nutritional Examples
musculoskeletal	full range of motion without joint swelling or pain, adequate muscle strength	inability to flex, extend, and rotate neck adequately	influences nutrition by interfering with ability to feed or make hand-to-mouth contact	
		decreased range of motion, swelling, impaired joint mobility, or other of upper extremities; muscle wasting on arms, legs, skin folding on buttocks	protein-calorie deficiency	
		swollen, painful joints	vitamin C	connective tissue disease
		enlargement of epiphyses at wrist, ankle, or knees	vitamin D or C deficiency	trauma, deformity, or congenital cause
		bowed legs	vitamin D deficiency or calcium deficiency	
		beading of ribs	vitamin D deficiency or calcium deficiency	renal rickets, malabsorption
		pain in calves, thighs	thiamine deficiency	deep vein thrombosis, other neuropathy
neurologic	alert, oriented, hand-to-mouth coordination; no weakness or tremors	decreased or absent mental alertness; inadequate or absent hand-to-mouth coordination	influences nutrition by the ability to feed or make hand-to-mouth contact	
		psychomotor changes, confusion	protein deficiency	trauma, neurologic disease
		peripheral neuropathy	thiamine, pyridoxine, or B_{12} deficiency	
	cranial nerves intact: primary nutritionally focused ones include trigeminal, facial, glossopharyngeal, vagus, and hypoglossal	tetany	calcium or magnesium deficiency	
	reflexes (biceps, brachioradialis patella, and Achilles' common in exam), functioning within normal range of 2++	hyperactive reflexes	hypocalcemia (R/O tetanus, upper motor neuron disease)	
	hypoactive reflexes	hypokalemia	associated with metabolic diseases such as diabetes mellitus and hypothyroidism	
		hypoactive Achilles', patellar reflex	thiamine or B_{12} deficiency	neurologic disorders

COPD, Chronic obstructive pulmonary disease.
Adapted from Hammond K: Physical assessment: A nutritional perspective. *Nurs Clin North Am* 32:779, 1997.

TABLE 3-4 U.S. National Center for Health Statistics—Male and Female Ideal Weight

	18-24 YR		25-34 YR		35-44 YR		45-54 YR		55-64 YR	
Height	M	F	M	F	M	F	M	F	M	F
4'10"		114		123		133		132	135	
4'11"		118		126		136		136	138	
5'00"		121		130		139		139	142	
5'01"		124		133		141		143	145	
5'02"	130	128	139	136	146	144	148	146	147	148
5'03"	135	131	145	139	149	146	154	150	151	151
5'04"	139	134	151	142	155	149	158	153	156	154
5'05"	143	137	155	146	159	151	163	157	160	157
5'06"	148	141	159	149	164	154	167	160	165	161
5'07"	152	144	164	152	169	156	171	164	170	164
5'08"	157	147	168	155	174	159	176	168	174	167
5'09"	162		173		178		180		178	
5'10"	166		177		183		185		183	
5'11"	171		182		188		190		187	
6'00"	175		186		192		194		192	
6'01"	180		191		197		198		197	
6'02"	185		196		202		204		201	

therefore, underestimates the energy requirements of trauma patients. Modifiers were developed to account for this; however, it is thought that the modifiers significantly overestimate requirements.[16,30,31] Table 3-5 illustrates some stress factor modifiers.

Males:

$$BEE = 66 + (13.7)(\text{weight in kg}) + (5)(\text{height in cm}) - (6.8)(\text{age in years})$$

Females:

$$BEE = 655 + (9.6)(\text{weight in kg}) + (1.7)(\text{height in cm}) - (4.7)(\text{age in years})$$

Ireton-Jones Equation. Another predictive equation is the Ireton-Jones equation.[32] Two hundred patients with a variety of medical and surgical diagnoses were measured by indirect calorimetry. Ventilator-dependent patients had a higher REE than spontaneously breathing patients.[27]

The Ireton-Jones equation for spontaneously breathing patients is as follows:

$$EEE = 629 - 11(A) + 25(W) - 609(O)$$

The Ireton-Jones equation for ventilator-dependent patients is as follows:

$$EEE = 1784 - 11(A) + 5(W) + 244(G) + 239(T) + 804(B)$$

where A = age (years), W = weight (kg), O = presence of obesity (>30% above ideal body weight; absent = 0, present = 1), T = diagnosis of trauma (absent = 0, present = 1), B = diagnosis of burn (absent = 0, present = 1), and EEE = estimated energy expenditure (kcal/day).

These and several other equations have been proposed to estimate energy requirements, but these equations have shown poor predictive value, have been formulated from subjects who are not representative of the general population, or have large standard errors for the estimate. However, these equations continue to be used in clinical practice despite a lack of evidence or supporting research.[29]

TABLE 3-5 Stress Factor Modifiers for Estimating Energy Expenditure

Injury	Factor
Simple starvation	0.85
Elective surgery	1.05-1.15
Sepsis	1.2-1.4
Closed head injury	1.3
Multiple trauma	1.4
Major burn	2.0

Data from Malone AM: Methods of assessing energy expenditure in the intensive care unit. *Nutr Clin Pract* 17:21, 2002.

LABORATORY ASSESSMENT

Nitrogen Balance

The ultimate goal of any nutritional support regimen is the provision of positive nitrogen (N) balance. Therefore, it is necessary to assess the adequacy of nitrogen delivery and loss. One of the more common methods involves the 24-hour collection of urine for the

determination of urinary nitrogen loss. In the absence of abnormal nitrogen loss from intestinal fistulas, diarrhea, and wound exudates, total nitrogen loss may be calculated. In general, a positive nitrogen balance greater than 2 is satisfactory. A balance less than 2 requires an additional protein supplement to the diet. A positive nitrogen balance is difficult to achieve in the critically injured patient, and is simply not attainable when severe hypermetabolism is present.[16]

Visceral protein reserves can be estimated by the measurement of specific serum proteins.[5]

The following are equations for nitrogen output and balance:

Nitrogen output:

24-hour urine urea nitrogen (UUN) (g/day)
= UUN (mg/day) × urine output (mL/day)
× 1 g/1000 mg × 1 dL/100 mL

Total nitrogen loss (g/day) 24-hour UUN (g/day)
+ (0.2 × 24-hour UUN g/day) + 2 g/day

Nitrogen balance:

24-hour intake protein (g)/6.25 (g)
– urinary nitrogen (g/day) = N/day

Serum Albumin

Albumin is not stored in the liver but is continuously secreted by the hepatocytes at approximately 17 g/day. Albumin levels fall slowly in starvation because of their long half-life. Once hepatic synthesis is slowed, the rate of serum decline of a protein is inversely proportional to its half-life.[5] Thus, serum proteins with shorter half-lives, such as prealbumin, transferrin, ceruloplasmin, and retinol-binding protein, respond to dietary changes much faster. Table 3-6 summarizes levels for some of these proteins.

These short half-life secretory proteins provide more sensitive indication of acute protein and energy depletion, but have not been shown to make a significant difference in the prediction of outcome.[16] Clinical studies have shown a direct correlation between low albumin levels and 30-day hospital mortality.[33] Gibbs et al, in a multicenter Veterans Administration (VA) prospective study of 54,215 noncardiac surgery patients found that a decrease in serum albumin from concentrations greater than 46 g/liter to less than 21 g/liter was associated with an exponential increase in mortality rates from less than 1% to 29% and in morbidity rates from 10% to 65%. The study concluded that albumin levels are a strong predictor of surgical mortality and morbidity, especially the risk of sepsis and major infections.[34] The clinician must be aware that serum albumin can be significantly altered with excessive protein losses, catabolism, decreased hepatic protein synthesis, and fluid resuscitation, creating dilutional effects; especially in the multisystem trauma victim.

NUTRITIONAL SUPPORT METHODS FOR PATIENTS WITH FUNCTIONING GASTROINTESTINAL SYSTEMS

In oral and maxillofacial surgery patients who are not nutritionally compromised, mild and transient nitrogen losses are easily tolerated. Nitrogen losses can readily be replaced by oral feeding. Recovery from moderate injury or uncomplicated elective surgery increases the metabolic rate only by about 10%. In the absence of hypermetabolism, nitrogen losses are minimal. If a patient is estimated to return to oral intake within a week, it is appropriate to provide only 5% dextrose solutions (500 kcal/day) as the only supplement. Additional nutritional support in such patients does not further improve outcome.[2]

However, the clinician will encounter the hypermetabolic, critically ill patient who will require aggressive nutritional support. In these patients, IV administration of only 5% dextrose with electrolytes is equivalent to starvation.[4] Nitrogen excretion can reach 200 to 400 mg/kg/day in stressed patients receiving only 5% dextrose. Ten days of such losses will result in severe malnutrition in a previously well-nourished patient.[35] Without proper supplementation, outcome has been shown to be worse in major surgery patients unable to eat for 14 days. As a threshold, nutrition supplementation should be given to previously well-nourished patients not expected to eat for 1½ to 2 weeks after injury or surgery.[2]

INDICATIONS FOR THE USE OF NUTRITIONAL THERAPY

Souba, in a review and meta-analysis of clinical trials, has described indications for the use of nutritional therapy.[2] Established indications are as follows: patients unable to eat or absorb nutrients for an indefinite period of time; well-nourished, minimally stressed patients unable to eat for more than 10 to 14 days; severely malnourished patients undergoing major elective surgical procedures; and patients with major trauma and bone marrow transplant recipients undergoing intensive anticancer therapy.

TABLE 3-6 Hepatic Protein Markers

			MALNUTRITION (G/DL)		
Protein	Half-Life (days)	Normal	Mild	Moderate	Severe
Albumin	20	3.5-5.0	2.8-3.5	2.1-2.7	<2.2
Prealbumin	2-3	16-43	10-15	5-9	<5
Transferrin	8	200-400	150-200	100-149	<100

Unproved indications for the use of nutritional therapy include cancer, AIDS and other immunodeficiencies, GI tract dysfunction, liver failure, renal failure, and critically ill patients (excluding trauma).

ORAL METHODS OF NUTRITIONAL THERAPY

Oral feedings using a functioning GI tract are the optimal route for nutritional therapy. Problems that may interfere with or limit adequate oral feeding include poor appetite, nausea and vomiting, and inability to swallow. Oral and maxillofacial surgery patients are especially vulnerable to oral dysfunction secondary to diseases, deformities, injuries, and the nature of the procedures used to treat them. Oral dietary supplementation (ODS) seems to be an attractive and easy nutritional therapy, but definitive proof of its efficacy is lacking. MacFie et al,[36] in a randomized, prospective, clinical trial of 100 patients undergoing laparotomy, found no significance between controls and ODS patients regarding weight loss or clinical outcome. They concluded that the routine use of perioperative ODS in well-nourished patients undergoing major GI surgery provides no clinical or functional benefit.

NONORAL METHODS OF NUTRITIONAL THERAPY

Nutritional support methods that bypass the oral cavity include enteral (tube feeding) and parenteral (IV feeding). Enteral feeding requires a functioning GI tract, whereas parenteral support is specifically used when the GI tract is nonfunctional. Sometimes, a patient's unique needs may be satisfied by the use of both methods concurrently. Enteral feeding has several advantages over parenteral feeding. Enteral feeding maintains gut structure and function by providing the gut mucosa with substrates essential for epithelial integrity. It does not require a central venous catheter for administration; therefore, there are fewer infectious or metabolic complications. Enteral feeding is less costly than parenteral support. For equivalent nutritional supplementation, parenteral support will cost $75 to $350, whereas enteral feedings will cost only $18 to $30/day.[37] Contraindications to enteral feedings include ileus, initial short bowel syndrome, intestinal obstruction beyond the duodenum, intractable vomiting and diarrhea, acute GI bleeding, intestinal ischemia, and severe inflammatory bowel disease.[38] Enteral feedings should be initiated after initial resuscitation from shock. Bowel necrosis has been reported following the delivery of enteral nutrition to patients in profound metabolic acidosis accompanying uncompensated shock. Figure 3-1 demonstrates an enteral feeding method algorithm.

NASOGASTRIC FEEDING TUBES

Nasogastric feeding tubes are small-diameter polyurethane or silicone tubes.[39] They can have weighted or nonweighted tips and removable internal stylets or none. These tubes are designed for only 4 to 6 weeks of use or less. Do not confuse these with the larger stiffer tubes that are only for temporary stomach decompression. All tubes have radiopaque stripes for tube location verification on radiographs and length verification markings on the side of the tube.

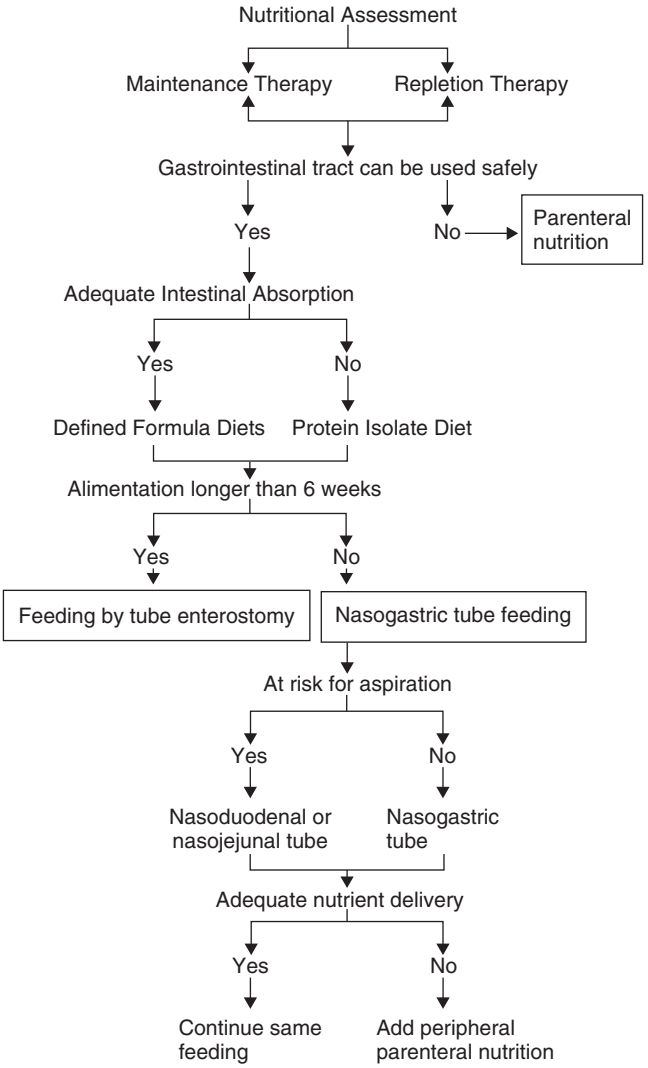


FIGURE 3-1 Enteral feeding method algorithm. (From Guenter P, Jones S, Ericson M: Enteral nutrition therapy. *Nurs Clin North Am* 32(4):651, 1997.)

Nasogastric Tube Insertion

Every clinician has his or her own favorite methods for nasogastric tube insertion. General guidelines are as follows[40]:

- Place the patient in an upright or semiupright position.
- For conscious patients, topically anesthetize the nasopharynx.
- Use caution with rigid stylet tubes because they can puncture hollow viscera.
- Lubricate the tube well with a water-soluble lubricant.
- Have the patient swallow some water to facilitate tube passage through the esophageal opening; use caution with aspiration risk in dysphagia patients.
- Clinically verify correct tube placement by auscultation during insufflation of air, aspiration of stomach contents, or irrigation with small volume of water.
- Secure the tube to the nose with tape without exerting pressure on the nasal alae.

- Confirm tube position by abdominal radiography.
- A 45-degree head of bed elevation will reduce reflux and aspiration during tube feeding.[41]

Verification of correct tube placement in the stomach, and not in the lungs, can involve a variety of maneuvers. The most traditional method, auscultation of air insufflation, is not reliable. Clinical studies have revealed that this method is unreliable in differentiating GI versus respiratory tract placement.[42] Air insufflation does prove the tube is not kinked. Aspiration of fluid and pH testing of aspirates have also proven to be unreliable in clinical trials.[43] Abdominal radiographs verifying the positioning of the nasoenteric tube remain the gold standard. Correct interpretation of these x-rays is important, with attention to differentiating the nasogastric tubes from the many other tubes that a critically ill patient may have,. For patients with a significant risk of aspiration, postpyloric placement is recommended. Weighted nasogastric feeding tubes spontaneously enter the duodenum only 15% of the time.[46] To improve passage into the duodenum, place the patient in the right side–down decubitus position. Administration of promotility agents such as metoclopramide or erythromycin before the nasogastric feeding tube is inserted will increase motility of the upper GI tract while relaxing the pylorus. These agents are not effective if administered after the tube is inserted in the stomach. A 90% postpyloric passage has been achieved with unweighted tubes using this method.[47] Endoscopic or fluoroscopic assistance can also be used to facilitate postpyloric passage. Repeat x-rays should be obtained after episodes of emesis, violent coughing, or any other factor that might cause displacement of the tube.[44]

Nasogastric Tubes in Patients with Midface or Skull Fractures

Caution should be exercised when passing nasogastric tubes in patients with midface or base of skull fractures. In these patients, the passage course should parallel and track along the floor of the nose. An alternative is first to insert a soft nasal airway and use this as a guide for passage of the nasogastric feeding tube through it. The nasal airway can then be removed once the nasogastric feeding tube is in place.[45] Be cautious with patients who have existing tracheotomy or endotracheal tubes because the feeding tubes have a propensity to follow these tubes into the trachea.[7]

TRANSCUTANEOUS ENTERAL FEEDING TUBES

Surgically placed enteral feeding tubes are indicated when the anticipated needs of the patient for nutritional support are longer than 4 to 6 weeks. This includes open or laparoscopic gastrostomy, surgically placed jejunostomy tubes, percutaneous endoscopic gastrostomy (PEG), and percutaneous endoscopic jejunostomy.

Percutaneous Endoscopic Gastrostomy

The most popular option is the PEG.[48] A gastrostomy can also be performed with fluoroscopic or endoscopic assistance or the traditional open approach. Moller et al[49] has compared 147 gastrostomy procedures performed with fluoroscopic or endoscopic assistance or the traditional open procedure. The results were a 30-day postoperative, procedure-related mortality of 3.2%, 0%, and 2.9%, respectively. Procedure-related morbidity was 16%, 8%, and 20%%, respectively.

Advantages of PEG. The advantages of PEG compared with traditional open gastrostomy are no laparotomy procedure or scar, decreased peritoneal adhesions, shorter anesthesia time, less postoperative pain, may be done as an outpatient procedure, immediate feeding, decreased expense, decreased complication rate, and decreased stomal leakage. The primary indications for PEG in head and neck surgery include neoplasia, neurologic dysphagia, cancer cachexia, and esophageal and pharyngeal obstruction. Contraindications to PEG include previous gastric surgery, morbid obesity, peritonitis, massive ascites, peritoneal dialysis, sepsis, inability to transilluminate the abdominal wall, poor gastric emptying, high risk of aspiration, and anatomic restrictions on endoscopy.[50]

PEG Procedure. The following is a brief description of the PEG procedure. The reader should consult general surgery texts and literature for a complete discussion.

After induction of a general anesthetic and tracheal intubation, the patient is placed in a reverse Trendelenburg position. This allows the migration of abdominal contents toward the pelvis. The upper left abdominal quadrant is prepared for a sterile procedure. A flexible video gastroscope is inserted through the esophagus into the stomach. The stomach is then insufflated to approximate the visceral wall of the stomach to the parietal wall of the peritoneal cavity. The lights of the operating room are turned off to allow the light of the gastroscope to transilluminate the gastric and abdominal walls. External digital palpation and indentation of the illuminated spot on the abdominal wall, usually several centimeters under the left costal margin, is confirmed through the gastroscope. A 16-gauge angiocatheter is inserted percutaneously at the transillumination spot, and its entry into the stomach is confirmed with the gastroscope. A wire loop or suture is passed through the catheter and retrieved in the stomach by the gastroscope's snare. The gastroscope with the wire or suture is removed through the mouth. The wire loop or suture is then attached to the external end of a gastrostomy tube. A 1-cm incision is made in the abdominal wall catheter puncture site to release the dermis. The gastrostomy tube is pulled by the wire or suture through the mouth and stomach to emerge at the small skin incision. A flange is attached to the tube at the skin level for tube retention. The stomach and tube are examined by reinsertion of the gastroscope, followed by stomach decompression.

PEG Complications. A review of four articles, encompassing a total of 637 PEG procedures, has revealed a minor complication rate of 7% to 13% and a major complication rate of 2% to 4%.[51-54] Minor complications included superficial wound infection, transient ileus, stomal leaks, accidental dislodgment of the tube, pneumoperitoneum, and clogged lumens. Major complications included gastric perforations, gastric bleeds, aspiration followed by pneumonia, peritonitis, esophageal injury, colonic perforation, and gastric erosion. A particularly disastrous complication for head and neck surgeons is oral or pharyngeal tumor transplantation to the PEG stoma site on the abdominal wall. There

have been eight reported cases of head and neck squamous cell carcinoma appearing at the PEG stoma. These seeding episodes were primarily in patients with bulky hypopharyngeal, laryngeal, or esophageal tumors.[55,56]

Harbrecht et al compared hospital costs for 6 years of PEG procedures versus traditional open gastrostomy. They concluded that both have equivalent complication rates (a view not supported by other investigators), but significantly less cost was associated with PEG when performed in the ICU or endoscopy suite.[57] PEG gastrostomy is also appropriate for pediatric cases, with documentation in patients as young as 2.4 months.[58] The mean useful life span of PEG tubes is approximately 4 months.[59] Current design of PEG tubes allows simple traction removal followed by dressing placement in an ambulatory clinic. PEG tubes should not be removed until the stoma has matured, which is approximately 6 weeks at the earliest. PEG gastrostomy should therefore not be done indiscriminately but only if the need exists for prolonged enteral nutritional support.

MECHANICS OF ENTERAL FEEDING

ENTERAL FORMULA DELIVERY

Delivery of enteral formulas into the stomach can be intermittent or continuous. Intermittent, bolus feeding using manual syringes or gravity feeding is more psychological, with less chance of contamination. Continuous delivery using a volumetric pump is mandatory when the feeding tube tip is in the small bowel. Interestingly, bolus feedings increase the REE of the patient by 8% to 10%, but continuous infusion does not significantly increase REE over the fasting level.[60] Volumetric pumps can also assist when the stomach is the enteral destination by decreasing gastric distention and associated risks of emesis and aspiration.[61] Highly stressed patients with gastric atony have a high incidence of aspiration. Jejunostomy, either surgically placed or endoscopically assisted (e.g., a PEG/PEJ arrangement), works well in these situations, even in the presence of mild ileus.[7] Regardless of the delivery method, all enteral feeding tubes require frequent flushing with water to maintain patency. Recommendations include flushing every 4 hours with continuous feedings, and before and after intermittent feedings or medication delivery.[62]

ENTERAL DIETS

Enteral formulas can be broadly classified according to the type of protein they contain. Three basic categories are intact protein (polymeric), hydrolyzed protein (oligomeric), and crystalline amino acids (monomeric).[45]

POLYMERIC ENTERAL FORMULAS

Polymeric enteral formulas are considered nutritionally complete because they contain sufficient fat, protein, carbohydrate, minerals, vitamins, and trace elements to prevent deficiencies when administered to meet the caloric requirements of the patient.[45] Most polymeric formulas contain 1 kcal/mL, but some concentrated formulas contain up to 2 kcal/mL. Concentrated formulas are used for patients requiring fluid or volume restriction or for patients who cannot tolerate the volume necessary to meet energy requirements with the less concentrated formulas.

The normal distribution of macronutrients in polymeric formulas is 40% to 60% carbohydrate calories, 30% to 40% fat calories, and 14% to 18% protein calories. The proteins in polymeric formulas are mostly high-biologic value milk or soy protein isolates. Use of these formulas may not be suitable for patients with malabsorption, because they may not be able to hydrolyze them. Carbohydrates are the main source of energy in normal diets and in polymeric enteral formulas. Derivatives from the hydrolysis of cornstarch are the source of these carbohydrates. Sucrose is sometimes added for sweetness. Lactose is not included in most nutritional products secondary to the prevalence of lactose intolerance. Fat calories are derived from vegetable oils alone or in combination with medium-chain triglycerides from coconut or palm oils. Vegetable oils contribute the long-chain fatty acids and the essential fatty acid linoleic acid, whereas the medium-chain triglycerides contribute calories.

OLIGOMERIC ENTERAL FORMULAS

Oligomeric enteral formulas contain protein that has been hydrolyzed into dipeptides, tripeptides, oligopeptides, and free amino acids. These peptides do not require enzymatic digestion and can be absorbed directly by gut mucosal cells. Unless a patient has GI dysfunction that compromises the ability to absorb proteins, there is no advantage to prescribing an oligomeric enteral formula.

MONOMERIC ENTERAL FORMULAS

Monomeric enteral formulas contain free, crystalline L-amino acids. The free amino acids are poorly absorbed and may contribute to gut mucosal atrophy. The indications for prescribing monomeric enteral formulas are limited.[63]

OSMOLARITY

Osmolarity is the concentration of an osmotic particle–containing solution per liter of the solution. A formula is usually considered hyperosmolar if its osmolarity exceeds that of blood (300 mOsm/liter). The oligomeric and monomeric formulas usually are hyperosmolar; the polymeric formulas are mainly iso-osmotic. Enteral osmolarity can affect patient tolerance of the formula and use of hyperosmolar formulas can result in diarrhea. Some polymeric enteral formulas contain fiber. Fiber is added for the beneficial physiologic effects on bowel function, such as prevention of diarrhea or constipation.

ENERGY SOURCES IN ENTERAL FORMULAS

Glucose and fat are the major sources of energy in enteral formulas. In the hypermetabolic critically ill patient, the normal response to exogenous nutrients found in starvation is absent.[7] Unlike in fasting starvation, exogenous nutrients do not decrease hepatic gluconeogenesis and lipolysis and have little effect on muscle and visceral

proteolysis.[64] In stress states, increased carbon dioxide levels, hepatic necrosis, hepatomegaly, and reactivation of the neuroendocrine axis can result from excess administration (>500 g/day) of glucose.[7,40] Adults should receive about 100 to 150 g/day of glucose, with sliding scale insulin or insulin drips used to maintain the serum blood glucose level between 100 and 140 mg/dL.[65]

Table 3-7 indicates that starving patients treated with nutritional resuscitation will tolerate greater than 150 nonprotein cal/g of nitrogen. Stressed patients requiring metabolic resuscitation will need no more than 100 nonprotein cal/g of nitrogen.[7] Stressed patients require more protein calories than carbohydrate calories. Fat can be administered in amounts of 1 to 3 g/kg/day in enteral nutritional therapy. Patients should receive about 1.0 to 2.5 g of protein/kg/day, depending on existing nutritional status, disease state, trauma, planned surgical procedures, and presence or absence of multisystem organ failure. Protein intake should be restricted in patients with renal failure unless dialysis is initiated.[40]

Vitamin and mineral requirements are difficult to quantitate in starvation and stress states. General recommendations are to administer 1 mL in children younger than 1 year old and 15 mL in adults of a multivitamin preparation per day with enteral feedings.[40] Vitamin C deficiency produces defective collagen cross-linking. Vitamin A deficiency also produces defective collagen cross-linking and affects cell morphology and ground substance production. Vitamin E functions as an antioxidant and as a free radical scavenger.[8] Nathens et al have documented that early administration of antioxidant vitamin supplementation reduces organ failure, shortens ICU stay, and speeds recovery when administered to trauma patients.[66]

TABLE 3-7 Stress Level Determinate of Nonprotein Calorie/Nitrogen Ratio

Stress Level	Example	Nonprotein/Nitrogen Ratio
0	Starvation	>150:1
1	Elective surgery	100:1
2	Multiorgan trauma	100:1
3	Sepsis/burns	80:1

(From Hickey MS: Nutritional management of the critically ill patient. In Weigelt JA, Lewis FR, editors: *Surgical critical care*, Philadelphia, 1996, WB Saunders.)

COMPLICATIONS OF ENTERAL NUTRITIONAL THERAPY

Enteral nutritional therapy may result in technical, functional, or metabolic complications.[30] Of enterally fed patients, 5% have technical complications, 15% to 30% have functional complications, and 5% have metabolic abnormalities. Box 3-1 lists enteral nutritional therapy complications in these three general categories. Table 3-8 summarizes the physiologic and metabolic complications from enteral nutritional therapy.

BOX 3-1 Enteral Nutritional Therapy Complications

TECHNICAL
- Abscess or erosion of nasal septum
- Acute sinusitis from obstruction of midface sinus drainage orifices
- Aspiration pneumonia
- Bacterial contamination of formula
- Esophageal ulceration, distal stenosis or erosion
- Gastrointestinal perforation
- Gastrostomy, jejunostomy dislodgment
- Hemorrhage
- Hoarseness
- Inadvertent tracheobronchial intubation
- Intestinal obstruction
- Intracranial passage of tube
- Knotting of tube
- Laryngeal ulceration
- Nasal alar rim erosions
- Necrotizing enterocolitis
- Otitis media
- Pharyngolaryngeal inflammation
- Pneumatosis intestinalis
- Rupture of varices
- Skin excoriation

FUNCTIONAL
- Abdominal distention
- Constipation
- Diarrhea
- Nausea
- Vomiting

METABOLIC
- Dehydration
- Essential fatty acid deficiency
- Hyperglycemia
- Hyperkalemia
- Hypernatremia
- Hyperosmolar nonketotic coma
- Hyperphosphatemia
- Hypocupremia
- Hypoglycemia
- Hypokalemia
- Hypomagnesemia
- Hyponatremia
- Hypophosphatemia
- Hypozincemia
- Liver function test elevation
- Overhydration
- Vitamin K deficiency

TABLE 3-8 Complications of Enteral Nutrition Therapy

Complication	Treatment
Diarrhea	Rule out infectious diarrhea (e.g., *C. difficile*) Check the actual serum albumin (ASA) level. If the ASA level is <2.5 g/dL, calculate the albumin deficit (AD) per the Andrassy formula: AD = (2.5 = ASA) × 0.3 ×10 × wt kg Then administer 25 g of 25% albumin intravenously q6h until the calculated albumin deficit is replaced. Recheck the serum albumin level. If the serum albumin level is ≥2.5 g/dL, no further albumin therapy is necessary. If the level is <2.5 g/dL, recalculate the albumin deficit and administer additional albumin intravenously until the serum albumin is ≥2.5 g/dL. or Hespan, 250 mL/day × 1 to 2 days Administer antidiarrhea agents: Deodorized tincture of opium (DTO) 15-20 drops PO or per nasointestinal tube q4-6h prn or Lomotil 1-2 capsules (2.5-mg/tablet) PO q4-6h or 5-10 mL (2.5 mg/5 mL) per nasointestinal tube q4-6hr prn or Imodium 1-2 capules (2 mg/capsule) PO q4-6h or 10-20 mL (1 mg/5 mL) per nasointestinal tube q4-7h prn or Sandostatin 50-350 μg SC or IV q6h prn If nos. 1 and 2 fail to treat the diarrhea, discontinue the current enteral diet and begin an oligomeric protein formula. If nos. 1, 2, and 3 fail, discontinue enteral nutritional therapy and begin parenteral nutritional therapy.
Residual (>150 mL)	Examine the patient and rule out the possibility of either a mechanical intestinal obstruction or paralytic ileus. Confirm the position of the feeding tube in the small bowel per kidney-ureter-bladder radiograph. Check the serum potassium and calcium levels. Consider a pharmacologic cause.
Hyperglycemia (>160 mg/dL)	Reduce the oral glucose intake and administer regular insulin subcutaneously per a sliding scale regimen. If this fails to maintain the serum glucose ≤160 mg/dL, consider an insulin drip. It may be necessary to reduce the rate of the enteral feedings temporarily until blood glucose control is obtained and then slowly advance back to goal rate.
Hypoglycemia (<70 mg/dL)	Administer (immediately) 1 ampule of D50W intravenously and then recheck the serum glucose level. If the serum glucose level remains <70 mg/dL, administer additional intravenous D50W until the serum glucose is ≥ 70mg/dL. If the patient continues to require intravenous glucose supplements, discontinue the current full-strength enteral diet and begin a new full-strength diet that has a greater glucose content at the previous infusion rate.
Hypernatremia (>145mEq/liter)	Evaluate need for increasing or decreasing water intake. If needed, reduce or discontinue all oral and intravenous sodium intake. If this fails to maintain the serum sodium to ≤145 mEq/liter, discontinue the current full-strength enteral diet and begin a less concentrated infusion of the current diet or begin a new full-strength diet that has a lower sodium content at the previous infusion rate.
Hyponatremia (<135 mEq/liter)	Evaluate need for increasing or decreasing water intake. If needed, administer additional sodium using NaCl tablets or salt packets until the serum sodium level is >135 mEq/liter. Laboratory testing to exclude SIADH.
Hyperkalemia (>5 (mEq/liter)	Discontinue all oral and intravenous potassium intake. If this fails to maintain the serum potassium level at ≤5 mEq/liter, discontinue the current full-strength enteral diet and begin a new full-strength diet that has a lower potassium content at the previous infusion rate.
Hypokalemia (<3.5 mEq/liter)	Administer additional potassium orally or intravenously until the serum potassium level is ≥3.5 mEq/liter. If the patient continues to require excessive oral or intravenous potassium supplements, discontinue the current full-strength enteral diet and begin a new full-strength diet that has a higher potassium content at the previous infusion rate.
Hyperphosphatermia (>4.5 mEq/liter)	Discontinue all oral and intravenous phosphate intake. If this fails to maintain the serum phosphate level to ≤4.5 mEq/liter, discontinue the current full-strength enteral diet and begin a new full-strength diet that has a lower phosphate content at the previous infusion rate.
Hypophosphatemia (<2.5 mg/dL)	Administer additional phosphate orally as NeutrA-Phos 1-2 packets up to 4 times per day, or intravenously as sodium or potassium phosphate to maintain the serum phosphate ≥ 2.5mg/dL. (Note: The daily intravenous phosphate dosage should not exceed 60 mM.)

TABLE 3-8 Complications of Enteral Nutrition Therapy—cont'd

Complication	Treatment
Hypermagnesemia (>2.7 mg/dL)	Discontinue all intravenous and oral magnesium intake. If this fails to maintain the serum magnesium level ≤2.7 mg/dL, discontinue the current full-strength enteral diet and begin a new full-strength diet that has a lower magnesium content at the previous infusion rate.
Hypomagnesemia (<1.6 mg/dL)	Administer additional magnesium orally or intravenously to maintain the serum magnesium level ≥ 1.6 mg/dL. Consult the *Physicians' Desk Reference* for the various enteral and parenteral magnesium preparations.
Hypercalcemia (>10.5 mg/dL)	Discontinue all oral and intravenous calcium supplements. If this fails to maintain the serum calcium level ≤ 10.5 mg/dL, discontinue the current full-strength enteral diet and begin a new full-strength diet that has a lower calcium content at the previous infusion rate.
Hypocalcemia (<8.5 mg/dL)	Administer additional calcium orally as Titralac or intravenously as calcium gluconate, 10-30 mEq daily to maintain the serum calcium level ≥ 8.5 mg/dL.
High serum zinc (>150 µg/dL)	Discontinue all oral and intravenous zinc intake until the serum zinc level is ≤150 µg/dL.
Low serum zinc (<55 µg/dL)	Administer additional zinc orally as zinc sulfate 200 mg three or four times daily or intravenously as elemental zinc 2-5 mg daily to maintain a serum zinc level ≥ 55 µg/dL.
High serum copper (>140 µg/dL)	Discontinue all oral and intravenous copper intake until the serum copper level is ≤140 µg/dL.
Low serum copper (<70 µg/dL)	Administer additional copper orally or intravenously as elemental copper 2-5 mg daily to maintain a serum copper level > 70 µg/dL.

(Adapted from Hickey MS: Nutritional management of the critically ill patient. In Weigelt JA, Lewis FR, editors: *Surgical critical care*, Philadelphia, 1996, WB Saunders.)

Small-diameter nasogastric feeding tubes tend to clog, especially when used as a portal for medication delivery. Instillation of water, meat tenderizer, sodium bicarbonate, and digestive enzymes has been described in attempts to dissolve clogs in enteral feeding tubes. Use of low-pH liquids, such as carbonated beverages or citrus juices, tends to precipitate proteins further and worsen feeding tube blockage. The following steps for unclogging a feeding tube have been recommended by Lord[44]:

- Aspirate as much fluid as possible with a 30- to 60-mL syringe attached to the enteral device. Discard the fluid.
- Refill the syringe with 5 mL of warm water and instill the water into the enteral tube using manual pressure for 1 minute, using a back and forth motion with the syringe plunger.
- Clamp the tube for 15 minutes and again try to aspirate fluid or flush with warm water.
- If this fails, repeat the procedure with a commercial solution marketed to unplug enteral feeding tubes.
- Mechanical reaming devices should be used only with great caution because they can easily perforate the tube and/or hollow organs.

Diarrhea is a common complication of enteral nutritional therapy. Various causes have been postulated, including intrinsic bowel disease, intestinal bacterial overpopulation, mechanical intestinal dysfunction, polypharmacy, osmolarity of the diet, and hypoalbuminemia.[30] Typically, the cause is assumed to be hyperosmolarity of the enteral formula, and the feeding is stopped or diluted. Keohane et al randomly assigned patients to hyperosmolar and iso-osmolar enteral nutritional therapy.[67] Patients in the hyperosmolar arm were further randomly assigned to a diluted or nondiluted initial feeding regimen. The investigators discovered that not only was there no effect of osmolarity or dilution on incidence of diarrhea, but adequate nutritional supplementation was delayed by deliberate dilution.

The most common cause of diarrhea associated with enteral nutrition therapy is medication. Many elixir medications commonly administered to enterally fed patients contain sorbitol. A 500-mg dose of acetaminophen elixir may contain as much as 5.47 g of sorbitol.[67] Twenty g of sorbitol will cause diarrhea in most patients. Edes et al used the calculation of stool osmotic gap compared with stool osmolarity to determine whether diarrhea in enterally fed patients is related to medications. They concluded if the stool osmotic gap was more than 140 mOsm/liter, osmotic medications or additives should be suspected. If the stool osmotic gap was less than 140 mOsm/liter, nonosmotic causes are likely.[68]

PARENTERAL NUTRITIONAL THERAPY

Parenteral nutrition therapy is indicated when the patient has severe bowel dysfunction or cannot absorb required calories and supplementation orally, enterally, or both. Parenteral nutrition therapy is contraindicated in patients with normal bowel function.

PERIPHERAL PARENTERAL NUTRITION AND TOTAL PARENTERAL NUTRITION

Parenteral therapy has two forms, peripheral parenteral nutrition (PPN) and total parenteral nutrition (TPN). The main difference between PPN and TPN is the route of delivery. PPN is delivered through a large-bore peripheral IV catheter; TPN requires a central venous catheter.

PPN is indicated when parenteral supplementation is required for 10 days or less. Because PPN is administered

through a peripheral vein, the osmolarity of the solution is limited to 1150 mOsm/liter. PPN solutions contain 20% dextrose and must be supplemented with fat emulsions. Fat then becomes the major source of calories.

TPN is indicated for the hypermetabolic, critically ill patient with bowel dysfunction. It is also indicated for the less severely ill patient if it is estimated that enteral intake will be delayed beyond 7 to 10 days. TPN solutions typically contain more concentrated glucose solutions, amino acids, and fat emulsions. A minimum of 2% to 4% of the total kilocalories should be administered as lipid to avoid essential fatty acid deficiency. The resultant hyperosmolar solution requires a central venous line for delivery.

Complications of TPN include central line infections, hyperglycemia, and electrolyte disorders. To prevent the latter complication, daily adjustment of electrolytes in the solution may be required. Vitamins are added to the solution daily. Insulin may also be added to the solution to help control serum glucose levels.

INITIATING NUTRITIONAL SUPPORT IN THE CRITICALLY ILL PATIENT

Although no randomized clinical trials exist regarding when to initiate feeding in the trauma patient, it is well accepted that nutritional support is necessary to attenuate the hypermetabolic response to injury, reduce the rate of infection, and maintain the integrity of the intestinal mucosa along with its immunologic defenses.[16] Early feeding (<72 hours) in the hemodynamically stable ICU patient is associated with a decrease in infection and a decrease in the length of hospital stay, without a significant difference in morbidity.

INTERMAXILLARY FIXATION

One of the most frequently performed oral and maxillofacial surgery procedures that interfere with oral nutrition is intermaxillary fixation (IMF). Patients with IMF face formidable dietary obstacles. IMF has been used as a weight loss technique in place of, preceding, or after traditional bariatric surgery. In the literature, IMF for weight loss is applied for a longer time than its traditional use after fractures or after elective maxillary and/or mandibular surgery. IMF for weight loss implies starvation by default.

However, in several obesity studies, IMF was applied for 1½ months to 18 months. Mean weight loss across all studies was 8 to 30 kg, with more weight loss in males than females.[69-71] Most of the weight was regained after IMF release; therefore, IMF alone is an unsuitable treatment for weight loss.[72] It would seem that IMF is not enough of an impediment to oral intake to result in nutritional deficits. Worrall[73] has compared weight loss in 13 patients with IMF to 9 patients with rigid fixation for orthognathic or facial fracture surgery. Six weeks after surgery, there was a 4.5 kg total weight loss for the IMF group and 1.1 kg total weight loss for the rigid fixation group. Routine orthognathic and facial fracture repair procedures, with or without IMF, do not seem to result in nutritional compromise for the short time they are used. However, additional studies are needed to determine whether premorbid or preoperative nutritional status has an effect on outcomes in patients who have altered diets or IMF following maxillofacial surgery.

SUMMARY

In summary, the physiologic response to starvation is markedly different than that seen in the hypermetabolic, critically ill patient. Nutritional assessment must examine the preexisting nutritional state and the degree of ongoing hypermetabolism. While a healthy, well-nourished patient may tolerate 7 to 10 days of inadequate oral intake, both the malnourished and the hypermetabolic patient may require early nutritional support, preferably by the enteral route. It is the responsibility of the surgeon to identify patients requiring such support and to initiate a suitable nutritional support regimen.

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PART TWO

Systematic Evaluation of the Traumatized Patient

CHAPTER 4

Initial Assessment and Intensive Care of the Trauma Patient

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OUTLINE

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PATIENT TRANSPORT

HISTORY OF MEDICAL TRANSPORTATION

Prior to the 1600s, several methods of moving the debilitated were in use around the world. In what would become the United States, Native Americans used the travois (Fig. 4-1), which was essentially a stretcher affixed at one end to a horse or large dog to pull as a conveyance for material or disabled people. In Egypt, camel stretchers called panniers, which were used in many regions around the world with other beasts of burden such as mules, were the method of choice, even through the Napoleonic period, to move the nonambulatory.

In the fifteenth century, King Ferdinand and Queen Isabella of Spain were instrumental in the deployment of *ambulancia* (mobile field hospitals) to provide rapid medical aid to their soldiers. In January 1777, George Washington instructed the army's Hospital Department Director General, Dr. John Cochran, to consult with another well-known physician of the day and his successor, Dr. William Shippen, to reorganize the medical system, including the flying hospitals, to accompany his armies in the field. Flying hospitals were equivalent to today's MASH units (mobile army surgical hospitals). They were semitransient field hospitals that were moved with the regiment or divisional army, providing immediate care to those wounded after a battle and to those who were ill.

The next significant development of U.S. emergency medical services (EMS) and medical transport took place during the Civil War (1861-1865). The Union Medical



FIGURE 4-1 A travois, which was essentially a stretcher affixed at one end to a horse or large dog to pull, was used by Native Americans to transport material or the injured.





FIGURE 4-2 **A,** The Union Medical Department during the Civil War implemented the use of committed, customized, horse-drawn wagons as ambulances. **B,** Stretcher litters and pack animal cacolets.

Department implemented the use of committed, customized horse-drawn wagons as ambulances (Fig. 4-2A) as well as stretcher litters and pack animal cacolets (see Fig. 4-2*B*). In addition, a dedicated group of stretcher bearers and ambulance wagon attendants and drivers was formed. They received specialized training by the medical department and a tiered transport system was developed. On March 11, 1864, President Lincoln signed a law that was passed by Congress, "An Act to Establish a Uniform System of Ambulances in the Armies of the United States." This legislation established a standardized system of ambulance service throughout the military. The law also mandated the use of special uniforms for the ambulance corps and special signs for the ambulances. Regulations issued during the war by both sides, and incorporated into this law, also conventionalized specific insignia and signage for the recognition of ambulances and hospitals.

In addition to litters, cacolets, and wagons, trains and boats were also used as medical transportation vehicles on a regular basis. Ambulance wagons took the wounded and sick from field hospitals to trains or hospital ships instead of directly to the general hospital. This would occur if the general hospital was too far to allow for reasonable transport by wagon, train, or ship. Thus, a stratified system of medical transport and transport vehicles developed.

The first civilian-manufactured ambulance in the United States, a specialized medical transport vehicle, was built in 1890 by the Hess-Eisenhardt Company of Cincinnati, Ohio. It was a horse-drawn wagon specifically designed to move the incapacitated in need of medical care. Shortly thereafter, the first motorized ambulance was made in Chicago and donated to the Michael Reese Hospital by five local businessmen in 1899. This was quickly followed by St. Vincent's Hospital of New York, which began operating an automobile ambulance in 1900. In 1910, the first known aircraft ambulance, a plane modified to carry a patient lying down, was built in North Carolina and tested in Florida. It failed shortly following take-off and crashed after flying only 400 yards in Fort Barrancas, Florida. By 1929, the U.S. Army Air Corps had been organized and designed three planes to perform as ambulances. They were built and equipped to carry two patients on stretchers, a pilot, and an attendant.

World War II, the Korean Conflict, and the Vietnam War all brought advances in trauma medicine to the military, which were then used in the civilian sector. Some improvements in military EMS organization and operations occurred; the most noteworthy was the use of helicopters to retrieve critically injured patients rapidly from the battlefield, transporting them to field hospitals (MASH units). Reducing the time from injury to surgical intervention, with on-scene advanced medical treatment such as the administration of IV fluids by nonphysicians, marked a milestone in the progression of clinical care.

A convergence of several landmark events occurred in the mid-1960s that altered the foundations of EMS in the United States, commencing a new era of modernity and organization. For decades, a crescendo had been building regarding the carnage experienced from automobile accidents on the nation's highways. Finally, in 1961, President John F. Kennedy noted that traffic accidents were one of the greatest of the nation's public health problems. Instantly, a new focus was established on the need for emergency medical aid for motor vehicle accident (MVA) victims.

Several crucial events took place during President Johnson's administration. First, in 1965, Medicare was created by an act of Congress. In the original legislation,

ambulance transportation was recognized as a covered beneficiary service. In so doing, the federal government established a long-term funding mechanism for EMS and medical transportation. The introduction and propagation of prehospital medical care, especially advanced clinical treatment, would not wait for government regulations. Often with no legislation regarding their activities, U.S. physicians were spearheading the use of medications, defibrillators, and other advanced medical treatment modalities in the field, at the scene of incipient need. Most of these services started with a single-minded focus of cardiac emergencies, but rapidly expanded into treating other medically urgent conditions. The emphasis was shifting from the rapid recovery and transport of victims to the rapid response of specialized personnel and apparatus, and the stabilization of patients before movement to a hospital.

The first advanced life support ambulance in the United States was used by St. Vincent's Hospital in New York. The first nonphysician, mobile, advanced medical treatment service in America began in Miami in 1968. Dr. Eugene Nagel blended the training of surrogate quasiphysicians with radiotechnology to devise the concept of a paramedic using telemetry communication to receive real-time medical commands from a physician at the hospital. This service was followed shortly thereafter by similar programs in Columbus, Ohio, Jacksonville, Florida, Seattle, and Los Angeles. By the end of the 1970s, EMS was firmly established in the medical infrastructure of the United States as its own discipline, with its own science. During the next several decades, it would become more sophisticated, evolving into an industry.

TYPES OF TRANSPORT

Helicopter

Helicopters offer several advantages over other transport vehicles (Fig. 4-3). They can travel at speeds of 120 to 180 mph, allowing for transport times to be up to 75% shorter when compared with ground transport. They can avoid traffic delays and ground obstacles and fly into locations inaccessible by other modes of patient transport. Helicopters have landing requirements, which is a disadvantage when compared with ground transport, but they can access more regions than fixed-wing transport. Helicopter cabins are not pressurized and, as a result, patients being transported are at some risk for barotrauma. Another disadvantage is that in most transport programs, helicopters are only permitted to fly under visual flight rules. Hence, weather conditions can limit this operation. However, some programs are now implementing instrument flight rules, which give greater flexibility to flying in less than ideal weather.[5]



FIGURE 4-3 Rescue helicopters allow for transport times to be up to 75% shorter when compared with ground transport. (From Day MW: Transport of the critically ill: The Northwest Medstar experience. *Crit Care Nurs Clin North Am* 17:183, 2005.)

The widespread use of helicopters for patient transport has led to increased accident rates (three times higher) when compared with general helicopter aviation.[6] Another study has demonstrated a lower accident rate among busier flight programs and programs that implement instrument flight rules.[7] Helicopter ambulances are best used when hospital ground transport time is expected to be longer than 35 minutes or when ground transport is not a viable option. Helicopter transport of critically injured patients from remote areas may be lifesaving; however, there is a large potential for its misuse. The annual cost of helicopter transport service can be at least $1 million for an institution. For effective use of this transport modality, it is generally recommended that helicopter transport should be integrated into the regional EMS system, staffed to provide advanced life support, and used based on medical need.

Severely injured patients transported by helicopter from the scene of an accident are more likely to survive than patients brought to trauma centers by ground ambulance, according to a recent study. This study was the first to examine the role of helicopter transport on a national level and includes the largest number of helicopter transport patients in a single analysis. The finding that helicopter transport positively affects patient survival comes amid an ongoing debate surrounding the role of helicopter transport in civilian trauma care in the United States, with advocates citing the benefits of fast transport times and critics pointing to safety, use, and cost concerns. The new national data show that patients selected for helicopter transport to trauma centers are more severely injured, come from greater distances, and require more hospital resources, including admission to the intensive care unit (ICU), use of a ventilator to assist breathing, and urgent surgery compared with patients transported by ground ambulance. Despite this, helicopter transport patients are more likely than ground transport patients to survive and be sent home following treatment.

Air medical transport is a valuable resource that can make trauma center care more accessible to patients who would not otherwise be able to reach these centers. The study included patients transported from the scene of an injury to a trauma center by helicopter or ground transportation in 2007. The team used the National Trauma Databank to identify 258,387 patients; 16% were transported by helicopter and 84% were transported by ground. The helicoptertransport patients were younger, more likely to be male, and more likely to be victims of MVAs or falls compared with ground transport patients. Overall, almost 50% of the helicopter transport patients

were admitted to the ICU, 20% required assistance in breathing for an average of 1 week, and almost 20% required surgery. Even though they were more debilitated when they arrived at the hospital, they ultimately fared better than those transported by ground.

Although the study has shown that air transport does make a difference in patient outcomes, there are no data available to explain why patients transported by helicopter do better than those transported by ground. The authors assumed that the speed of transport—helicopters are capable of higher speeds over longer distances, regardless of terrain—and the ability of air medical crews to provide therapies and use treatment modailities not universally available to ground unit crews are the main drivers of positive patient outcomes. However, the study has some limitations. It is not possible to evaluate the many factors that drive the decision to transport a patient by helicopter in all cases. In addition, the general nature of the data set limits specific conclusions that could be applied to any individual trauma system.

Helicopter transport has been an integral component of trauma care in the United States since the 1970s, mainly because of the military's experience in transporting sick or injured soldiers during wartime. The availability of helicopters in the civilian setting has been credited with improving trauma center access for a significant percentage of the population.

Fixed-Wing Air Ambulance

This mode of patient transport is usually implemented for distances longer than 150 miles. One of the biggest disadvantages is that fixed-wing aircraft require landing strips and airports. This type of service is ideal for long distance transport of donors, organs, and patients to specialized institutions, such as burn and transplantation centers. Various types of fixed-wing aircraft are available for patient transport. Light single-piston or twin-piston engine planes are typically unpressurized and provide minimal room for patient care. These aircraft generally fly at speeds of 100 to 160 mph. Medium-range aircraft (600 to 1200 miles), which may be powered by pistons or turboprops, are usually pressurized and have a speed of 200 to 300 mph. Small jets have pressurized cabins, have the longest range (1500 to 2500 miles), and can travel at speeds of 400 to 550 mph. Pressurized cabins help prevent the development of barotrauma in the patient.

INITIAL ASSESSMENT

Trauma annually affects hundreds of thousands of individuals and costs billions of dollars in direct expenditures and indirect losses. Trauma care has improved over the past 20 years, largely from improvements in trauma systems, assessment, triage, resuscitation, and emergency care.

The American College of Surgeons Committee on Trauma (ACS-COT) and the American Association for the Surgery of Trauma (AAST) acute care surgery initiative is designed to integrate trauma, emergency general surgery, and surgical critical care and to bolster new trainee interest in this field. The ACS-COT applies rigorous standards to performance improvement prior to verifying U.S. trauma centers. For this improvement to occur, the ongoing application of the unique principles and practice of intensive care medicine is necessary. Patient outcomes after major trauma have improved in regions in which comprehensive trauma systems have evolved. Crucial components of such a system should include a coordinated approach to prehospital and hospital care and to training providers in both areas. Paramedics and medical staff should be provided with a clear and objective framework for assessing patients, establishing and engaging treatment protocols, following triage guidelines, engaging in transportation, and using communication protocols.

The accurate and systematic assessment of injury is essential to establish the extent of injury to vital structures. This forms the basis of Advanced Trauma Life Support (ATLS) protocols. It is estimated that approximately 25% to 30% of deaths caused by trauma can be prevented when a systematic and organized approach is used.[8] Trauma patients should undergo an initial assessment and treatment that is prioritized and corresponds to their injuries and stability of their vital signs. When critical injuries are present, lifesaving measures necessitate that a logical and sequential treatment priority be established, based on the overall assessment of the patient.

Trauma injuries can generally be divided into three categories—severe, urgent, and nonurgent.

- Severe injuries are those that are an immediate threat to life because they interfere with vital physiologic functions. These severe injuries make up approximately 5% of all patient injuries, but represent more than 50% of all trauma deaths.
- Urgent injuries constitute 10% to 15% of all injuries and are not an immediate threat to life. These patients will usually require surgical intervention, but they have stable vital signs.
- Nonurgent injuries account for the remaining 80% of all trauma cases. These do not constitute an immediate threat to life. These patients will generally require medical or surgical intervention after significant evaluation and/or observation.

The main goal of the initial assessment of the trauma patient is to recognize the patient who does have life-threatening injuries, establish treatment priorities, and manage them aggressively.

ASSESSMENT PRINCIPLES

These principles are involved in the initial assessment of a patient with major trauma and have been outlined by the American College of Surgeons (ACS) in their guidelines regarding ATLS protocols.[43] These principles are as follows:

1. Preparation and transport
2. Primary survey and resuscitation, including monitoring and radiography
3. Secondary survey, including special investigations, such as CT scanning or angiography
4. Ongoing reevaluation
5. Definitive care

Preparation and Communication

A variety of EMS systems exist based on the level of need of a community or jurisdiction. Prehospital medicine exists in two levels of care, basic life support and advanced life support services. Services are provided by emergency medical technicians (EMTs) who are trained at these two different levels of care.[9]

Hospitals receiving trauma patients should receive advance communication from emergency medical services care providers about the impending arrival of seriously injured patients. The patient's mechanism of injury, vital signs, field interventions, and overall status should be communicated. This allows for the in-house trauma team to be called and for the emergency department staff to make appropriate preparations.

On the patient's arrival, a concise but comprehensive transfer of the patient from the paramedics should occur. One person should be talking while everyone else is listening. In many trauma centers, the team leader is a senior or chief resident in surgery or emergency medicine, with close supervision from appropriate attending staff. Most trauma centers use a system of prehospital triage that categorizes patients into those with physiologic derangements and those who have a suggestive mechanism of injury. Those with obvious derangements should prompt a full team response, whereas patients with less injury may be cared for by a modified team complement.

Hospital Phase

Once the trauma patient arrives to the hospital, it is of paramount importance to pay attention to every detail. This could mean the difference between life and death.

A fully equipped resuscitation area must be available. The equipment has to be organized, tested, and stored where it is immediately accessible. Airway equipment (e.g., laryngoscopes, endotracheal tubes, suction, tracheostomy and cricothyrotomy kits, bougies) and IV resuscitation equipment (e.g., warmed IV crystalloid solutions, different gauge IV needles, central line and arterial line kits) need to be readily available. Adequate monitors and radiologic and laboratory resources are an integral part of this phase. Transfer agreements between institutions have to be established and operational.

Protective precautions are mandatory for the personnel who have contact with the patient (e.g., gowns, face mask, eye protection, gloves, shoe covers or boots). The Centers for Disease Control and Prevention (CDC) consider these to be minimum precautions. Standard precautions are also included in Occupational Safety and Health Administration (OSHA) requirements in the United States.

Trauma Scoring

Trauma scoring systems describe injury severity and correlate with survival probability. Various systems facilitate the prediction of patient outcomes and the evaluation of aspects of care. The scoring systems vary widely, with some relying on physiologic scores (e.g., Glasgow Coma Scale [GCS] score, Revised Trauma Score), and others relying on descriptors of anatomic injury (e.g., Abbreviated Injury Score, Injury Severity Score). No universally accepted scoring system has been developed; each system has unique limitations. These limitations have resulted in the use of a various scoring systems in different centers worldwide.

TABLE 4-1 Glasgow Coma Score

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4 = Spontaneous	5 = Normal conversation	6 = Normal
3 = To voice	4 = Disoriented conversation	5 = Localizes to pain
2 = To pain	3 = Words, but not coherent	4 = Withdraws to pain
1 = None	2 = No words only sounds	3 = Decorticate posture
	1 = None	2 = Decerebrate
		1 = None

Total = E + V + M

Glasgow Coma Scale. This scale is commonly used and was developed by Teasdale and Jennet. The Glasgow Coma Scale (GCS) was the first system to attempt to quantify the severity of a head injury.[10] There are three variables used with the scale—best motor response, best verbal response, and eye opening. The key to calculating the GCS is to determine the best response in each category. The motor response is used to assess the level of central nervous system (CNS) function, whereas a verbal response shows the ability of the CNS to integrate information. Eye opening will demonstrate brainstem activity. Each category is given a score, as outlined in Table 4-1, with total scores ranging from 3 to 15. Higher scores represent increased levels of consciousness. The letter T is used to designate that the patient was intubated at the time of examination. A GCS of 8 or less is generally accepted as a definition of coma. At this level, there will often be a monitor placed to measure intracranial pressure (ICP) because it is difficult to monitor these patients for neurologic deterioration as compared with a patient with a higher GCS. Of patients with a moderate head injury (GCS, 9 to 13), approximately 10% to 20% will deteriorate and lapse into a coma. This can influence the timing of the management of maxillofacial injuries. Unless the maxillofacial injures compromise the airway, these patients are not routinely intubated as long as the airway can be protected. For patients with a severe head injury (GCS, 3 to 8), urgent management is critical. These patients are at the greatest risk for mortality and morbidity. They will generally have an endotracheal tube placed to protect their airway. Airway protection can be complicated in the head-injured patient because there are often concomitant maxillofacial injuries.

Trauma Score and Revised Trauma Score. The Trauma Score was initially developed in 1981 by Champion et al. The purpose of the score was to assess the extent of injury to vital systems and the severity of injury quickly to aid with triage and the treatment of the trauma patient.[11] In 1989, Champion et al revised this scoring system, which became the Revised Trauma Score.[12] The Trauma Score and Revised Trauma Score quantify the physiologic status

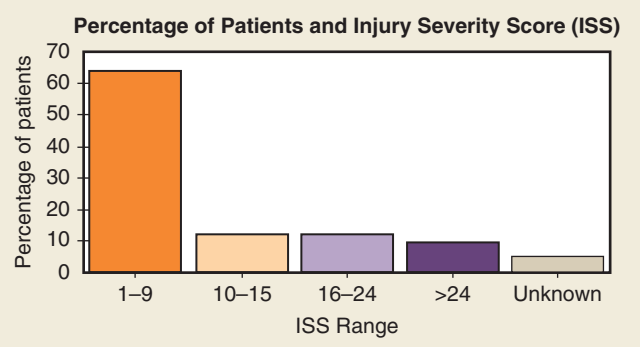


FIGURE 4-4 Percentage of patients by injury severity score (ISS) range. (The percentage of patients = number of patients for each ISS range divided by the total number of patients × 100.) (Courtesy American College of Surgeons, National Trauma Data Bank, 2004.)

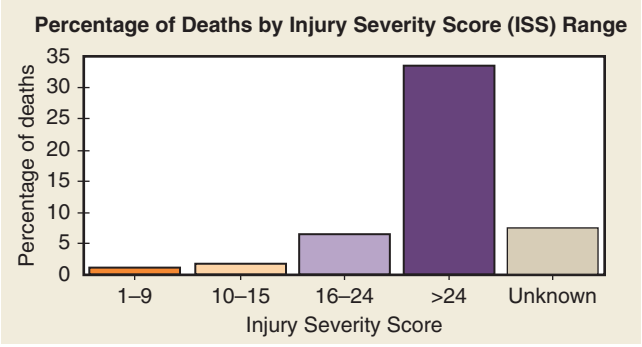


FIGURE 4-5 Percentage of deaths grouped by ISS range. (Percentage of deaths = number of deaths divided by the total number of patients × 100 by ISS range.) (Data Courtesy American College of Surgeons, National Trauma Data Bank, 2003)

of the patient's respiratory, cardiovascular, and neurologic systems. The revised trauma score has a coded value for three variables—respiratory rate, systolic blood pressure, and GCS. Each variable is coded from 0 to 4, with a total score ranging from 0 to 12. Lower scores represent an increased severity of injury.

Injury Severity Score. The Injury Severity Score (ISS) was developed to deal with multiple traumatic injuries to multiple organ systems. Scoring is based on the severity of injury to the three most injured organ systems, including respiratory cardiovascular systems, CNS, abdomen, extremities, and skin. Each of the three most injured organ systems is graded from 1 (minor) to 6 (fatal). The grades are then squared and added to come up with a total score from 3 (12 + 12 + 12) to 108 (62 + 62 + 62). The ISS has a practical range from 1 to 75; the risk of death increases with a higher score. ISS rates have been used to predict mortality rates by comparing ISS with mortality by age group. The National Trauma Data Bank (NTDB) uses this scoring system and categorizes an ISS from 1 to 9 as minor, 10 to 15 as moderate, 16 to 24 as severe, and more than 24 as very severe. Figures 4-4 and 4-5 show NTDB data as they relate to the ISS. The average length of stay increases approximately 3 days for each level of severity, as outlined in Figure 4-6.

Other Scoring Systems. Other scoring systems have also been created to aid triage and outcome prediction in the trauma patient. These include the Pediatric Trauma Score (PTS), the Trauma Injury and Injury Severity Score (TRISS) method, A Severity Characteristic of Trauma Score (ASCOT) and, more recently, the *International Classification of Diseases,* 9th edition, which uses ICD-9 nomenclature.

Mechanism of Injury

The mechanism of injury can provide insight into other possible injuries that have not yet resulted in significant changes in vital signs or physiologic function. The mechanism of injury is usually one of the first issues communicated by EMS to the trauma team as a patient enters the trauma bay. Factors with a high correlation with life-threatening injuries are outlined in Box 4-1.

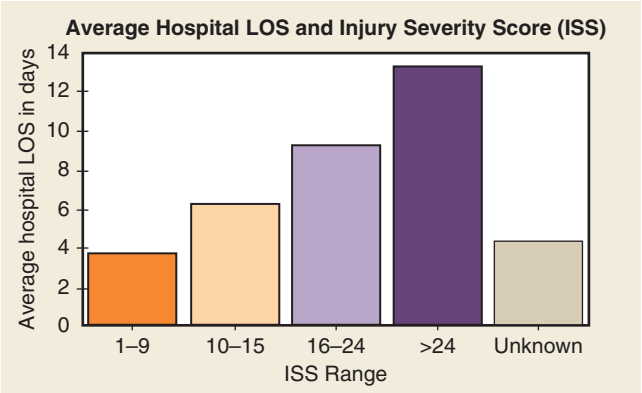


FIGURE 4-6 Average hospital length of stay for each category of ISS range. (Average hospital length of stay = total hospital length of stay for each ISS range divided by the total number of patients.) (Courtesy American College of Surgeons, National Trauma Data Bank, 2004.)

BOX 4-1 Factors Highly Correlated with Life-Threatening Injuries

- Collisions involving high-energy dissipation or rapid deceleration
- Falls > 20 feet
- Dangerous environments (burning building, icy waters)
- Automobile accidents that result in > 20 min to remove the patient
- Significant damage to passenger compartments
- Rearward displacement of front axle
- Automobile rollover
- Death of other passengers in the automobile
- Patient ejection

Data from Committee on Trauma of the American College of Surgeons: Hospital and pre-hospital resources for optimal care of the injured patient. *Bull Am Coll Surg* 68:11, 1983.

Other Factors

Various anatomic factors that correlate with high mortality include those listed in Box 4-2. Other factors that have been shown to worsen prognosis of a trauma patient with only a moderately severe injury include concurrent

BOX 4-2 Anatomic Factors Correlated with High Mortality

- Penetration trauma to head
- Penetration trauma to neck
- Penetration trauma to groin
- Penetration trauma to thigh
- Flail chest
- Major burns
- Amputations
- Two or more proximal long bone fractures
- Paralysis

Data from Mullins RJ, Mann NC: Development of a systematic review of published evidence regarding the efficacy of trauma systems. *J Surg Outcomes* 1:45, 1985.

disease, age younger than 5 years or older than 55 years, and cardiac or respiratory disease.

PRIMARY SURVEY

Once the trauma victim arrives at the resuscitation room, a quick assessment of major injuries, vital signs, and mechanism of injury is done to prioritize what needs to be treated first. The airway is always the first concern. A trauma team leader will command the personnel regarding actions to take. A team member will be ready to write all the important information regarding the patient who has just arrived. There are formats specially designed for this purpose with human body graphics in different views, whereby drawings could be made for better documentation of the injuries. EMS personnel usually will describe the information regarding the patient as they enter the room.

The treatment could be summarized as primary survey, resuscitation of vital functions, a second survey, once the patient has stabilized, and definitive care. The logical sequence of this process constitutes the ABCDEs of trauma:

Airway maintenance with cervical spine protection
Breathing and ventilation
Circulation with hemorrhage control
Disability—neurologic status
Exposure, environmental control: Undressing the patient but preventing hypothermia

During the primary survey, the life-threatening conditions are identified and treated and vital signs are stabilized. Different conditions are frequently treated simultaneously.

Special consideration must be given to pediatric, pregnant, and older patients. In general, the guidelines are the same, but the health care provider should take into consideration the differences in physiology, metabolism, response to trauma, amount of fluids, resuscitation techniques, and medication dosage. In the pregnant patient, the condition can be assessed by palpating the abdomen or a performing a human chorionic gonadotropin test. It is important to mention that in the geriatric population, comorbidities such as diabetes, cardiovascular disease, coagulopathies, liver and renal disease, and the long-term use of medications can alter the physiologic response to injury.

AIRWAY MAINTENANCE WITH CERVICAL SPINE CONTROL

On initial evaluation, ascertaining patency of the airway is of the highest priority. The perfusion of the brain and other vital structures will mean the difference between life and death, or permanent disability. Maintaining oxygenation and preventing hypercarbia are critical, especially for patients who have sustained head trauma. In a practical way, in a nonintubated patient, asking brief questions will lead to a quick evaluation of the airway and neurologic status. If the patient can talk, the airway is often patent. This assessment has to be repeated constantly.

The airway is especially critical in the head and/or maxillofacial trauma patient. The causes of upper airway compromise in the trauma patient may be tongue position, aspiration of foreign bodies, regurgitation of stomach contents, or facial, mandibular, tracheal and/or laryngeal fractures, bleeding, a retropharyngeal hematoma resulting from cervical spine fractures. or traumatic brain injury.[13] Any laceration to the neck is classified according to the anatomic level and aggressively explored. Placement of a surgical airway may be necessary if endotracheal intubation is not possible.

A patent airway must be established while still protecting the cervical spine. A jaw thrust or chin lift procedure can be used to accomplish this. The jaw thrust maneuver involves the placement of the fingers of both hands on the angle of the mandible and the thumbs over the teeth or chin. The mandible is then gently pulled forward and rotated inferiorly. The elbow may be placed on the surface next to the patient to assist with stability. This procedure is the safest method of jaw manipulation in a patient with a suspected cervical spine injury. However, it does necessitate the use of both hands, and assistance is required to remove debris from the airway.

A chin lift procedure may be performed by placing the thumb over the incisal edge of the mandibular anterior teeth and wrapping the fingers tightly around the symphysis of the mandible. The chin is then gently lifted anteriorly and the mouth opened, if possible. Care should be taken not to extend the neck while the other hand should be used to clear any debris from the oral cavity.

Tonsillar suction should also be used to clear any secretions or other accumulations from the pharynx. A nasogastric tube or soft suction catheter may be used in patients without suspected substantial midface fractures or cranial base fractures because these tubes can be inadvertently passed into the cranial vault. An oral or nasal airway should be placed to keep the airway patent and the tongue elevated off the posterior pharynx. A nasal airway is better tolerated in an awake patient.

Any patient with multisystem trauma, altered level of consciousness, or blunt trauma above the clavicle should be assumed to have a cervical spine injury. Hyperextension or hyperflexion of the patient's neck during airway establishment should be avoided. Excessive movement can turn a cervical spine injury without neuronal damage

into neuronal deficit or even paralysis. In a multiple trauma patient with an altered level of consciousness, or any injury above the clavicle, the cervical spine (C-spine) has to be protected and stabilized. The presence of distracting injuries may deflect the attention to the C-spine; usually, a cervical collar is placed until the C-spine could be cleared. If during the placement of a definitive airway, the cervical collar has to be removed, another trauma team member will hold the head and neck to provide temporary stabilization. The cervical spine can be maintained in a neutral position using a backboard, bindings, and purpose built head immobilizers. The use of soft or semirigid collars is discouraged because they only stabilize 50% of movement. Cervical spine control should be maintained in a patient with suspected cervical spine injury until it can be ruled out clinically and/or radiographically during the secondary survey. Patients with severe head injury and an altered level of consciousness because of alcohol or drugs, or with a Glasgow Coma Score (GCS) of 8 or less, usually require the placement of a definitive airway with C-spine protection.

A patient who does not demonstrate purposeful motor responses or a patient who is combative often requires definitive airway management, with placement of an airway cuffed tube in the trachea and the tube connected to some form of oxygen ventilatory system. All airway management equipment should always be taped in place.

Endotracheal Intubation

When intubation is to be performed, two trained individuals should be involved, one performing the intubation and the other administering medications such as sedatives and muscle-paralyzing drugs, which should be used with caution. In many cases in which intubation is performed during the initial assessment, the use of sedatives and muscle blockers are unnecessary.

Oral or nasal intubation should be performed according to the operator's preference and the injuries that are present. Extraordinary care must be taken in cases of facial trauma (e.g., nasal, frontal, midface fractures) In addition, applying cricoid pressure during the intubation will help to prevent aspiration.

The mnemonic LEMON (Table 4-2) is useful when assessing the difficulty for intubation:

- ***L***ook externally.
- ***E***valuate 3-3-2 rule
- ***M***allampati scale
- ***O***bstruction
- ***N***eck mobility

After initial preoxygenation, a direct laryngoscopy is performed. The tube is inserted in the trachea and the cuff inflated; once the position of the tube is verified, it is secured and connected to a ventilation system. A gum elastic bougie can be used in cases of difficult intubation.

Airway control by rapid-sequence tracheal intubation (oral endotracheal tube [OETT]) is performed with in-line stabilization of the cervical spine. Correct placement of the endotracheal tube can be confirmed using the following:

1. End-tidal carbon dioxide monitoring device
2. Observation of the tube passing through the vocal cords
3. Auscultation of the chest

The pediatric patient needs special consideration regarding instrumentation and knowledge of different anatomic features, such as the position of the larynx. In addition, obesity and retrognathia are other medical conditions for which intubation may be more challenging.

Several well-defined options for achieving airway control must be established in the event that OETT placement is not able to be achieved. These options include laryngeal mask airway (LMA), intubating LMA, fiberoptic intubation, percutaneous cricothyroidotomy, and surgical cricothyroidotomy (tracheostomy in children). Tracheal inspection is essential to determine

TABLE 4-2 LEMON Chart

Physical Signs	Less Difficult Airway	More Difficult Airway
Look externally	Normal face and neck No face or neck pathology	Abnormal face shape Sunken cheeks Edentulous Buck teeth Receding mandible Narrow mouth Obesity Face or neck pathology
Evaluate 3-3-2 rule	Mouth opening > 3F Hyoid-chin distance > 3F Thyroid cartilage to mouth floor distance > 2F	Mouth opening < 3F Hyoid-chin distance < 3F Thyroid cartilage to mouth floor distance < 2F
Mallampati scale	Class I and II (can see the soft palate, uvula, fauces, ± facial pillars)	Class III and IV (can only see the hard palate ± soft palate ± base of uvula)
Obstruction	None	Pathology within or surrounding the upper airway (e.g., peritonsillar abscess, epiglottis, retropharyngeal abscess)
Neck mobility	Can flex and extend the neck normally	Limited range of motion of the neck

whether there is peritracheal crepitus or deviation from the midline indicating potential direct airway, intrathoracic pulmonary, or major vascular injury.

Surgical Airway

When intubation is not possible, such as edema of the glottis, laryngeal fracture, or profuse hemorrhage, a surgical airway must be established. A cricothyrotomy is preferred to a tracheotomy because it involves less time and is associated with less bleeding.

Needle Cricothyroidotomy. Needle cricothyroidotomy is a temporary airway. The patient can be oxygenated for a maximum of 30 to 45 minutes. A needle is inserted into the cricothyroid membrane. A jet system is then connected, which will provide oxygen until a more definitive airway can be established. It is not indicated for patients with abnormal pulmonary function or chest injury. Furthermore, it is not indicated for patients with head trauma caused by CO_2 retention.

Surgical Cricothyroidotomy. Surgical cricothyroidotomy is a surgical incision made on the skin, extending to the cricothyroid membrane. A hemostat or scalpel handle may be used to dilate the opening, followed by the insertion of a small-caliber tube into the trachea (5 to 7 mm outer diameter [OD]). This is not recommended for children because of potential damage to the cricoid cartilage.

Airway Evaluation

The airway can be compromised at any time. It could be sudden, progressive, total, or partial. Therefore, paying constant attention to the airway will warrant its patency. The following are signs of airway obstruction:

1. Observation. Agitation, labored breathing, using accessory muscles indicates hypoxia; obtundation indicates accumulation of carbon dioxide or hypercarbia. Cyanosis, a late sign, will indicate inadequate oxygenation. The use of pulse oxymetry is an adjunct for blood oxygen saturation.
2. Listen for abnormal sounds. Stridor can be associated with partial obstruction of the airway. Hoarseness implies functional laryngeal obstruction.
3. Palpate the trachea and determine whether it is in the midline

BREATHING

Once a patent airway is verified or established, pulmonary function should be assessed. Adequate exchange of gas is required for oxygenation and elimination of carbon dioxide. Gas exchange necessitates adequate ventilation. The lungs, chest wall, and diaphragm must all function adequately to ensure proper ventilation.

Breathing evaluation is most readily accomplished by visual inspection and palpation of thoracic cage movement and auscultation of gas entry. The patient is assessed for inequalities in chest movement from one side to the other, crepitus, and local movement asymmetry, as in paradoxic thoracic cage movement in flail chest. A trained provider should also be evaluating the patient for signs of impending respiratory failure, such as uncoordinated thoracic cage and abdominal wall movement, accessory muscle use, and stridor.

Inadequate ventilation may result in hypoxemia, hypercarbia, cyanosis, depressed level of consciousness, bradycardia, tachycardia, hypertension, and/or hypotension. As a general rule, until stability has been ensured, one should administer high-flow oxygen by mask to all patients to abrogate the potential for hypoxemia.

If the patient is breathing spontaneously and ventilation is adequate, supplemental oxygen can be administered by face mask. Assisted ventilation has to be instituted if opening of the airway does not result in spontaneous ventilation. Compromised ventilation could be the result of airway obstruction, altered mechanics, or CNS depression.

The exchange of air does not guarantee adequate ventilation. There are injuries that may impair ventilation, causing an obstructive or mechanical impairment. For example, a patient with a pneumothorax, flail chest, or hemothorax may have a chest wall that moves but ventilation may still be inadequate. In addition, shallow breaths or slow rates may not allow for adequate ventilation. Very slow and rapid rates of respiration suggest poor ventilation. Older patients with pulmonary dysfunction fall into a group with an increased risk of developing mechanical pulmonary problems.

A patient's respiratory status should be monitored constantly. Signs of ventilator deterioration warrant placement of a secured airway via an endotracheal tube or the initiation of assisted ventilation. At this point, a patient can be artificially ventilated by a bag-valve mask or a bag attached to an endotracheal tube. Patients who require assisted positive-pressure ventilation with an Ambu-Bag or mechanical ventilator should be closely monitored if their chest status has not been completely evaluated. A simple pneumothorax can be converted into a tension pneumothorax when the intrathoracic pressure increases (Fig. 4-7). In the presence of a pneumothorax, especially a tension pneumothorax, immediate treatment with a regular chest tube or needle insertion is necessary.

During the primary survey, the chest should be fully exposed and inspected for any signs of obvious injury. Presence of bruising, flail chest, penetration, and bleeding should be noted. The chest should be palpated for signs of rib or sternal fractures. Any subcutaneous emphysema should be appreciated. The neck should be evaluated for any sign of tracheal deviation and jugular venous distention. Chest expansion should be equal bilaterally, without intercostal or supraclavicular muscle retractions during respiration. The breathing rate should be assessed for signs of abnormality, such as tachypnea. Tachypnea with shallow respirations is suggestive of chest injury and impeding hypoxia. Distant heart sounds and distention of the neck veins can be suggestive of cardiac tamponade. If any of these conditions is suspected, before or after intubation and initial ventilation, a chest x-ray would be mandatory.

Head injuries may result in abnormal breathing patterns that could alter normal ventilation. Spinal cord injuries at the level of the cervical spine may affect normal muscle function and therefore compromise oxygen demands. Complete cervical transection at the C3 and C4 levels will compromise the phrenic nerves,



FIGURE 4-7 **A,** Simple pneumothorax. **B,** Tension pneumothorax.

resulting in abdominal breathing and paralysis of the intercostal muscles.

Standard monitors with a capnometer and pulse oximeter ensure adequate ventilation evaluation. Remember that the pulse oximeter does not measure the partial pressure of oxygen (Pao_2) and, according to the position of the oxyhemoglobin dissociation curve, the Pao_2 could vary. The use of pulse oximetry alone cannot distinguish between oxyhemoglobin and carboxyhemoglobin or methemoglobin. This is an important consideration in patients with vasoconstriction and carbon dioxide poisoning.

CIRCULATION MANAGEMENT

In the primary survey, circulation becomes the priority after airway and breathing have been definitively managed. Delivery of oxygen to the tissues is dependent on adequate circulation. The main cause of deaths that can be prevented is caused by hemorrhage. It is estimated that hemorrhage accounts for 30% to 40% of trauma mortality, with 35% to 65% of deaths occurring in the prehospital period; 50% of deaths secondary to hemorrhage occur within the first 24 hours after the initial trauma.[14] In general, blood volume is 7% of body weight; in children, it is considered to be 8% to 9%. Shock in a trauma patient is primarily hypovolemic secondary to trauma, although the patient may present with cardiogenic, neurogenic, or even septic shock. There are conditions that will contribute to a shocklike tension pneumothorax by reducing venous return. Extensive damage to the CNS or spinal cord may result in a neurogenic shock. In very unusual situations, septic shock may be present if the treatment of the patient was initiated several hours after the initial trauma.

Response to Shock

The initial circulatory response to hemorrhage is physiologic compensation. There is a release of endogenous catecholamines and hormones. The catecholamines will increase vascular resistance, increasing diastolic blood pressure and reducing pulse pressure—difference between the maximum and minimum blood pressures produced during one heartbeat.

Other vasoactive hormones that are released include histamine, bradykinin, beta endorphins, and a cascade of postanoids and cytokines. Vasoconstriction of the skin and extremities help maintain perfusion to the vital organs of the brain, kidneys, and heart. There will be an increase in heart rate to maintain cardiac output. Initially, blood pressure is not affected, although once compensatory mechanisms are overcome, it will be affected.

In cases of major fractures of the tibia or humerus, blood loss could reach up to 1.5 units. In femoral fractures, it could be twice that amount and, in pelvic fractures, blood loss could be significant, presenting as a retroperitoneal hematoma. Edema, in the case of increased endothelium permeability fluid loss, will shift from the plasma to the extravascular space.

Poor perfusion will affect aerobic metabolism, with nutrients and oxygen being deprived, and consequently result in anaerobic metabolism, with the formation of lactic acid. This will ultimately lead to metabolic acidosis. If this situation continues, the cell membrane will lose its integrity and there will be progressive cellular damage. Continuous hypoxia will result in cellular death. Research has shown that the lethal triad of acidosis, hypothermia, and coagulopathy initially cause the first problems in the polytrauma patient, associated with high mortality.[15]

The initial damage to soft tissues and organs, and fractures in the trauma patient, represents a major challenge. The local tissue damage resulting from contusions or lacerations, hypoxia, and hypotension result in further damage from local and systemic responses. These processes are activated to preserve immune system integrity and stimulate reparative mechanisms. This systemic inflammation is known as SIRS (systemic inflammatory response syndrome). In addition, the initial inflammatory response is augmented by a second hit, such as ischemia and reperfusion injuries and surgical interventions or infections (two-hit theory).[16]

In this chapter, the differences between adults and children are discussed, followed by a review of circulatory compromise and its definitive management. Causes of circulatory compromise include the following:

- Respiratory failure, hypoxemia, hypercarbia
- Blood loss, internal or external
- Tension pneumothorax
- Pericardial tamponade
- Ruptured ventricle (rare)
- Cardiac contusions
- Acidosis
- Burns
- Profound hypothermia

Effects of circulatory compromise include the following:

- Decreased or complete loss of consciousness
- Respiratory distress, failure
- Hypovolemia—reduced cardiac output leads to inadequate blood flow to all body organs (hypovolemic shock)
- Tissue hypoxia, metabolic acidosis, increased respiratory rate
- Ischemic injury to the brain, heart, kidneys, liver, and bowel, with cell death and inadequate function of these organs

ASSESSMENT

The surgeon should be aware of the simultaneous compensatory mechanisms in a trauma patient and also the individual situation. In the older patient, for example, aging will affect general and specific organ systems that are predictive of failing health. Medically compromising conditions and the use of different medications are frequently present in this group and can contribute to high morbidity and mortality rates with relative minor injuries.[17] According to the ABCs of trauma, once the airway is patent and ventilation is reestablished, the hemodynamic status (blood volume and cardiac output) should be assessed, as follows

- Level of consciousness: Cerebral perfusion indicates an adequate circulating volume of blood, although a conscious patient may have a significant amount of blood loss.
- Pulse: The pulse should be checked in central arteries such as the femoral and carotid arteries.

Rapid pulse may indicate blood loss whereas an irregular pulse may indicate cardiac dysfunction. Different age categories should be taken into consideration when assessing heart rates. For example, young patients with normal vagal tone and older patients with pacemakers and/or beta blockers will have different responses to hypovolemia. The heart rate will not increase as expected.

- Respiratory rate: According to the degree of hemorrhage present, patients may become tachypneic as a physiologic response to the need for more oxygen to be delivered to the tissues.
- Blood pressure: If the blood loss is significant (>30% of blood volume), there will be changes in blood pressure.
- Skin color: A gray, pale ashen tone may indicate hypovolemia; pink skin is an indication of good perfusion.
- Urinary output: Urinary output is considered to be in normal limits with approximately 0.5 mL/kg/hr for the adult and 1 mL/kg/hr for children. A decrease of urinary output to less than 30 mL/hr in an adult may indicate hypovolemia in the absence of other medical conditions (e.g., renal damage).
- Acid-base balance: In early hemorrhagic shock, there is an initial respiratory alkalosis, followed by metabolic acidosis in severe cases.

In general, children have an extraordinary physiologic reserve and will not show signs or symptoms of hypovolemia until there is a severe circulating blood depletion. A cool patient with tachycardia is considered to be in shock, until proven otherwise.

TREATMENT

Definitive Hemorrhage Control and Resuscitation

The first step in hemorrhage control is to find the source of bleeding. In a penetrating injury with active bleeding, the anatomic location of entrance and exit wounds guide the surgical procedure to stop the bleeding. Hemodynamically unstable patients have to be taken for a quick radiologic screening to search for possible sources of bleeding in the chest, abdomen, or pelvic compartments. The use of multislice abdominal ultrasound (focused assessment with sonography for trauma [FAST]) can be performed to find abdominal free fluid. A positive result necessitates an emergency laparotomy.[14] Advances in resuscitation techniques have reduced the mortality rate, along with the use of interventional radiology for embolization of vessels without the need for immediate surgical intervention.[18]

External hemorrhage is managed with pressure and bandages. Small wounds and scalp lacerations represent a potential source for blood loss; 300 mL of blood could be easily lost in 1 hour if there are several small bleeding wounds. During the second survey, it may be necessary to use staples or sutures to control bleeding.

In respect to resuscitation, venous access and fluid replacement are an important part of prehospital advanced life support (ALS) and are carried out based on clinical judgment.[19] At least two large-caliber IV catheters should be used. It is important to remember that the amount of fluid delivered is determined by the diameter of the gauge of the catheter compared inversely with its length, and not by the caliber of the vein where it is placed. Other IV lines are placed as necessary, according to the trauma leader who is directing the treatment of the patient. In children younger than 6 years, the placement of an intraosseous needle should be attempted before placing a central line. Intraosseous access is also possible in adults. Blood tests are done at this time, including arterial blood gas (ABG) tests, complete blood count (CBC), and cross-matching.

When aggressive resuscitation is necessary, 1 liter of warm crystalloid solution (normal saline or lactated Ringer's solution) should be given as a bolus at the beginning of the procedure, followed by another liter according to vital signs. Giving 20 mL/kg is recommended for pediatric patients. Shock usually is hypovolemic in origin in the trauma patient. Generally, 3 mL of crystalloid are needed for each milliliter of blood loss. If the patient does not respond to this therapy, blood transfusion may be required.

Hypothermia should be prevented by protecting the patient using a warm solution (39° C [102.2° F]) and increasing the temperature of the resuscitation room. Blood cannot be warmed in a microwave but can be passed through IV fluid warmers.

After the initial resuscitation, the patient's response in terms of adequate perfusion and oxygenation is assessed by mental status, urinary output, and vital signs. Improvements in central venous pressure (CVP) and skin circulation are good indicators of adequate therapy. Acid-balance is continuously monitored and, in cases of metabolic acidosis, bicarbonate is rarely indicated.

Blood Replacement

Blood replacement may be necessary, despite the fact that resuscitation is provided through the administration of crystalloids. The transfusion of blood restores the intravascular oxygen- carrying capacity. When there is no information regarding the patient's type-specific blood, type O packed cells are indicated in case of emergency hemorrhage. Unmatched, type-specific blood is preferred over type O in life-threatening situations.

Coagulopathy

Patients who suffer severe trauma may present with a coagulopathy, which is a complex and multifactorial condition that includes tissue trauma, shock, hemodilution, hypothermia, acidemia, and inflammation, with the primary factors being direct trauma in conjunction with shock and hypoperfusion. This process seems to occur immediately after an accident; studies have shown a 24% to 36% incidence of early post-traumatic coagulopathy.[20,21]

Determination of coagulation factors, prothrombin time (PT), partial thromboplastin time (PTT), and platelet counts are usually ordered within the first hour, especially with a previous history of coagulation disorders or use of medications that affect coagulation (e.g., warfarin, nonsteroidal anti-inflammatory drugs [NSAIDs]). Transfusion platelets, cryoprecipitates, and fresh-frozen plasma (FFP) are administered accordingly. Biswadev et al has shown increased initial survival in association with higher FFP-to-RBC (red blood cell) ratios in massive transfusion in cases of higher proportions of blunt injuries.[22] Patients with brain injuries release thromboplastin and are prone to coagulation disorders. Close monitoring is mandatory.

DISABILITY

During the acute resuscitation period, a brief assessment of neurologic status should be performed. This assessment should include the patient's posture (e.g., any asymmetry, decerebrate or decorticate posturing), pupil asymmetry, pupillary response to light, and global assessment of patient responsiveness. A recommended system is the AVPU method:

A—Patient is awake, alert, and appropriate.
V—Patient responds to voice.
P—Patient responds to pain.
U—Patient is unresponsive.

During the secondary survey, a complementary assessment using the GCS should be made and whenever the patient's mental status appears to change. A more detailed assessment of the patient's neurologic status is made during the secondary survey. The assessment during the primary survey establishes a baseline; if the patient's neurologic condition varies from the primary to the secondary survey, a change in intracranial status may be present. A decrease in the level of consciousness may indicate decreased cerebral oxygenation or perfusion, which will necessitate a reevaluation of the ABCs.

The reactivity of the pupils to light provides a quick assessment of cerebral function. The pupils should react equally. Changes may indicate cerebral nerve damage, optic nerve damage, or changes in ICP. Further changes in pupil reactivity or levels of consciousness may be caused by alterations in ventilation or oxygenation status. The most common causes of coma or depressed levels of consciousness are hypoxia, hypercarbia, and hypoperfusion of the brain.[23] Depressed levels of consciousness with narrow pinpoint pupils may be seen with opiate overdose. In overdose with meperidine hydrochloride (Demerol), the pupils may appear normal or dilated. Treatment necessitates the narcotic antagonist naloxone hydrochloride (Narcan), with 0.4 mg given initially. Care should be taken to avoid a quick, violent withdrawal phase in the opiate abuser, which is accompanied by profound distress, nausea, agitation, and muscle cramps. Hypoglycemia and hyperglycemia can cause depressed levels of consciousness. If a quick blood glucose level cannot be obtained because of other injuries, the patient can be given an immediate bolus of 25 g of glucose to manage the more critical hypoglycemia. A benefit of the glucose load is the attainment of a hyperosmolar status, which may reduce cerebral edema for a short time.[24]

EXPOSURE

Patients should be completely disrobed during the initial assessment and the subsequent secondary survey. This

helps ensure the observation and assessment of significant injuries. At the same time, efforts to prevent significant hypothermia using a warm ambient room (82° to 86° F [28° to 30° C]), overhead heating, and warmed IV fluids, should be instituted. The patient's temperature should be measured on arrival at the emergency room and strenuous efforts should be made to avoid significant hypothermia during resuscitation and therapeutic intervention.

SECONDARY ASSESSMENT

The secondary assessment is initiated once the primary assessment has been completed and management of life-threatening conditions has begun. The patient's vital signs and condition should be constantly monitored to assess the effects of intervention during the primary survey and any life-threatening issue that was undiagnosed during the primary survey. Changes in the patient's vital signs, respiratory and cardiovascular function, and neurologic status are expected to occur within the first 24 hours after the initial trauma.[24] A secondary assessment includes a subjective and objective evaluation of the trauma patient.

The secondary assessment begins with a subjective evaluation. If possible, this should include a brief interview with the patient. In the comatose patient, family members, bystanders, or other victims are a good source of information for subjective evaluation. A brief health history should be obtained, which includes medications, allergies, previous surgeries, and history of the injury (e.g., location, duration, time frame, intensity).

The history should include an assessment based on the following protocol. This can be remembered by using the AMPLE acronym: ***A,*** allergies; ***M,*** medications; ***P,*** past medical, surgical, and social history; ***L,*** last meal; and ***E,*** events leading to injury, scene findings, notable interventions, and recordings en route to the hospital.

An objective evaluation should include inspection, palpation, percussion, and auscultation of the patient from head to toe. All segments of the body (head and skull, maxillofacial area, neck, chest, abdomen, extremities) and neurologic status should be evaluated to provide a baseline of the patient's current condition. Furthermore, special procedures such as peritoneal lavage, radiographic studies, and further blood studies may be performed during this evaluative phase. Many institutions will conduct FAST as part of their primary survey rather than as part of the secondary survey.

FACE, HEAD, AND SKULL INJURIES

The next sequential phase in the evaluation of injury and instability is the palpation of the entire cranium and face. Sutures, staples, or Raney clips may be helpful in controlling bleeding from large scalp flaps. Palpate for facial crepitus and a mobile middle third of the face as a clue to potential difficulty in airway control. Hemotympanum and the presence of bruising around the eyes (raccoon eyes) and mastoid process (Battle sign) suggest a basal skull fracture.

Recheck the pupils and repeat GCS scoring. Evaluate the cranial nerves, peripheral motor and sensory function, coordination, and reflexes. Identify any neurologic asymmetry. Patients with lateralizing signs and those with an altered level of consciousness (GCS score < 14) should undergo cranial computed tomography (CT) scanning. Patients with a traumatic brain injury (TBI) are particularly susceptible to secondary brain injury from hypoperfusion, hypoxia, hypercarbia, hyperglycemia, hyperthermia, and seizure activity.

Injuries to the head and skull may include lacerations, abrasions, avulsions, and contusions, fracture of the cranium, cerebral contusions, and shearing injuries. These can result in intracranial bleeding, hypoxia, and ischemia, which can lead to secondary brain injury. A cerebral ischemia may not be caused by a head injury but can result from an arterial hypotension. Cranial injuries can cause an ischemia as a result of elevated ICP and pressure on intracranial vessels from an expanding hematoma. Severe volume changes taking place within the fixed volume of the skull can lead to herniation of the brain from the cranial vault.

Cerebral hypoxia is caused by impaired oxygen delivery to the brain, which can occur from ischemia or from reduced oxygen-carrying capacity of the blood. Secondary effects of hypoxia and many forms of ischemia are usually preventable. Approximately 50% of patients with head injuries have some degree of reversible injury caused by increased ICP that can be controlled with aggressive measures. Failure to control an increased ICP is the most common cause of death in hospital patients with head injuries. Hypertension with concomitant bradycardia and irregular respirations (Cushing's triad) can be a sign of increasing ICP. In contrast, hypotension with tachycardia usually indicates hypovolemia from blood loss. Shock is rarely associated with primary neurologic injury, and therefore a systemic source of blood loss should be investigated. Cushing's triad is usually present in less than 25% of patients with increased ICP, even in cases of ICP higher than 30 mm Hg (>15 mm Hg is considered abnormal).

Head CT scanning is a rapid noninvasive technique that can accurately assess neurologic injuries and detect mass lesions. It causes no life-threatening risk to the head-injured patient and establishes a baseline for future studies. In patients with suspected intracranial injury, CT scans can quickly and easily diagnose and localize intracranial hemorrhage, contusions, foreign bodies, and skull fractures. Also, a CT scan can demonstrate secondary effects of trauma, such as edema, ischemia, infarction, brain shift, and hydrocephalus. With acutely traumatized patients, CT scans have an almost 100% accuracy in the diagnosis of intracerebral and extracerebral hematoma formation. Substantial lesion masses can cause cerebral ischemia by elevation of the ICP or compression vasculature. Following stabilization of the trauma patient, a CT scan should be performed immediately rather than waiting for clinical signs of an expanding intracranial hematoma. In patients with a suspected head injury, the possibility of a lesion mass has been reported in 40% of adults and 25% of children (<18 years). Controversy remains concerning indications for a head CT scan; however, it should usually be performed in patients with seizure activity, unconsciousness lasting

longer than a few minutes, abnormal mental status, abnormal neurologic evaluation, and clinical evidence of skull fracture. The use of CT scans has been suggested for patients with blunt trauma and those who have experienced any loss of consciousness or mild amnesia, even with normal neurologic findings.[25]

Types of Injuries

Skull Fractures. Skull fractures may present in the cranial vault or the base of the skull. The classic signs of a skull base fracture include periorbital ecchymosis (raccoon eyes) retroauricular ecchymosis (Battle sign), cerebrospinal fluid (CSF) leakage from the nose, and cranial nerve VII or VIII impairment. Carotid arteries may be affected. CT scans are mandatory. An arteriography should also be performed in a patient with a suspected injury to the carotid.

Intracranial Lesions. Intracranial lesion can be diffuse or focal. A significant brain injury can occur from a diffuse brain injury caused by a mild concussion, with only brief periods of apnea, or from more severe diffuse injuries in which there is a prolonged period of apnea or shock. The CT scan may appear initially normal or may appear swollen, with loss of the normal gray-white appearance. Diffuse axonal injuries may present as multiple punctuated hemorrhages through the cerebral hemispheres. These are usually the result of a high-velocity impact or deceleration.

Hematomas

Epidural Hematomas. Epidural hematomas result from the tear of the medial meningeal artery and, occasionally, from a venous sinus. The hematoma materializes between the dura and inner table of the skull. The shape is biconvex or lenticular. They are relatively uncommon and constitute only 0.5% of all brain injuries.

Subdural Hematomas. Subdural hematomas develop from the shearing of small surface or bridging blood vessels of the cerebral cortex. The shape follows more along the contour of the brain and are more common than epidural hematomas.

Contusion or Intracerebral Hematomas. Contusion or intracerebral hematomas occur frequently in the frontal and temporal lobes. They may transform into intracerebral hematomas or the contusion may evolve to such a magnitude that it requires surgical evacuation.

Diagnostic Testing and Evaluation

In patients with brain injuries, a CT scan should be ordered, if available, within 24 hours of the initial evaluation and a neurosurgery consultation obtained once cardiopulmonary stabilization is achieved. Because of the high incidence of cervical spine fractures in patients with head and facial trauma, extreme care should be taken when moving this patient to the CT scanner.[26] If there is suspicion of spinal trauma, the cervical spine should remain immobilized before movement of the patient and the CT examination should be extended to include visualization of the cervical spine. If facial injuries are suspected, the CT scan should be extended inferiorly to allow visualization of the inferior border of the mandible. However, in many cases, evaluation and treatment of facial injuries are delayed because of the needless transport of the patient back to the CT scanner for additional imaging as a result of failure to extend the initial CT examination to all structures of the face.

As the ICP continues to rise further above normal (15 mm Hg), there is a standard progression of neurologic abnormalities that are seen clinically. In order of occurrence, these abnormalities include factors affecting the following:

1. Cerebral cortex and altered state of consciousness
2. Midbrain, exhibited by dilation and then progressive fixation of the pupils
3. Pons, resulting in loss of the corneal reflex and abnormal doll's eye sign
4. Medulla, demonstrated by apnea and followed by hypotension and death

A careful physical examination of the head should include examination of the scalp for lacerations and foreign bodies. Because of the rich vascular supply of the scalp, substantial blood loss can result. Lacerations overlying injury to the cranium or intracranial hemorrhage may be present. These wounds can act as portals for the entry of bacteria into the injured area(s), resulting in meningitis or brain abscess. Hematoma formation or ecchymosis over the mastoid (Battle sign), hemotympanum, CSF rhinorrhea or otorrhea, and subscleral hemorrhage all suggest basilar skull fracture. In patients with suspected basilar skull fractures, nasogastric tube placement should not be performed because the tube can inadvertently be passed into the cranial vault.

A brief neurologic examination should evaluate the level of consciousness, motor and cranial nerve function (suggestive of mass lesions), brainstem findings, and trends in neurologic status. A neurologic examination can be complicated by trauma patients under the influence of drugs and alcohol. A decreased level of consciousness should not be solely attributed to alcohol or drug intoxication until intracranial pathology or injury has been ruled out.

As noted, the GCS (see Table 4-1) provides a simple grading of consciousness and functional capacity of the cerebral cortex. This scale is based on three behavioral responses—eye opening, best verbal response, and best motor response. This scale can be used in the field and as a reassessment tool to assess brain function, brain damage, and patient progress. Injury to two areas of the brain, regardless of cause, can produce unconsciousness, the cerebral cortices bilaterally and the brainstem reticular activating system.[27]

The assessment of motor function is part of the GCS and provides information about asymmetrical function. Conscious patients should be asked to move their extremities in response to commands. The inability to follow these commands may indicate damage to the spinal cord. In the unconscious patient, deep tendon reflex and the plantar response can demonstrate the functioning of sensory input and motor output. Any nonpurposeful or abnormal postural movements are of special concern. Abnormal flexor activity (decorticate) involves flexion of the forearms on the chest, with flexion of the wrists and fingers. Abnormal extensor posturing occurs when the arms, hands, and fingers are extended, with the hands abducted. In both situations, the lower extremities are

extended and there is no attempt to localize the point of stimulation. Bilateral extensor plantar responses are nonspecific. However, a unilateral Babinski sign suggests corticospinal tract damage. The level of consciousness and brainstem function can be assessed through pupillary function. Eye movements and eye opening can provide information about the degree of injury. The size, shape, and reactivity of the pupil to light provide insight with respect to the function of cranial nerves II and III and midbrain activity. A sluggish but reactive pupil or a dilated nonreactive (blown) pupil unilaterally indicates compression of cranial nerve III by brain herniation in the unconscious patient. The pupillary light reflex can be used to evaluate cranial nerve function and possible elevated ICP with brain herniation.

With normal pupillary activity, light directed into one eye produces contraction of pupils in both eyes. The optic nerve (cranial nerve II) carries visual and papillary fibers. Bilateral optic nerves connect shortly after they leave the retina to form the optic chiasm. At the optic chiasm, the nasal fibers cross to join the temporal fibers from the other eye and the visual fibers and terminate in the visual cortex of the occipital lobe. The pupillary fibers are relayed bilaterally to the Edinger-Westphal nucleus of the oculomotor nerve (cranial nerve III). Cranial nerve III supplies neurologic function to the sphincter muscle of the iris, causing it to contract. Autonomic innervation also supplies the eyes. The iris is supplied by sympathetic and parasympathetic fibers. Stimulation of the sympathetic fibers causes the pupil to dilate and elevation of the upper eyelid via Muller's muscle. If a light is shone into the right eye and the left eye does not respond, there may be disruption of the right optic nerve or left oculomotor nerve. A light should then be shone in the left eye. If the left eye does not respond, disruption of the cranial nerve III should be suspected. Pupillary dilation of one eye may be caused by brain herniation developing on the ipsilateral side, with bilateral papillary dilation suggestive of notable midbrain injury or loss of parasympathetic function. Conversely, pinpoint pupils after head trauma may indicate drug overdose or loss of sympathetic tone as seen in Horner's syndrome.

The brainstem can be assessed through an evaluation of the corneal reflex. The corneal reflex involves sensory input from the trigeminal nerve (cranial nerve V). The oculocephalic maneuver (doll's eye test) necessitates an intact vestibulocochlear nerve (cranial nerve VII) to permit head rotation to evaluate reflexive movement of the eyes. This maneuver should not be used in patients with suspected cervical spine injury.

The oculovestibular response test can be used to evaluate cranial nerves III, IV, VI, and VIII along with brainstem activity. This test is performed by irrigating the external auditory canal with cold water. If the reflex is intact, there should be full eye movement toward the ear canal lavaged with cold water. If there is no movement, disruption of the neural tract of the tympanic membrane should be suspected.

A lumbar puncture should not be performed in patients with acute head injuries. Changes in pressure associated with removal of CSF from the lumbar region may precipitate cerebral herniation in the trauma patient with an elevated ICP. CSF emerging from the nose (rhinorrhea) or ear (otorrhea) is commonly associated with a basilar skull fracture. Clear or red-tinged fluid that drains from the nose or ear should be considered to be CSF. No reliable method exists in the emergency room for distinguishing CSF from nasal mucus. The use of glucose indicator sticks has been associated with a high incidence of false-positive results. A useful aid may be a ring sign. A drop of blood from the nose or ear is placed on a piece of filter paper. The blood components of the fluid remain in the center and rings of clear fluid may develop about it, thus producing the ring sign.[9] This test often lacks sensitivity and specificity. A positive laboratory test for beta-2 transferrin confirms the presence of CSF in the fluid. With confirmation of a CSF leak, a CT scan should be performed to determine the presence, location, and extent of fracture. The head of the bed should be elevated to 90 degrees. If indicated, the fracture should be reduced and the leakage should resolve within 7 days. If a CSF leak is persistent, neurosurgical procedures may be indicated to repair the dural tear.

The performance of a rectal examination is important for patients with head injuries. The presence of rectal sphincter tone suggests intracranial injury only. With the absence of rectal tone, a coexisting spinal cord injury should be suspected. Coexisting head and spine injuries should be suspected until proven otherwise.

A head injury can be initially classified as mild (GCS, 13 to 15), moderate (GCS, 9 to 12), or severe (GCS < 8). Patients with a head injury and no loss of consciousness, no amnesia, no palpable fractures, and a GCS score of 15 can be discharged to a reliable caretaker without brain imaging. However, it is generally recommended to have CT imaging because of the convenience and decreased cost of this imaging modality. Patients with a history of loss of consciousness, amnesia, or a GCS score of 13 to 14 must undergo immediate head CT. With a negative finding in the CT examination, the patient can be discharged with instructions. Admission to an ICU or neurologic observation unit for continuing care and observation is indicated for a patient presenting with focal neurologic abnormalities, a GCS score of less than 13, or an intracranial lesion on a head CT. Prophylactic phenytoin (Dilantin) administration with an IV loading dose of 18 mg/kg is used by some trained individuals for control of possible seizure activity.

Benzodiazepines can be used for ongoing seizure activity. A neurosurgical consultation should always be obtained early in the management of any obvious head trauma or head injury. Severely headiinjured patients (GCS < 8) should undergo rapid sequence intubation to protect the airway and achieve better control of ICP. An increased ICP can be controlled using various techniques, including the following: (1) reverse Trendelenburg's position; (2) osmotic diuresis (mannitol); (3) hyperventilation of intubated patient (little reported benefit and this has fallen out of favor); (4) sedation; (5) pharmacologic paralysis; and (6) phenobarbital coma (last resort). The judicious use of resuscitative fluids (limited fluid resuscitation) and control of systemic hypertension will also help control an elevated ICP.

NECK INJURIES

Maintaining cervical spine stabilization when removing a rigid cervical immobilization device is imperative. Penetrating injuries of the neck may require angiographic, bronchoscopic, or radiologic examination, depending on the level of injury (i.e., zone I, II, or III). In particular, zone II injuries that violate the platysma may be readily explored. Those injuries that correlate to zone I or III benefit from additional investigation because of the difficulty in identifying and controlling injuries in those zones.

CHEST INJURIES

During the entire secondary assessment of the multiple injured patient, the primary evaluation of airway, breathing, and circulation must be continually monitored for the detection of new or overlooked findings and/or injuries. A pneumothorax, open pneumothorax, hemothorax, flail chest, and cardiac tamponade can develop after primary assessment and must be treated accordingly. Chest injuries are directly responsible for more than 25% of the 50,000 to 60,000 fatalities that result annually from MVAs and contribute significantly to at least 25% more of these fatalities. Therefore, continued monitoring and management of chest injuries are essential.

Open Pneumothorax

An open pneumothorax occurs when there is a defect in the chest wall, allowing air to be moved in and out of the pleural cavity with each respiration. This condition may result when a large defect of the chest is present.

If the opening is two thirds of the diameter of the trachea, air will enter through this opening, resulting in ventilatory impairment, hypoxia. and hypercarbia. The loss in chest wall integrity allows an equilibrium to develop between intrathoracic pressure and atmospheric pressure. With each respiration, the involved lung collapses on inspiration and slightly expands on expiration, allowing air to be sucked in and out of the chest wall. Hence, this pathology is referred to as a sucking chest wound. If the chest wall defect (wound) is two thirds of the size of the diameter of the trachea, air will pass through the chest wall defect because it becomes the path of least resistance. As the involved lung collapses, the loss of negative pleural pressure causes the expired air from the normal lung to pass into the affected lung on expiration and back into the normal lung on inspiration. This results in an enlarged functional dead space in the normal lung and loss of the involved lung, thereby causing a ventilation perfusion problem.

The treatment of this condition involves coverage of the defect with a sterile occlusive dressing secured at three of the four sides. The unsecured side acts as a one-way valve, allowing air to escape the pleural cavity on expiration. Taping of all edges could cause accumulation of air within the thoracic cavity and subsequent development of a tension pneumothorax. Vaseline gauze can be used as a temporary occlusive dressing over large defects during the initial examination. Chest tube placement at a distant site on the affected chest wall should be performed to prevent development of a tension pneumothorax; the ensuing wound should eventually be closed in the operating room. If, after closure of the wound in the operating room, the lung fails to expand or poor ventilation persists, the patient should be placed on a ventilator with positive end-expiratory pressure (PEEP) to allow expansion of the lung. While on the ventilator with PEEP, the patient should be closely monitored with a chest tube in place to prevent the development of a tension pneumothorax. The signs of a tension pneumothorax in patients on ventilators include increased airway resistance, tracheal shift, and diminished tidal volume.

Closed Pneumothorax

A closed pneumothorax can result from blunt trauma to the chest or fracture of a rib with laceration to the lung. An external communication into the pleural space, such as through a wound, is not present in the closed pneumothorax. Air from the lungs is allowed to pass through a lung laceration into the pleural space, equalizing the negative pleural pressure and subsequently collapsing the lung. This collapse of the lung results in a ventilation perfusion deficit because blood is still circulated to the collapsed lung but is not oxygenated. Percussion to the side of the chest with a pneumothorax elicits hyperresonance. Breath sounds on the affected side are usually distant or absent. Confirmation and evaluation of a pneumothorax are done via upright chest radiography.

On seriously injured patients, studies have shown that 76% of occult pneumothoraces have gone undiagnosed when x-rays were interpreted by the trauma team. It is imperative to pay attention to clinical predictors in patients who are at greater risk. These risks include subcutaneous emphysema, pulmonary contusions, rib fractures, and female gender). Occult pneumothoraces are prone to develop further complications in patients with a decreased cardiac pulmonary reserve or in those who need positive-pressure mechanical ventilation.[28]

An open pneumothorax with an occlusive dressing placed over the wound is transformed into a closed pneumothorax. Traumatically induced pneumothoraces are frequently treated with tube thoracostomy (chest tubes). This procedure is used to correct respiratory compromise. However, a small pneumothorax may be treated by hospitalization with close observation if the patient is otherwise healthy, symptom-free, does not require general anesthetic or mechanical ventilation, and if the size of the pneumothorax is not increasing, as measured on serial 24-hour x-rays.[29,30] This situation is rarely seen with the trauma patient, and a chest tube should be placed in a patient with a pneumothorax and accompanying multiple injuries.

A chest tube is generally placed anteriorly in the second intercostal space midclavicular line or in the fourth or fifth intercostal space midaxillary line. The level generally corresponds to the level of the nipple. If a patient has large breasts, it is essential to have an assistant medialize the breast tissue. The midaxillary site is generally preferred for cosmetic reasons, and it can effectively remove fluid and air if the tube is positioned properly (superiorly toward the apex of the lung). The second intercostal space location is usually reserved for needle

decompression of a suspected tension pneumothorax. A moderately sized tube is most often the choice for this procedure. A size 32 to 40 Fr is acceptable in adults and size 26 to 30 Fr in children. If a hemothorax is present, a larger tube is generally selected. The region is prepped and draped and a local anesthetic is generally used. A skin incision that is approximately 3 cm in length is first made at one intercostal space below the intended placement of the tube. This is to allow the tube to maintain an increased seal and conform to the profile of the rib cage. Subsequently, a gloved finger is used to traverse bluntly through the subcutaneous tissue in a superior direction to locate the intercostal space intended for tube placement. A curved Kelly clamp is used to separate the intercostal muscles. A finger is inserted to verify entrance into the pleural cavity. The tube is then placed superiorly and posteriorly into the pleural cavity and secured to the skin with suture; an occlusive dressing placed over the defect around the tube. The next phase of the procedure is connection of the tube to an underwater sealed drainage to remove air or fluid. After final placement, an upright posteroanterior and lateral chest radiograph should be taken to confirm the position of tube, position of the last drainage hole in the tube, and amount of air and fluid remaining in the pleural cavity. Serial daily radiography and physical examination should be performed to monitor the progress of air and fluid removal. If the tube becomes blocked and substantial amounts of air or fluid remain, a new chest tube should be inserted.

Tension Pneumothorax

The presence of a tension pneumothorax should be considered in patients who rapidly become acutely ill and develop severe respiratory distress. Patients who fall into this category will exhibit decreased breath sounds, hyperresonance on one side of the chest, distended neck veins, and deviation of the trachea away from the involved side. A tension pneumothorax develops when an injury through the chest wall or from the lung acts as a one-way valve, allowing air to enter the pleural cavity on inspiration without escape on expiration. This results in a progressive increase of intrapleural pressure, causing complete collapse of the affected lung. With increasing pressure, the trachea and mediastinum are displaced to the opposite pleural cavity and impinge on the normal lung. Compression on the normal lung causes shunting of blood flow to nonventilated areas and results in severe ventilation-perfusion disturbances. The positive intrapleural pressure causes compression of the vena cava, resulting in decreased cardiac output. All these changes develop into a rapid onset of hypoxia, acidosis, and shock.[30] Common causes of tension pneumothorax include mechanical ventilation with PEEP and spontaneous pneumothorax with blunt chest trauma in cases in which the parenchyma has failed to seal. Traumatic chest defects have also been occasionally known to cause tension pneumothorax.[27]

A tension pneumothorax can rapidly become fatal if not treated quickly. Treatment includes release of intrapleural pressure as soon as possible. This can be accomplished by inserting a large-bore needle (14 to 16 gauge) anteriorly in the affected hemithorax through the second or third intercostal space in the midclavicular line. This maneuver can quickly convert a tension pneumothorax to a pneumothorax, which can then be treated by insertion of a chest tube.

It is critical that maxillofacial patients with chest wall injuries be closely monitored during the perioperative period. If a patient has been given positive-pressure ventilation during an operative procedure, there is a risk of a tension pneumothorax. If a patient does have a chest tube in place, its removal may in part be dictated by the timing of the repair of the maxillofacial injuries. Communication with the trauma surgeon and anesthetist regarding the timing of maxillofacial repairs is beneficial. It is important to be vigilant when treating maxillofacial injuries in a trauma patient who has had a small pneumothorax that was not managed with a chest tube or in a patient who has had a chest tube removed before surgery. The positive-pressure ventilation delivered by an anesthetist can expand the prexisting pneumothorax.

Hemothorax

A hemothorax is a collection of blood within the pleural cavity. It occurs most frequently as a result of penetrating injuries. It can also result from any blunt trauma that disrupts the vasculature. Initial blood accumulation within the pleural cavity is bleeding that is coming from the lung parenchyma. This process is usually slow in nature because of the low pulmonary pressure. A massive hemothorax with rapid accumulation of blood is a result of injuries of the aortic arch, pulmonary hilum, internal mammary arteries, and/or intercostal arteries. A hemothorax can notably reduce vital capacity of the lung and result in hypovolemic shock in the presence of a large amount of blood in the chest. A hemothorax, usually associated with a pneumothorax, can produce metabolic and respiratory acidosis via decreased cardiac output and hypoxia, respectively.

When evaluating a patient with reduced breath sounds and dull percussion to one lung field and who also has a history of sustaining penetrating or blunt chest trauma, a hemothorax should be suspected. Neck veins can be flat because of severe hypovolemia or distended because of the mechanical effect of a chest full of blood.[27] The loss of a small amount of blood (<400 mL) makes it more difficult to diagnosis a hemothorax. This is because changes in the patient's appearance, vital signs, or physical findings may be minimal. A collection of fluid more than 200 to 300 mL can be observed on good upright chest radiographs, with blunting of the costophrenic angles evident. A supine chest radiograph is considered to be less accurate.[30]

The treatment of a hemothorax includes airway control, supported ventilation as required, drainage of the accumulated blood in the pleural cavity, and restoration of the circulating blood volume. The blood volume can be restored by transfusion of fluids, volume expanders, blood or blood products via large-bore IV lines. Blood can be evacuated from the pleural cavity as outlined earlier through insertion of a large chest tube (36 to 40 Fr) at the fifth or sixth intercostal space at the midaxillary line. The tube should be directed superiorly

and posteriorly to prevent damage to an elevated diaphragm. The chest tube is to be connected to an underwater seal with continuous suction (20 to 30 cm of water). A new chest tube should be inserted in lieu of irrigating a tube that becomes clogged or fails to drain. Patients with massive bleeding can have an autotransfusion of the evacuated blood until banked blood becomes available.[31] It is critical to evaluate the amount of fluid being evacuated from the chest tube initially and on an ongoing basis. Surgical exploration via thoracotomy is indicated in patients with persistent hemorrhage. Massive hemothorax can result from the accumulation of more than 1500 mL of blood or one third of the patient's blood volume in the thoracic cavity. Conditions that exacerbate the need for surgical exploration include the following: (1) an initial drainage of more than 20 mL/kg of blood; (2) persistent bleeding at a reate of more than 7 mL/kg/hr; (3) increased appearance of hemothoraces on serial chest x-rays; and (4) hypotension despite volume replacement. Furthermore, when other sites of blood loss have been ruled out or the patient decompensates after initial response to resuscitation, exploratory surgery may be indicated.[30] In a few cases, presentation to a critical care unit may necessitate an emergency thoracotomy. This procedure, however, is associated with an increased mortality rate.

Flail Chest

Flail chest occurs when a disruption of the bony integrity of the chest is present. A flail chest is a result of multiple rib fractures. These fractures present at multiple sites along the rib, creating an unstable fragment of chest wall. This fragment moves paradoxically during respiration, moving inward with inspiration and outward with expiration. The mechanical effects of paradoxical movement of the fragment and the pain associated with respiration may lead to hypoxemia. A fractured rib can also puncture the lung, causing pneumothorax and/or hemothorax. Often, the problem that occurs with a flail chest is a contused lung. Initially, the contused lung may be asymptomatic, but later may interfere with gas exchange as fluid moves into the lung and decreases compliance. In many cases, it is the contusion injury to the lung in a flail chest that causes most of the hypoxia and morbidity. Mortality rates in patients who sustain severe blunt injury to the chest remain high, at 12% to 50%.[32]

A flail chest is apparent on visual inspection in an unconscious patient. However, in the conscious patient, the injury may not be as apparent because of splinting of the chest wall by the patient. Because of the paradoxical movement of the chest wall fragment and the ensuing pain on respiration, movement of the thorax can be asymmetrical and uncoordinated, resulting in poor ventilation. The area of the fractures can also be tender to palpation.

The management of flail chest injuries generally involves three steps. The first stage involves stabilization of the loose segment with an external splint. A sandbag, rolled sheet, or IV fluid bag can be taped over the area with paradoxical movement. This splinting of the fractured segment of chest wall allows for reduced movement of the segment and reduced pain associated with movement. The splinting tends to reduce vital capacity; however, it increases the ventilation efficiency. This form of treatment is adequate for approximately 30 minutes until more definitive treatment can be rendered. A prolonged period of splinting can produce atelectasis. Severe injuries can be managed with open reduction and internal fixation.

The second stage of treatment provides prolonged relief of pain via intercostal nerve blocks. Pain relief from the fractured segment allows the patient to breathe deeply and cough.

The final stage of treatment uses a volume-cycled respirator with endotracheal intubation to provide PEEP and intermittent mandatory ventilation (IMV). The effective internal splinting with the ventilator support allows for adequate depth of ventilation, improves oxygen absorption in the areas of contusion, and decreases atelectasis. If the initial stage of treatment is implemented early, the respiratory support is generally only required for 2 to 4 days. If there is a delay until the patient shows signs of respiratory difficulty, prolonged therapy is generally required for up to 14 days.

Cardiac Tamponade

Cardiac tamponade occurs when the pericardium is filled with blood from the heart, great vessels, or pericardial vessels. This injury is a direct result of blunt or penetrating trauma. A small amount of blood is required to fill the pericardial sac, causing restriction of cardiac activity and filling. Signs of cardiac tamponade include the classic Beck triad, comprised of venous pressure elevation, decrease in arterial pressure, and muffled heart tones. Pulseless electrical activity (PEA) is suggestive of cardiac tamponade. Diagnosis is established with the use of the echocardiogram, FAST, or pericardial window.

Surgical intervention with rapid evacuation of pericardial fluid is indicated for unstable patients who do not respond to resuscitation measures and are suspected of having cardiac tamponade. Pericardiocentesis could be diagnostic and/or therapeutic in cases without an experienced surgeon available, but it would not be definitive.

The secondary assessment of the trauma patient includes evaluation of an upright view chest radiograph for the presence of air in the mediastinum or under the diaphragm, a widening of the mediastinum with a shift toward the midline, thoracic injuries and fractures with lung expansion, and the presence of fluid collection. In most cases, the multisystem-injured trauma patient will be immobilized on a backboard and a supine film can be substituted for an upright one. If suspicion of a chest injury exists, a CT scan of the chest should also be obtained. An electrocardiogram (ECG), arterial blood gas analysis, hematocrit, and urinalysis should also be obtained. Six potentially lethal injuries that should be evaluated in the secondary assessment are pulmonary contusion, aortic disruption, tracheobronchial disruption, esophageal disruption, traumatic diaphragmatic hernia, and myocardial contusion.[27]

The treatment for pulmonary contusions with or without accompanying flail chest is the same. Pulmonary

contusions occur commonly with blunt chest trauma because the capillary damage in the lungs results in interstitial and intra-alveolar edema and shunting. Pulmonary contusions and adult respiratory distress syndrome (ARDS) are the most common potentially lethal chest injuries seen in the United States. This is because resulting respiratory failure does not occur instantaneously, but develops 24 to 72 hours after the initial injury. Patients with these injuries often complain of pain and dyspnea. Their blood gas levels tend to deteriorate progressively over the initial 48 to 72 hours because of the increasing edema developing in the alveoli. Chest x-rays may demonstrate opacification developing in the involved areas. Treatment for these conditions include adequate ventilation of the lungs, chest physiotherapy, supplemental oxygen, coughing with deep breathing, and nasotracheal suction. If the patient should require ventilator assistance, spontaneous ventilation with intermediate mechanical ventilation provides a much better ventilation-perfusion ratio, better hemodynamics, and quicker weaning than pure assisted ventilation. The use of steroids to reduce inflammation is a controversial issue for these patients.

Tracheobronchial Tree Injury

Tracheobronchial tree injury ensues when there is injury to the trachea or main bronchus. At times, this injury is overlooked, resulting in death of the trauma patient. The typical presentation is a patient with subcutaneous emphysema, hemoptysis, or tension pneumothorax.

If a pneumothorax continues to present an air leak, tracheobronchial tree injury should be suspected and immediate surgical consultation should be obtained. Intubation in these cases is difficult secondary to distortion of normal anatomy. In this situation, surgical intervention should be immediately performed.

Damage to intrathoracic large arteries or veins via blunt or penetrating trauma is the most common cause of sudden death after an MVA or a fall from a great height.[33] The aortic root and descending aorta at the origin of the ductus arteriosus and diaphragm are common sites of injury.

Complete rupture is fatal unless the surgical intervention is performed within a few minutes. A small number of patients, approximately 15% of those with thoracic aortic injuries, reach the hospital alive. It is not uncommon for the aortic intima and media to be fractured circumferentially, with only the adventitia and surrounding mediastinal tissue preventing fatal hemorrhage. Patients with this type of injury may appear clinically stable; however, failure to recognize this vascular injury leads to eventual death. The indicators found on chest radiographs that are suggestive of thoracic vascular injury include a widened mediastinum, fractures of the first and second ribs, obliteration of the aortic knob, deviation of the trachea to the right, presence of a pleural cap, deviation of the esophagus to the left, and downward displacement of the left mainstem bronchus. Patients with suspected aortic rupture on clinical or radiographic examination should have arteriography performed. It is imperative that the patient be restricted from excessive coughing or gagging—as can occur with the placement of a nasogastric tube—and becoming hypertensive until an aortic rupture can be ruled out by arteriography.

Blunt Cardiac Injury

Blunt cardiac injury resulting from blunt trauma can cause cardiac chamber rupture, dissection of coronary arteries, thrombosis, valvular disruption, and/or cardiac tamponade. In a conscious patient, this can manifest as chest discomfort presenting as a rib or sternum fracture contusion. Hypotension and dysrhythmias may be present. The implementation of FAST is of paramount importance in the diagnosis during the initial assessment. A two-dimensional echocardiogram will confirm the diagnosis. Electrocardiographic changes are variable and troponins are nonspecific.

OTHER POTENTIALLY LIFE-THREATENING INJURIES

Traumatic Esophageal Rupture

Traumatic esophageal rupture occurs as a direct result of penetrating trauma and, less frequently, blunt trauma. This rupture occurs when gastric contents are forced into the esophagus from a blow to the abdomen that produces a tear. Thus, the gastric contents leak into the mediastinum, resulting in mediastinitis or pleural space empyema. Treatment mandates a direct repair with wide drainage of the mediastinum and pleural space.

Traumatic Diaphragmatic Injury

Blunt trauma may result in large tears of the diaphragm, whereas penetrating trauma will produce small tears. It is usually missed in the x-rays. This injury is more common in the left than the right side. Treatment consists of direct repair.

ABDOMINAL AND PELVIC TRAUMA

Abdominal trauma and pelvic trauma include diaphragm injuries, duodenal injuries, genitourinary injuries, small bowel injuries, solid organ injuries, and pelvic fractures.

Diaphragm Injuries

Blunt tears occur more frequently in the left side in this type of injury. X-ray findings include blurring of the hemidiaphragm or hemithorax. As noted, these injuries are often undiagnosed.

Duodenal Injuries

Duodenal injuries occur as a result of a direct blow to the abdomen. This trauma is often seen in unrestrained drivers in MVAs or in bicycle riders who sustain a frontal collision. X-ray films will show retroperitoneal gas or the gastric aspiration will show blood.

Genitourinary Injuries

A traumatic impact to the back may result in renal abdominal injuries. These may present with gross hematuria, microscopic hematuria in patients with penetrating abdominal wounds, and hypotension. In addition, anterior pelvic fractures are frequently correlated with urethral injuries.

Small Bowel Injuries

Small bowel injuries occur in the process of deceleration, with tearing at a fixed point. Typically, these patients will show ecchymosis in the lower abdomen resulting from the seat belt (seat belt sign). The presence of a distracting lumbar injury should suggest a small bowel injury.

Solid Organ Injuries

Injuries to the liver and/or spleen could be present in an unstable patient. A laparotomy is indicated in patients in whom injury to these organs is suspected, with evidence of hemodynamic instability or shock.

Pelvic Fractures

Pelvic fractures are frequently the result of high-impact injuries sustained through MVAs (car, pedestrians) or motorcycle accidents associated with extensive blood loss. Pelvic fractures coincide with peritoneal and retroperitoneal visceral and vascular injuries. Tears to the thoracic aorta may be present. Simple immobilization with a sheet will decrease the pelvic volume. Rapid transportation to a trauma center is essential.

SPINE AND SPINAL TRAUMA

Spine and spinal injury should always be suspected in the trauma patient and special care should be implemented when transporting such individuals. These patients should be immobilized during transport. The transportation board is to be removed as soon as possible to minimize further damage, but only after injuries to the cervical, thoracic, and lumbar spine have been ruled out. It is important to recognize the importance of removing the board in a timely manner and avoiding prolonged use. Shock patients have a reduced perfusion pressure. Patients with spinal injuries and a potential neurologic deficit in the autoregulation of blood pressure, as well as those with loss of pain sensation, are at greater risk of developing sores. Ironically, however, these patients are more likely to spend more time on boards because of complicated treatment plans.[34]

During the examination, neurologically intact patients who are alert and awake can be asked simple questions that may help expedite care to the target area of the injury. The initial evaluation should be followed by lateral cervical x-rays and chest and pelvic radiography. If a patient is unconscious, a craniocervical 2-mm-thick CT scan should be obtained. If this study is negative, magnetic resonance imaging (MRI) is the preferred study of choice for excluding instability.[35]

MAXILLOFACIAL INJURIES

The involvement of a maxillofacial surgeon in the polytrauma patient is important. Early treatment of facial injuries, whenever possible, will result in better functional and esthetic outcomes.[36]

It is possible for maxillofacial injuries to result in airway compromise because of the following: (1) blood and secretions; (2) mandibular fracture that allows the tongue to fall posteriorly against the posterior wall of the pharynx; (3) midface injury that causes the maxilla to fall inferiorly and posteriorly into the nasopharynx; and (4) foreign debris, such as avulsed teeth or dentures. The oral cavity and pharynx should be cleared of any debris or secretions using a large tonsillar suction. An oral airway assists with tongue position; however, care must always be taken to prevent manipulation of the neck and to provide for access to the oral cavity and dentition for reduction and fixation of any fractures requiring some period of intermaxillary fixation (IMF). Neither the presence of midface fractures nor cerebrospinal fluid (CSF) rhinorrhea is a contraindication to nasal intubation. Nevertheless, care should be taken to pass the tube along the floor of the nose into the pharynx, and the tube should be visualized before intubation of the trachea.

A thorough physical examination should begin with an evaluation for soft tissue injury. Lacerations should be débrided. Vital structures, such as the facial nerve or parotid duct, should be carefully examined for signs of disruption. Eyelids should be elevated to examine for neurologic and possible ocular damage. The face should be examined for asymmetry, discolorations, or swelling suggestive of bony or soft tissue injury. All bony landmarks should be palpated for signs of crepitus, steps, or other irregularities. Palpation should include the supraorbital, lateral, and infraorbital rims, malar eminences, zygomatic arches, nasal bones, maxilla, and mandible. Any steps or irregularities along the bony margin may be suggestive of a fracture. Paresthesia over the area of distribution of the trigeminal nerve is usually noted with fractures of the facial skeleton. An oral cavity examination should include an evaluation of lost teeth, lacerations, and alterations in the occlusion. Teeth avulsed at the time of injury must be accounted for because the tooth may have been inadvertently aspirated or swallowed.

There should be a concomitant examination of the neck for injury. Subcutaneous air may be visualized if massive injury is present; if subtle, it may only be detected by palpation. The presence of air in the soft tissue (tissue emphysema) may be the result of tracheal damage. Any externally expanding edema or hematoma of the neck must be monitored closely. Continued expansion can lead to an airway compromise. Carotid pulses should be assessed bilaterally. Palpation of the thyroid cartilage should be performed to ascertain abnormalities in the contour and midline position of the trachea.

EXTREMITIES AND FRACTURES

Hypovolemia caused by life-threatening blood loss can be a result of pelvic fractures, fractures of the femur, and multiple fractures of other long bones—the primary site of which may be difficult to determine. Blood loss of 1 to 5 liters can occur with closed fractures of the pelvis, femur fractures can sustain a loss of 1 to 4 liters of blood, and arm fractures may induce losses of 0.5 to 1 liter of blood from the vasculature. Some extremity injuries are considered life-threatening when accompanied by complications associated with massive open fractures with a jagged dirty wound, bilateral femoral shaft fractures (open or closed), vascular injuries, with or without fractures, proximal to the knee or elbow, crush injuries of the abdomen and pelvis, major pelvic fractures, and traumatic amputations of the arm or leg.[27]

The physical examination should include inspection and palpation of the chest, abdomen, pelvis, and all four extremities. Areas of tenderness, discoloration, swelling, and deformity should be assessed and proper radiographs obtained. All peripheral pulses should be examined for evidence of vascular injury. The pulse rates should be equal; any abnormality of distal pulse rates suggests a vascular injury and requires additional exploration. Doppler examination of the extremity is useful, but angiography is best for definitively evaluating a suspected vascular injury if the diagnosis is in doubt.[27]

Direct pressure can be used to control hemorrhage and fractures should be splinted as quickly as possible. Splints should include joints about and below the site of injury. A prompt orthopedic consultation needs be obtained.

Fat embolism syndrome is frequently associated with major long bone fractures, especially fractures of the femur, in which the patient will be stable for 24 to 48 hours and then develop progressive respiratory and CNS deterioration. Concomitant laboratory value changes include hypoxemia, thrombocytopenia, fat in the urine, and a slight drop in the hemoglobin level. Fat enters the venous sinusoids at the fractured site and becomes lodged in the lung alveoli. Fat embolism syndrome has been reported to occur in 30% to 50% of major long bone and pelvic fractures. However, with current trends in the coordinated management of multiple-injured patients, the incidence of fat embolism syndrome and ARDS has decreased. Pelvic fracture treatment and expeditious management of femoral shaft injuries account for this reduction. The primary treatment is ventilator assistance. Steroid and aspirin therapy have been shown to be helpful, possibly because of a reduction of platelet aggregation.

A better understanding of fluid and electrolyte therapy, early aggressive management of hemorrhagic shock, and prompt surgical treatment are now possible. However, in the interest of acute resuscitation, orthopedic injuries are often overlooked initially and are treated at a later date. When these injuries involve the spine, pelvis, or femur they necessitate immobilization of the patient for the purpose of traction. In immobilized patients with unstable fractures, there is an increased morbidity caused by respiratory failure or sepsis with related multiple organ failure. If the severely injured patient with orthopedic fractures survives the acute phase of treatment, the ensuing regimen frequently follows a prolonged course in the ICU. This phase leads to morbidity secondary to decreased musculoskeletal function characterized by muscles wasting, stiff joints, and limb length loss. The reduction in musculoskeletal function is attributed to delays in fracture stabilization and subsequent patient immobilization. Studies have shown that early fracture stabilization can significantly lower mortality, decrease musculoskeletal morbidity, and decrease the cardiopulmonary and metabolic consequences commonly associated with polytrauma patient care.[37]

Long bone fractures are a common cause of fat embolism syndrome and ARDS. Fat embolism syndrome can be prevented by operative fixation of long bone fractures in patients with multiple injuries within the first few days of injury.[38] Primary rigid fixation allows the patient to get out of bed and assume the upright position, thus improving pulmonary and musculoskeletal function. Early mobilization, along with the use of mechanical ventilation with PEEP, lowers the incidence of ARDS and remote organ failure.[39]

INITIAL MANAGEMENT OF THE TRAUMA PATIENT IN THE INTENSIVE CARE UNIT

Trauma patients who are transferred to the ICU present with multiple challenges. They require immediate, thorough, and ongoing evaluation guided by but not limited to the initial report by the emergency care clinician and/or the trauma team's initial evaluation.

The concept of tertiary survey was introduced by Enderson et al. This occurs after the patient has been stabilized. The patient is subsequently reevaluated to confirm the initial diagnosis and to determine whether there are any other injuries missed during the primary and secondary surveys.[40] These patients may be stable on arrival, but continue to remain at risk for deterioration because of unrecognized injuries, initial surgical or medical management complications, unknown past medical history, and possible allergic reactions to essential medications.

INITIAL AND ONGOING ASSESSMENT

After receiving the transfer report for an incoming trauma patient to the ICU, all members of the responsible health care team discuss the nature of the injuries, patient's past medical history (PMH), hemodynamic status, and nature and timing of the surgical interventions performed by the field crew, emergency clinicians, or trauma surgeons for damage control.

The ICU team then quickly designs a protocol detailing immediate steps and accommodations. In addition, they ascertain whether other specialists need to be involved and formulate a plan of care. Once the patient arrives at the ICU, an immediate and comprehensive reassessment is performed in accordance with ATLS guidelines.

Airway, Breathing, and Circulation

If the patient has been intubated, the position of the tube should be assessed clinically. This is accomplished by verification of breath sounds bilaterally. Additionally, an x-ray should be taken to ensure placement and rule out dislodgment during transport.

Appropriate IV and arterial access are mandated to obtain blood gas samples and to accommodate rapid infusion in case of fluid resuscitation. A thorough neurologic assessment should be conducted, along with documentation of the patient's GCS score.

Obtaining Past Medical History

Trauma patients transferred to the ICU may be unable to supply a detailed PMH, perhaps because of altered mental status or because they are intubated and sedated. The availability of family members is also unpredictable. ICU staff members are frequently confronted with these

situations. A patient's social history, with possible substance abuse, is a vital piece of information in the management of withdrawal syndromes. It is estimated that more than 30% of trauma patients present with ongoing drug or alcohol dependence.

The ICU team needs to obtain a PMH for the trauma patient so that they may better predict possible mortality or morbidity outcomes. Studies have corroborated a direct relationship between the number of underlying medical conditions that trauma patients have and the mortality rate. Furthermore, a rapid recovery following surgical intervention can be negatively affected by the presence of chronic medical problems.

In the ICU, trauma patient status is continuously updated and ongoing assessment is of paramount importance. The possibility of undiagnosed injuries (e.g., intracranial hemorrhage, intra-abdominal injuries, iatrogenic complications of procedures) or studies that patients underwent during the initial management may require emergency procedures such as chest tube placement, endotracheal intubation, emergency cricothyroidotomy, or tracheotomy.

The ICU team is constantly evaluating the treatment plan:

- The dynamics of reevaluation are accomplished through updating current studies, laboratory test results, and values reflecting the hemodynamic status of the trauma patient. Accordingly, the recommendations would be implemented to correct and normalize the resulting values, with the goal being to optimize the chemical and electrolyte balance.
- The surgical plan prepared by the various surgical teams involved in the management of the trauma patient's injuries should be discussed in terms of keeping the NPO status and securing all surgical and anesthesia consents from the patient or family.
- The hemodynamic status is updated and any corrections needed are confirmed (e.g., fluid resuscitation, blood or blood product transfusion).
- Initiation and follow-up of the secured airway or tracheotomy are carried out as needed and per recommendation by the surgical team.
- Follow-up on studies or procedures ordered or taken by different services is carried out and the appropriate documentation is made.
- Secure a safe and monitored patient transport for different studies or procedures, as required.
- Follow isolation protocol as recommended by infectious disease guidelines when indicated.
- Follow hospital guidelines in terms of restraints and one on one observation, as needed.
- Determine the necessary consults with different specialists or services and act on their recommendations in a timely manner.
- Carry out intensive maintenance of the IV or arterial lines, airway circuits, or ventilator, including ongoing blood gas testing, and make the appropriate changes to the existing settings, as needed.
- Continually assess and update the status of the patient surgically, medically, hemodynamically, and neurologically and make appropriate recommendations for transfer to the regular floor or the step-down unit.
- Follow-up on a secured airway or tracheotomy as needed and as recommended by the surgical team.

TYPES OF INTENSIVE CARE UNITS

An ICU is a specially staffed and equipped, separate, and self-contained area of a hospital dedicated to the management of patients with life-threatening illnesses, injuries, and complications and monitoring of potentially life-threatening conditions. It provides special expertise and facilities for the support of vital functions and uses the skills of medical, nursing, and other staff experienced in the management of these problems.

In many units, ICU staffs are required to provide services outside the ICU such as emergency response (e.g., rapid response team) and outreach services. Where applicable, the hospital must provide adequate resources for these activities. Specialized types of ICUs include the following:

- Neonatal intensive care unit (NICU)
- Special care nursery (SCN)
- Pediatric intensive care unit (PICU)
- Psychiatric intensive care unit (PICU)
- Coronary care unit(CCU)
- Cardiac surgery intensive care unit (CSICU)
- Cardiovascular intensive care unit (CVICU)
- Medical intensive care unit (MICU)
- Medical-surgical intensive care unit (MSICU)
- Surgical intensive care unit (SICU)
- Overnight intensive recovery (OIR)
- Neurotrauma intensive care unit (NICU)
- Neurointensive care unit (NICU)
- Burn wound intensive care unit (BWICU)
- Trauma intensive care unit (TICU)
- Surgical trauma intensive care unit (STICU)
- Trauma-neuro critical care (TNCC)
- Respiratory intensive care unit (RICU)
- Geriatric intensive care unit (GICU)
- Mobile intensive care unit (MICU)
- Postanesthesia care unit (PACU)

MECHANICAL VENTILATION

A significant number of patients in the ICU will require some form of mechanical ventilator support. Mechanical ventilation refers to the use of life support technology to perform the work of breathing for patients who are unable to breathe effectively on their own. Patients requiring mechanical ventilation include the following:

1. Trauma patients who have been intubated to secure the airway
2. Critically ill patients with advanced and potentially reversible respiratory failure because of pulmonary or nonpulmonary processes
3. Patients who are only temporarily unable to ventilate adequately on their own following general anesthesia
4. Patients who have chronic respiratory or neuromuscular disorders that may prevent them from breathing effectively without mechanical support.

A maxillofacial trauma patient with panfacial fractures will often be intubated during the perioperative period and would therefore require admission to the SICU.

BOX 4-3 Criteria for Intubation

CLINICAL
- Shortened speech
- Use of accessory muscles
- Subjective air hunger
- Change in mental status
- Poor inspiratory force

LABORATORY*
- $Pa_{O_2}/Fi_{O_2} < 250$
- $Pa_{CO_2} > 50$ mm Hg
- pH < 7.25
- RR > 35 breaths/min
- <–25cm H_2O
- $\dot{V}_E > 12$ liter/min

*Although the decision to intubate should primarily be a clinical decision, several laboratory values and pulmonary tests can help guide this decision.

Data from Townsend CM, Mattox B, Evers M, Beauchamp D, editors: *Sabiston textbook of surgery*, ed 16, Philadelphia, 2001, WB Saunders.

RR, Respiratory rate; $\dot{V}_E$, minute ventilation.

BOX 4-4 Common Misconceptions Regarding Weaning from Ventilator Support

- The method of weaning determines the ability to wean.
- The longer the duration of mechanical ventilation, the more difficult to wean.
- Diaphragm weakness is a common cause of failed wean attempts.
- Removal of the endotracheal tube reduces the work of breathing.

Data from Marino PL: *The ICU book*, ed 2, Philadelphia, 1998, Lippincott Williams & Wilkins.

Indications

There are many algorithms that have been devised to aid the clinician when deciding to initiate mechanical ventilation. The following three rules can aid in the decision making process:

1. The indication for intubation and mechanical ventilation is "thinking of it." There is often a tendency to delay this form of therapy in the hope that it will not be necessary. It is important to note that elective intubation carries fewer risks to the patient than an emergent intubation.
2. Intubation is not an act of weakness. Physicians often believe that they must apologize on morning rounds when they have intubated a patient. However, no one should be faulted for securing an airway in a critically ill patient.
3. Endotracheal tubes are not a disease and ventilators are not an addiction. Endotracheal tubes and ventilator support do not create a need for mechanical ventilation, but cardiopulmonary and neuromuscular diseases do.[31]

Airway protection in the form of endotracheal intubation is common in many of the patients with whom an oral and maxillofacial surgeon will be involved. This can be lifesaving in the multisystem trauma patient with maxillofacial injuries. Other criteria for intubation are listed in Box 4-3.

Weaning

Weaning from mechanical ventilation generally refers to the progressive reduction in mechanical support that is delivered to a patient. The concept of weaning patients from ventilator support has been dominated by numerous misconceptions, some of which are listed in Box 4-4. No single method of weaning has been proven to be better than others. Most agree that when a patient is ready to wean from mechanical ventilation, they will be weaned. Regardless of the method used, most weaning occurs in the same sequence. Initially, the Fi_{O_2} is reduced to 40% or less and PEEP is reduced to its minimal value. The rate is then reduced. If the rate is low and PS is added, it should be weaned. During these alterations in support, the patient is appropriately evaluated for changes in hemodynamics, oxygenation, and ventilation. The patient should be awake, alert, and able to protect his or her airway before extubation. This usually means that sedation, which is frequently propofol (Diprivan) delivered by a pump, has been discontinued.

If there is a concern about airway edema, as is common with some maxillofacial trauma patients, a cuff test should be performed. In this test, the patient is removed from the ventilator, the endotracheal tube cuff is deflated, and it is verified that the patient can exchange air (inspiratory and expiratory) around the occluded endotracheal tube. Hemodynamic and metabolic stability should also be verified before extubation. For many facial trauma patients, it is prudent to have the necessary equipment ready in case reintubation is required. This could include wire cutters, equipment for a surgical airway, and possibly a fiberoptic endoscope. Some clinicians will temporarily remove a nasal endotracheal tube from the trachea and leave a portion in the nasopharynx. It will act as a nasal airway during the first several minutes as the clinician verifies that the patient has good control of the airway. If the patient maintains the airway, the tube is removed completely. If the patient is not protecting the airway despite a cuff test showing good air exchange, then the endotracheal tube is at the nasopharynx and can be advanced into the trachea.

Bedside Weaning Parameters. The first step in this phase is to identify which patients are candidates for weaning. Patients should have an adequate blood gas Fi_{O_2} of 0.5 or less and PEEP set at 5 cm H_2O or less. After this is verified, there are a number of parameters that should be evaluated (Table 4-3). Although most of these values are easily obtained, many have poor predictive value for individual patients.[32]

Two of the most predictive parameters include maximal inspiratory pressure and the frequency-to-volume ratio. The respiratory therapist is frequently asked to check weaning parameters in the ICU. These are obtained with patients who have had their level of sedation weaned so that can cooperate and reduce the respiratory effects of the sedation on their values.

TABLE 4-3 Bedside Weaning Parameters

Parameter	Normal Range (Adult)	Threshold for Weaning
PaO_2/FiO_2	>400	200
Tidal volume	5-7 mL/kg	5 mL/kg
Respiratory rate	14-18/min	<40/min
Minute ventilation	5-7 L/minute	<10 L/min
Vital capacity	65-75 mL/kg	10 mL/kg
Maximum inspiratory pressure	>–90 cm H_2O (female) >–120 cm H_2O (male)	–25 cm H_2O
Respiratory rate/tidal volume	<50/min/liter	<100/min/liter

The maximum inspiratory pressure is calculated from the ventilator by having the patient maximally exhale and then inhale maximally against a closed valve. A low maximum inspiratory pressure has more value in identifying which patients will fail to wean as opposed to a high maximum inspiratory pressure, which indicates that weaning will be successful.

The frequency-to-volume ratio attempts to identify patients who are breathing rapidly and in a shallow manner. This ratio is calculated by comparing the respiratory rate to the tidal volume. A value of less than 50 breaths/min/liter is normal. Patients who fail to wean often have values greater than 100 breaths/min/liter. This ratio has a high predictive value for identifying patients who will be able to wean.

Methods of Weaning. There are generally two types of methods for the withdrawal of ventilator support. One involves periods of time during which the patient is breathing spontaneously interspersed with periods of ventilator support. This method is often referred to as T-piece weaning. The other type of weaning uses a gradual withdrawal of ventilator support using IMV.

T-Piece Weaning. As noted, this method of weaning has the patient breathing spontaneously for a period of time. It originally consisted of a piece of equipment that was shaped like a T that connected to the endotracheal tube. It allowed inspiration of oxygen and outflow of exhaled gases. Currently, most ventilators will allow the patient to breath spontaneously without the need for a T-piece. When the spontaneous breathing takes place with the ventilator, a minimum amount of pressure support is often added to overcome the resistance in the ventilator tubing.

Intermittent Mandatory Ventilation Weaning. As noted, IMV provides a set number of machine breaths per minute and allows the patient to breathe spontaneously between machine breaths. The number of machine breaths per minute can then be gradually decreased, allowing the patient to support her or his pulmonary function with spontaneous breaths. A minimal amount of pressure support is usually added for the same reason as mentioned for T-piece weaning.

NONRESPIRATORY ISSUES TO MONITOR IN THE INTENSIVE CARE UNIT

VASCULAR ACCESS

Vascular access can essentially be divided into two broad categories, peripheral and central venous access. Peripheral short-term catheters are safe for giving many IV medications (e.g., antibiotics), for providing maintenance IV fluids, and for blood sampling for laboratory tests. However, numerous fluids and medications (e.g., hyperosmolar solutions, resuscitative drugs) cannot be given through peripheral catheters because of local and venous irritation. Children who require long-term treatment (e.g., antibiotics), emergency medications (e.g., inotropes, medications to manage cardiopulmonary arrest), chemotherapy, and total parenteral nutrition (TPN) require central venous access.

General indications for central access include administration or facilitation of the following:

- Total parenteral nutrition
- Chemotherapy
- Venous access in chronically ill children who require repeated venous punctures for blood sampling and medication
- Long-term antibiotics (e.g., >3 to 4 weeks)
- Emergency access (e.g., to manage cardiopulmonary arrest or trauma)
- Critical care monitoring (e.g., of pulmonary wedge pressure) and medications (e.g., inotropes)

GASTROINTESTINAL STRESS ULCER PROPHYLAXIS

Physiologic stress associated with illness and hospitalization is known to result in gastrointestinal ulceration, especially among the critically ill. The complication of this stress-related mucosal disease can be prevented with appropriate application of pharmacologic prophylaxis. Gastrointestinal prophylaxis guidelines in the ICU are important in the prevention of stress gastritis. The incidence of clinically significant bleeding in critically ill patients is 2% to 15%. However, it is important to understand the actual indications for prophylaxis.

Most experienced clinicians believe that it should be limited to patients at high risk for stress-related mucosal diseases, which include the following conditions: mechanical ventilation longer than 48 hours; coagulopathy; endoscopic or radiographic diagnosis of peptic ulcer or gastritis; history of an upper gastrointestinal (GI) bleed less than 6 weeks prior to admission; significant burns (>15% of total body surface area); traumatic brain injury; and large doses of glucocorticoids (e.g., >50 mg hydrocortisone/day).

The commonly used GI prophylactic medications to prevent stress gastritis include antacids, sucralfate, histamine-2 receptor antagonists (H2 blockers), and proton pump inhibitors (PPIs). Cook et al, in a multicenter, randomized, double-blind controlled trial, demonstrated that H2 blockers (ranitidine) compared with sucralfate decreased clinically significant bleeding, with no difference in ventilator-associated pneumonia.

VENOUS THROMBOEMBOLISM PROPHYLAXIS

Until the 1990s, venous thromboembolism (VTE) was viewed primarily as a complication of hospitalization for major surgery, or associated with the late stage of terminal illness. However, recent trials in patients hospitalized with a wide variety of acute medical illnesses have demonstrated a risk of VTE in medical patients comparable with that seen after major general surgery. In addition, epidemiologic studies have shown that between 25% and 50% of all clinically recognized symptomatic VTEs occur in individuals who are neither hospitalized nor recovering from a major illness. This expanding understanding of the population at risk challenges physicians to examine risk factors for VTE carefully to identify high-risk patients who could benefit from prophylaxis.

Isolated factors sufficient to prompt physicians to consider VTE prophylaxis include major surgery, multiple trauma, hip fracture, or lower extremity paralysis caused by spinal cord injury. Additional risk factors, such as previous VTE, increasing age, cardiac or respiratory failure, prolonged immobility, presence of central venous lines, estrogens, and a number of inherited and acquired hematologic conditions contribute to an increased risk for VTE. These predisposing factors are seldom sufficient by themselves to justify the use of prophylaxis. Nevertheless, individual risk factors, or combinations thereof, can have important implications for the type and duration of appropriate prophylaxis and should be carefully reviewed to assess the overall risk of VTE in each patient.

A thrombus in the proximal deep venous system of the lower extremities is often silent until a clot is dislodged and sent toward the lungs. A thrombosis below the knee is not a common source of pulmonary emboli. The trauma patient carries an increased risk for a thromboembolism. Those at the highest risk are patients with a spinal cord injury, fractures of the lower extremities or pelvis, and multisystem involvement. These patients will often have a vascular injury, venous stasis, and a generalized hypercoagulable state. Depending on the level of risk, appropriate methods of prophylaxis intervention will include sequential compression devices (SCDs), low dosage of heparin (5000 IU twice daily), low-molecular-weight heparin (enoxaparin, Lovenox; 30 mg SC twice daily) and a low dosage of coumadin. When anticoagulation is contraindicated, a nonpharmacologic intervention would be a vena cava filter (e.g., Greenfield filter). Most ICUs have protocols for screening lower extremities with ultrasound to detect venous thrombosis. Often, a combined venous compression ultrasound and Doppler ultrasound is performed (duplex scan).

The clinical manifestations of an acute pulmonary embolism are varied and not very sensitive or specific. The diagnostic tests of value include a ventilation-perfusion scan, spiral chest CT, and pulmonary angiography. The main treatment is heparinization. It is important for the oral and maxillofacial surgeon to maintain hypervigilance with respect to VTE when a trauma patient has had a prolonged stay in the ICU and is then transferred to the oral and maxillofacial surgery service to have facial fractures managed.

ENTERAL NUTRITION

Enteral nutrition in the trauma patient is recommended to be initiated as early as feasible in recently published practice guidelines. The provision of enteral nutrition for the critically ill patient preserves physical barrier gut function. The host defense function of the intestine reduces the translocation of bacteria from the gut to the bloodstream and thus decreases the possibility of a systemic infectious complication. There is a significant reduction in mortality when enteral nutrition is provided within 24 hours.[41]

ELECTROLYTE MANAGEMENT

Daily monitoring of electrolyte levels is mandatory in the ICU setting. Electrolyte imbalances may lead to instability that could worsen the status of the trauma patient. The electrolytes are usually checked before rounds in educational institutions so that replacements could be done in a timely fashion. In some cases, electrolyte imbalances are related to total body water. The following is a description of the most common situations.

Sodium

Hypernatremia. The normal serum sodium concentration is 135 to 145 mEq/liter. Hypernatremia is a result of a serum sodium level above 145 mEq/liter. This situation occurs in hypotonic fluid loss, defined as an increase in the sodium concentration that occurs when there is a loss of water from a normal serum sodium level, or hypertonic fluid gain in which there is an increase of the serum sodium level along with an increase of water (Fig. 4-8).

To treat this, the extracellular volume must be determined based on invasive hemodynamic monitoring, if available, or clinical observation. The loss of fluid occurs with loss of sodium, affecting the total body water. The loss of sodium predisposes the patient to hypovolemia and hypertonicity.

Low Extracellular Volume. All body fluids, with the exception of small bowel and pancreatic secretions, contain sodium. Hypernatremia with low extracellular volume occurs when there is hypotonic fluid loss—that is, excessive diuresis and diarrhea. The major concern in these cases is to maintain cardiac output by replacing volume. Treatment consists of rapid sodium deficit replacement and slow water replacement, maintaining intravascular volume and avoiding intracellular water shift. This is of particular importance to avoid cerebral edema. Once hypovolemia is corrected, the replacement of free water has to be accomplished. Free water deficits have to be replaced slowly so that the sodium level decreases no faster than 0.5 mEq/liter/hr.

Normal Extracellular Volume. This condition occurs when there is a net loss of water as with diabetes insipidus, central or nephrogenic, or a loss of hypotonic fluids in the diuresis process. The diagnosis of diabetes insipidus is confirmed when there is failure of the urine osmolarity to increase more than 30 mOsm/liter in the first hours of complete fluid restriction. The treatment is replacement with isotonic saline in a 1:1 ratio. The replacement is done slowly, over 2 to 3 days, to prevent intracellular overhydration as can occur in cerebral edema.

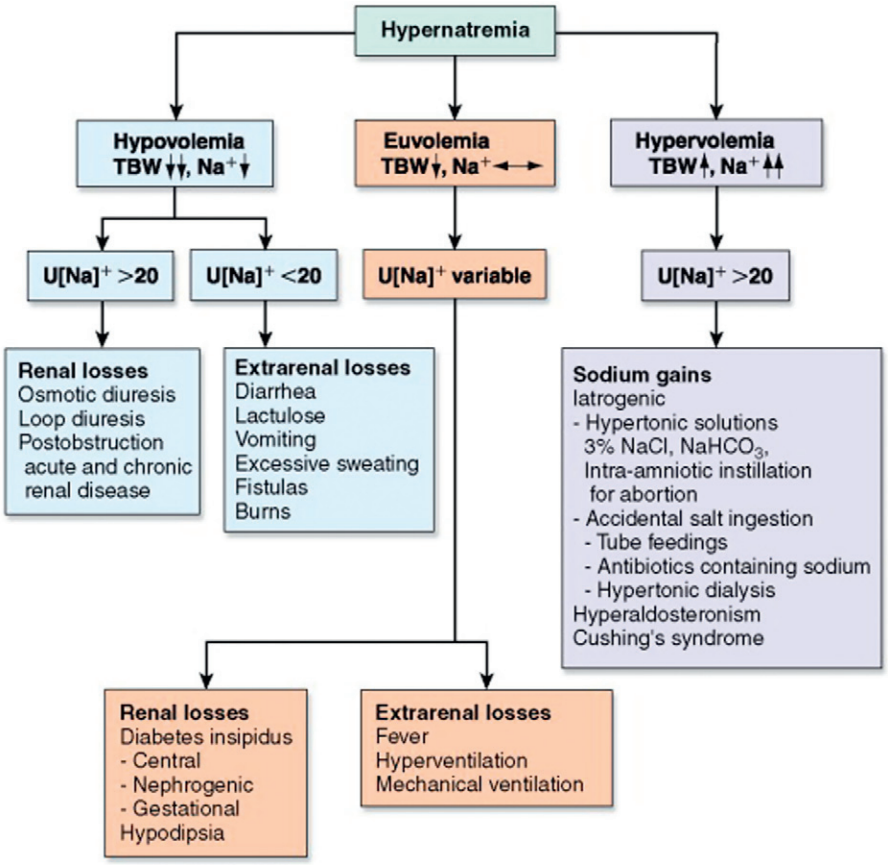


FIGURE 4-8 Algorithm for diagnosing the causes of hypernatremia. *Na^+,* Sodium; *NaCl,* sodium chloride; *$NaHCO_3$,* sodium bicarbonate; *TBW,* total body water; *U,* urinary. (Adapted from American Association of Critical Care Nursing, Carlson K: *AACN advanced critical care nursing*, St. Louis, 2009, WB Saunders, Elsevier.)

In central diabetes insipidus, administration of vasopressin is indicated to prevent further free water losses. The usual dose is 2 to 5 units of subcutaneous aqueous vasopressin every 4 to 6 hours. The serum sodium level has to be aggressively monitored.

High Extracellular Volume. Nonketotic hyperglycemia is a generally uncommon voluminous state that occurs when there is a gain of hypertonic fluids via the administration of a hypertonic solution for resuscitation or sodium bicarbonate solution for metabolic acidosis. Treatment is with diuresis, which will induce sodium loss in the urine. The urine volume loss is replaced with hypotonic fluids related to the urine.

Hyponatremia. Hyponatremia manifests when serum sodium levels of less than 135 mEq/liter are found in hospitalized patients with neurologic disorders, those with AIDS and, less commonly, older and postoperative patients.

Pseudohyponatremia. The traditional methods of measuring sodium levels use the aqueous and nonaqueous phases of plasma. Sodium is only present in the aqueous phase of plasma; thus, the amount measured will be lower than the measured level. Elevation of the protein or lipid concentration will increase the volume of the nonaqueous phase of plasma, so the measured sodium level will be decreased. Pseudohyponatremia occurs as a result of this condition. There is, however, a specific laboratory test (ion-specific electrode) that is conducted to measure the plasma sodium concentration in pseudohyponatremia.

Hypotonic Hyponatremia. Hypotonic hyponatremia is an increase of free water in relation to sodium in the extracellular fluids. In patients who present with this condition, there will be fluid losses in addition to hypotonic fluid replacement. Consequently, there will be a net sodium loss in relation to total free water.

The urine sodium level concentration will help determine if the cause for the loss is renal or extrarenal. If the urine sodium is more than 20 mEq/liter, it is renal; if less than 20 mEq/liter, it is extrarenal.

Renal losses are indicative of adrenal insufficiency and cerebral salt wasting syndrome. Extrarenal losses manifest with symptoms of vomiting and diarrhea. Treatment is implemented with the administration of hypertonic saline 3% in symptomatic patients and isotonic saline in asymptomatic patients

Isovolemic Hyponatremia. Isovolemic hyponatremia is hyponatremia with a small amount of gained water, as in inappropriate release of antidiuretic hormone (ADH). This excretion of ADH results in an inappropriate urine concentration and urine osmolality above 100 mOsm/kg H_2O with hypotonic plasma. Additional medical conditions that can produce hyponatremia are excessive ingestion of water secondary to a psychogenic cause (polydipsia) and syndrome of inappropriate ADH (SIADH), which occurs in patients with certain infections and tumors. Treatment is the administration of hypertonic saline with furosemide diuresis in symptomatic patients and isotonic saline in asymptomatic patients.

Hypervolemic Hyponatremia. Hypervolemic hyponatremia is a direct result of an excess of sodium and water when the amount of water gain exceeds the sodium gain. Medical conditions associated with hypervolemic hyponatremia include heart failure, renal failure, and hepatic failure. The presence of sodium in the urine in the clinical presentation may be misleading in patients for whom diuretics are used.

Treatment for these patient is diuresis induced by furosemide, with strictly monitored hypertonic saline levels. Treatment is determined by the extracellular volume and neurologic status of the patient. A rapid correction of the sodium level may lead to central pontine myelinolysis, a condition that can lead to permanent CNS damage or death. The recommended rate of rise in sodium plasma should be no more than 0.5 mEq/liter/hr and the final plasma concentration should not exceed 130 mEq/liter. The goal of replacement is 130 mEq/liter. The formula for sodium replacement is as follows:

$$\text{Sodium deficit (mEq)} = \text{normal TBW} \times (130 - \text{current plasma sodium}) \times (32.8)$$

where TBW is total body water (in liters), 60% of the lean body weight in men (in kg) and 50% of the lean body weight in women (in kg).

Potassium

Potassium is the most important intracellular cation of the intracellular fluid. The normal serum potassium concentration is 3.5 to 5.5 mEq/liter. A total body potassium deficit of 200 to 400 mEq will reduce the serum potassium by 1 mEq/liter; a potassium excess of 110 to 200 mEq will raise the concentration of serum potassium by 1 mEq/liter.

Hypokalemia. Hypokalemia is present when the potassium level falls below 3.5 mEq/liter. Severe hypokalemia serum levels below 2.5 mEq/liter may present with muscle weakness. Mild cases are asymptomatic. In 50% of these cases, electrocardiographic changes may be present with U waves, flattening or inversion of T waves, and prolonged T waves. Possible causes include the following:

1. Transcellular shift: Uptake of beta agonists (minimal effect), alkalosis, hypothermia, and insulin
2. Potassium depletion: Depletion secondary to renal or extrarenal loss
   Renal loss: The main cause of renal loss is diuretics, nasogastric tubes, alkalosis, and magnesium depletion (impairs potassium absorption).
   Extrarenal potassium loss: The major cause is diarrhea.

Treatment. The health care provider must first stop the condition that is causing the potassium shift and then replace the potassium. The most common form is potassium chloride as fluid replacement (1 and 2 mEq/mL) in ampules containing 10 to 40 mEq of potassium; these solutions are hyperosmotic and have to be diluted. Potassium, 20 mEq, is usually added to 100 mL of isotonic saline and is infused over a 1-hour period. The maximum dose is 20 mEq/hr and a central line should be used because of the irritant properties of the solution. Oral potassium administration is another form of replacement. If the potassium levels do not rise as expected, the magnesium levels should be checked

Hyperkalemia. Hyperkalemia occurs when the serum concentration level increases to more than 5.5 mEq/liter. Significant hyperkalemia can be life-threatening, with the most dangerous manifestation being slowing of the electrical conduction of the heart. Initially, it will present as tall T waves in V2 and V3; as hyperkalemia progresses, the PR segment lengthens, as well as the QRS segment, culminating in ventricular asystole.

It is important to distinguish hyperkalemia from pseudohyperkalemia, in which traumatic hemolysis occurs from venipuncture, potassium released from the muscles distal to a tourniquet, or potassium released from cells in a clot formation in the specimen. Hence, whenever there is an asymptomatic patient, the test should be repeated. Hyperkalemia could be caused by cellular potassium release or impaired renal excretion. A urine potassium level higher than 30 mEq/liter suggests a transcellular shift, whereas a potassium level lower than 30 mEq/liter is more consistent with problems caused by impaired renal excretion.

Acidosis is related to hyperkalemia when associated with renal failure and tubular acidosis. Renal failure occurs when the glomerular filtration rate falls below 10 mL/min or total urinary output is less than 1 liter/day. Drugs associated with the impairment of renal excretion of potassium by blocking the renin-angiotensin-aldosterone system include angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, potassium-sparing diuretics, NSAIDs, heparin, trimethoprim-sulfamethoxazole, and pentamidine.

Digitalis toxicity is related to hyperkalemia by the transcellular potassium shift. In addition, rhabdomyolysis and acidosis with impaired renal function and massive blood transfusions in shock patients contribute to hyperkalemia.

Treatment. This is guided by the level of potassium present.

1. Electrocardiographic changes or potassium level more than 7 mEq/liter:
   - Calcium gluconate 10%, 10 mL IV over 10 minutes, repeat every 5 minutes; response lasts 20 to 30 minutes; do not give bicarbonate after calcium.
   - Electrocardiographic changes and circulatory compromise—calcium chloride 10%, 10 mL over 3 minutes (contains three times more calcium than calcium gluconate).
2. AV block refractory to calcium treatment:
   - 10 U of regular insulin in 500 mL of 20% dextrose; infuse in 1 hour (should drop serum potassium by 1 mEq/liter for 1 or 2 hours).
   Treatment includes transvenous pacemaker.
3. Digitalis toxicity:
   - Magnesium sulfate, 2-g IV bolus; digitalis antibodies if necessary (do not use calcium).
4. Postacute phase:
   - Sodium polystyrene (Kayexalate), PO, 30 g in 50 mL of 20% sorbitol or rectal dose of 50 g in 200 mL 20% sorbitol as retention enema; oral route is better tolerated; if renal failure is present, hemodialysis is indicated.

Magnesium

Magnesium is the second most abundant intracellular cation and works as a cofactor for more than 300 enzyme reactions. The normal value for an average adult is 24 g of magnesium.

Magnesium deficiency is common in hospitalized patients. It is frequently affiliated with other electrolyte abnormalities of calcium and phosphate. Patients may present with arrhythmias, with the most serious being torsade de pointes. In addition, there are neurologic manifestations of altered mentation, seizures, tremor, and progressive obtundation. Common causes of magnesium deficiency include diuretic therapy, antibiotic therapy, digitalis, epinephrine, alcoholism, secretory diarrhea, diabetes mellitus, acute myocardial infarction, and the chemotherapeutic agents cisplatin and cyclosporine,

Magnesium Replacement Therapy. Oral and parenteral solutions are available. Parenteral solutions are preferred. The standard preparation is magnesium sulfate, 1 to 4 g IV, added to current bag, 100 mL over 2 to 3 hours. A 50% sulfate solution has to be diluted to 10% or 20% for IV use because of its osmolality. Saline solution should be used; calcium in Ringer's solution will counteract magnesium.

Magnesium Excess. Magnesium excess presents with hyporeflexia, weakness, altered consciousness, bradycardia, respiratory failure, nausea and vomiting. AV blocks with progressive cardiac arrest can occur with the elevation of serum magnesium levels. Causes: Exogenous administration, rhabdomyolysis, adrenal insufficiency, hemolysis, impaired renal function, hyperparathyroidism and lithium intoxication. To treat magnesium excess, give 10 mL of calcium gluconate 10% over 10 to 20 minutes in 50 to 100 mL of dextrose 5% in water (D5W), one ampule D50 with 5 to10 U of regular insulin IV, furosemide 20 mg IV.

DIALYSIS

Calcium. Calcium is the most abundant electrolyte in the body; 99% of the body's calcium is stored in bones. The calcium level in the body 9.0 to 10 mg/dL; the ionized calcium leve is 4.5 to 5.0 mg/dL.

Hypocalcemia. This is caused by magnesium depletion, sepsis, alkalosis, blood transfusions, renal failure, pancreatitis and drugs such as aminoglycosides, cimetidine, and heparin.

Clinical manifestations of hypocalcemia include the following:

1. Neuromuscular excitability (tetany, hyperreflexia, paresthesias, and seizures). Chvostek's sign is nonspecific and Trousseau's sign is insensitive.
2. Cardiovascular manifestations. Hypotension, ventricular ectopic activity, decreased cardiac output; heart block, and ventricular tachycardia are seen in more severe hypocalcemia.

Treatment of hypocalcemia consists of identifying and treating the cause first. For symptomatic hypocalcemia, infusion of calcium in a central vein is preferred. However, with a peripheral vein, calcium gluconate is administered; 200 mg of calcium is given as a bolus in 100 mL of isotonic saline over 10 minutes, followed by an infusion of 1 to 2 mg of elemental calcium/kg/hr for 6 to 12 hours.

Hypercalcemia. Hypercalcemia is present in cases of hyperparathyroidism or malignancy; less common conditions are immobilization, thyrotoxicosis, and some drugs (e.g., lithium, thiazide diuretics).

Clinical manifestations of hypercalcemia include the following:

1. Gastrointestinal: Nausea, vomiting, ileus, constipation, pancreatitis
2. Renal: Nephrocalcinosis, polyuria
3. Cardiovascular: Hypotension, hypovolemia, shortened QT interval
4. Neurologic: Confusion, altered mental status

Treatment. If the patient is symptomatic or if the serum calcium level is higher than 14 mg/dL (ionized calcium > 3.5 mg/dL), infuse IV fluids (saline) to restore volume, correct hypovolemia, and induce calcium excretion. This is followed by the administration of furosemide (40 to 80 mg every 2 hours) to promote calcium elimination. For malignancy, calcitonin will prevent bone resorption, with 4 U/kg given every 12 hours. Hydrocortisone is given in addition to calcitonin in a dosage of 200 mg IV daily, divided in two or three doses. Dialysis is used for patients with hypercalcemia and renal failure.

Phosphorus

Most of the body phosphorus is located in the skeleton (85%); the rest is present as free phosphorus (15%). The normal values are 2.5 to 5.0 mg/dL.

Hypophosphatemia. Causes include diabetic ketoacidosis, glucose loading, respiratory alkalosis, beta receptor agonists, sepsis, and phosphate binding agents. Hypophosphatemia may affect aerobic energy production and present as muscle weakness. This occurs as a result of impaired myocardial contractibility, reduced cardiac output, hemolytic anemia, and depletion of 2,3-diphosphoglycerate, which deviates the oxyhemoglobin dissociation curve to the left.

To treat severe hypophosphatemia without adverse effects, with a phosphate level below 1 mg/dL, administer Sodium Phosphate or Potassium Phosphate 0.6 mg/kg/hr IV. To treat hypophosphatemia with adverse effects, with a phosphate level below 2 mg/dL, administer Sodium Phosphate or Potassium Phosphate 0.9 mg/kg/hr. Serum phosphate levels should be checked every 6 hours.

Hyperphosphatemia. Most cases of hyperphosphatemia are related to renal failure with impaired excretion or release from widespread cell necrosis (rhabdomyolysis or tumor lysis). Clinical manifestations usually present with hypocalcemia and tetany as a consequence.

Treatment of hyperphosphatemia includes the following:

1. Calcium acetate tablets (PhosLo) will raise the serum calcium and lower serum phosphorus levels.
2. Promote phosphorus binding in the GI tract with sucralfate or aluminum-containing acids.
3. GI dialysis should be initiated.

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CHAPTER

5

Emergency Airway Management in the Traumatized Patient

Alisha Moreno | Jacob G. Calcei | Michael P. Powers

OUTLINE

Systematic Approach to Airway Management
Initial Assessment: Recognition of Airway Obstruction and Breathing
Airway Maneuvers and Adjuncts
Bag-Valve-Mask Ventilation
Adjuncts to Intubation
Endotracheal Intubation
Adjuncts to Intubation
Surgical Airway
Pediatric Considerations
Anatomy
Tips for Specific Intubation Procedures on Children

Traumatic injuries to the head, neck, and maxillofacial structures can easily compromise an individual's ability to get oxygen to their lungs. If oxygen is unable to reach the lungs and diffuse through alveolar walls to be picked up by red blood cells, a number of adverse consequences occur, ranging from loss of consciousness to organ failure and eventual death. It is only a matter of seconds to minutes before an individual loses consciousness in the absence of oxygen. As a result, successful trauma intervention consists of the ability to establish and maintain a patent airway, including proper ventilation, while avoiding aspiration.

Airway-related medical interventions have been documented for centuries. The Bible contains references to airway interventions dating back as far as 850 BC.[1,2] More recently, in 2007, the National Confidential Enquiry into Patient Outcome and Death published a report regarding trauma care in the United Kingdom. The study found that although 19.8% of patients presented noisy or blocked airways, the failed intubation rate for prehospital intubations was 12%.[3] Another study, from 2010, focused on the airway management of combat-injured patients in Operation Iraqi Freedom. Although the effectiveness of body armor has improved, the head and neck regions are often unprotected and susceptible to serious injury. The high-velocity, penetrating trauma associated with war only adds to the difficulty of securing a patent airway in these patients. Of the 196 patients in the study, 68% of them had potentially lifesaving airways placed.[4] This high percentage reinforces the priority of the airway and the role of a head and neck or airway specialist in times of trauma.

Despite recent major medical advancements, the basic priorities and principles of airway management remain the same. The American College of Surgeons Advanced Trauma Life Support (ATLS) ABCDE mnemonic is as follows: **A**, airway maintenance with cervical spine protection; **B**, breathing and ventilation; **C**, circulation with hemorrhage control; **D**, disability, the neurologic status of the patient; and **E**, exposing or undressing the patient to facilitate a complete physical examination.[5] This chapter focuses on the first two steps.

SYSTEMATIC APPROACH TO AIRWAY MANAGEMENT

The approach to airway management in the patient who has suffered from trauma is as follows:

1. Initial assessment: Recognize airway obstruction.
2. Perform airway maneuvers, clear the airway, and reposition the patient
3. Use artificial airways, and perform bag-valve-mask ventilation
4. Perform endotracheal intubation.
5. Create a surgical airway if unable to intubate.

Recognizing a patient in clear respiratory distress requires little to no clinical medical experience. On the other hand, it takes a greater degree of clinical experience and knowledge to identify at risk patients prior to the onset of an airway problem. When possible, it is advisable to intervene before a major crisis occurs by anticipating problems and taking action early. The stepwise approach should be used as a general guideline and not as a rigid set of rules that cannot be modified. For example, there are times when an emergency cricothyrotomy is indicated, before attempting earlier, futile efforts.

INITIAL ASSESSMENT: RECOGNITION OF AIRWAY OBSTRUCTION AND BREATHING

The primary goal of the initial assessment is to determine if the injuries sustained have compromised the airway and/or breathing in the patient (Figs. 5-1 and 5-2). A failure to recognize the need for an airway or the inability to establish an airway can lead to death.[6] Injuries that result from direct airway trauma are dynamic and require frequent reexamination (Tables 5-1 and 5-2). A standard

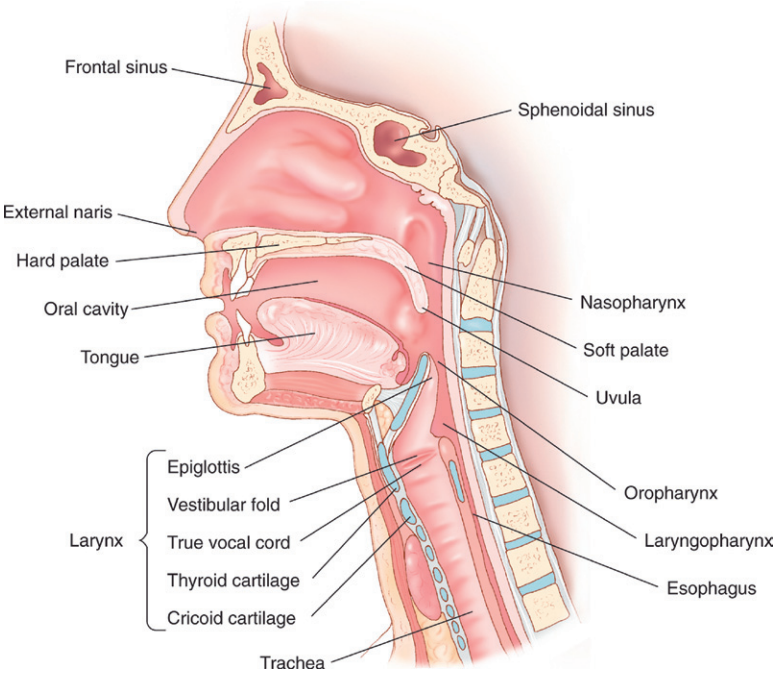


FIGURE 5-1 Airway anatomy. (From Chapleau W, Pons P: *Emergency medical technician*, St. Louis, 2007, Mosby/JEMS.)



FIGURE 5-2 Airway damaged by burns.

TABLE 5-1 Causes of Airway Injury Specifically Related to Maxillofacial Trauma

Unstable facial fractures	Hemorrhage
Soft tissue swelling and edema	Displacement of larynx and trachea
Smoke inhalation	Airway burns
Hematoma	Head injury, reduced consciousness
Aspiration caused by fractured teeth, bone fragments, vomitus, blood, debris, foreign body obstruction	

Adapted from references 7, 8, and 9.

TABLE 5-2 Signs of Airway Compromise

Direct	Indirect	Developing
Dyspnea	Tracheal deviation or other anatomic abnormality of larynx or trachea	Nonsuperficial neck or face burns (see Fig. 5-2)
Stridor	Painful swallowing	Severe bleeding in oropharynx or nasopharynx
Signs of bruising on neck because of underlying laryngotracheal injury	Trismus	Hematoma in neck or lower face
	Drooling	Subcutaneous air in neck or upper chest Hoarseness or other voice alterations Sense of shortness of breath, despite adequate oxygen saturation

Adapted from references 8 and 10.

"look, listen, and feel" approach is often used to identify breathing difficulties.

Look. Patients with a compromised airway tend to sit upright because they do not tolerate the supine position well. A look of panic or terror may accompany obvious breathing irregularities such as gasping, retraction, heaving, quick but shallow breaths, and large gulping breaths. Anxiety or agitation may be a sign of hypoxia and the patient should be treated as such until proven otherwise. Obtundation, on the other hand, may be a sign of hypercapnia and the patient's quality of air movement should be examined. The oropharynx should be inspected using a light source and tongue blade to evaluate injuries and to clear the airway of blood, emesis, secretions, loose teeth, bone fragments, or foreign bodies.[6,11-13]

Listen. A quick test of airway patency is to have the patient describe the injury. If they are able to speak clearly and coherently, in full and nonlabored sentences, the patient is unlikely to require immediate airway assistance. Chest sounds should be noted to determine the quality of air flow. It should be assumed that an unresponsive patient has a compromised airway until proven otherwise. Obstruction of the airway causes noisy breathing such as stridor, gurgling, or wheezing. Foreign body upper airway obstruction, secretions in the airway, soft tissue damage, and respiratory tract irritation are all potential causes of an obstructed airway.[10,11,14]

Feel. It is of importance to feel for facial fractures, soft tissue swelling, subcutaneous air in the neck, laryngeal tenderness, and dental injuries during the primary assessment. Additionally, all blunt trauma patients should be assumed to have a cervical spine injury until examinations show otherwise. In the absence of imaging studies, a trauma patient can be cleared of a cervical spine injury if they exhibit the following[6,11]:

- Neurologically intact
- Not under the influence of drugs
- Free of tenderness in posterior midline of cervical spine
- Free of distracting, painful injuries.

In cases in which the patient is neurologically unstable, the cervical spine must be immobilized until further examinations can clear the patient of such an injury.[12]

Assessing for Potentially Difficult Laryngoscopy

The clinician must be able to quickly determine the potential for a difficult airway in each patient before choosing the best intubation method (Fig. 5-3). The inability to recognize general characteristics associated with a difficult airway can lead to a failed airway. The mnemonic LEMON describes a series of physical evaluations that assist in determining the difficulty of the airway[9,15]:

L: Look externally. What is the general impression of the airway? Is there any unusual anatomy or facial trauma that should be considered?

E: Evaluate (3-3-2 rule). Take note of the following measurements: mouth opening, mandible size, and distance between the mentum and hyoid bond. If these measurements are not represented in the 3-3-2 relationship (Box 5-1), the possibility for a difficult laryngoscopy remains (Fig. 5-4).

M: Mallampati score. This assessment serves as a predictor for difficult intubation. By estimating the space for oral intubation, in addition to determining the mouth opening and the size of the tongue, the Mallampati score gives clinicians another tool for assessing potentially difficult airways. There are four classes:

- Class I: Clear visibility of tonsillar pillars, uvula, soft palate, and fauces
- Class II: Tongue obscures view of tonsillar pillars but soft palate, uvula, and fauces are visible
- Class III: Only soft palate and base of uvula visible
- Class IV: No soft palate visibility[9,15]

Classes I and II on the Mallampati spectrum should result in an easy laryngoscopy. Difficulty should be expected with class III; the most extreme cases of difficult airways are class IV (Fig. 5-5).

In cases of severe trauma or unconsciousness, when the patient is unable to assist in the Mallampati assessment, the clinician should use a laryngoscope blade or tongue blade and attempt to open the mouth and examine the tongue-to-oropharynx ratio. If this test shows a large tongue to oropharynx ratio, a difficult laryngoscopy should be anticipated.[9,15]

O: Obstruction, obesity. An upper airway obstruction can block access to the airway and make it difficult to view the glottis. In obese patients, the extra tissue layers in the upper airway make direct laryngoscopy more challenging.

N: Neck mobility. Because maxillofacial trauma patients are assumed to have a cervical spine injury until otherwise cleared, the patient may not be able to maintain the proper positioning for an intubation. If possible, patients are put in the sniffing position prior to intubation. In cases of cervical spine immobilization during intubation, the view of the glottis is limited. Neck mobility is also limited in patients with severe rheumatoid arthritis and ankylosing spondylitis.

Beyond the LEMON test, other potentially difficult situations include airway burns, notable facial trauma with bleeding and swelling of the soft tissues, and patients with previously difficult intubations. Securing an airway in trauma patients is vital and clinicians must anticipate these potentially complex scenarios before they occur to increase the chances of maintaining a successful airway.[9,15]

AIRWAY MANEUVERS AND ADJUNCTS

Basic Maneuvers

Once it has been determined that an airway is obstructed because of maxillofacial trauma, basic airway maneuvers are used (Fig. 5-6). The most basic airway maneuvers are the chin lift and jaw thrust. In the case of an unconscious patient, these simple techniques may be enough to restore the airway if the obstruction was caused by the tongue or relaxed upper airway muscles. Severe fractures of the mandible can also cause the tongue to become displaced posteriorly, leading to an obstructed airway.[6,16,17]

In a patient who has not been cleared of a cervical spine injury, these maneuvers must be done without significant neck extension. The chin lift (Fig. 5-7) is performed to open up the hypopharynx. It is done with two fingers under the point of the chin and jaw while the soft tissue is lifted forward. The jaw thrust (Fig. 5-8) is

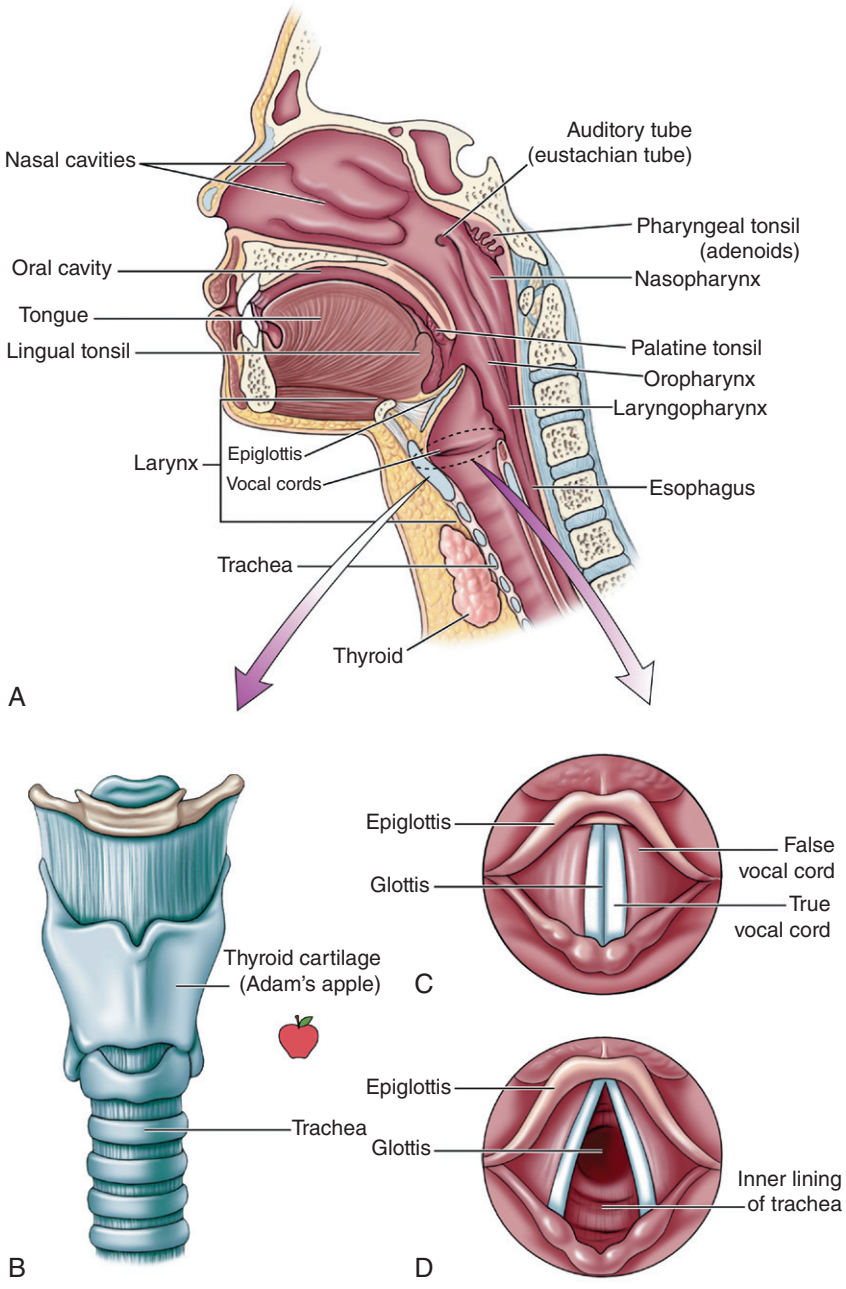


FIGURE 5-3 Anatomy for direct laryngoscopy. (From Herlihy B, Maebius N: *The human body in health and illness*, Philadelphia, 2000, Saunders.)

BOX 5-1 3-3-2 Rule for Difficult Airway Assessment: LEMON Test

3—A normal* patient can open the mouth wide enough for three of his or her own fingers to fit between the incisors. This eases airway access and facilitates the insertion of the laryngoscope and the view of the glottis.
3—A normal* patient can place three of his or her own fingers between the mentum and the neck-mandible junction near the hyoid bone. This is an estimation of the volume of the submandibular space.
2—A normal* patient can place two fingers in the space between the superior notch of the thyroid cartilage and the neck-mandible junction near the hyoid bone, also called the superior laryngeal notch. This identifies the location of the larynx in relation to the base of the tongue. The positioning of the larynx is important in difficult airway assessment.

*A normal patient in this case signifies a person for whom a difficult laryngoscope is not expected.
Adapted from references 9 and 15.

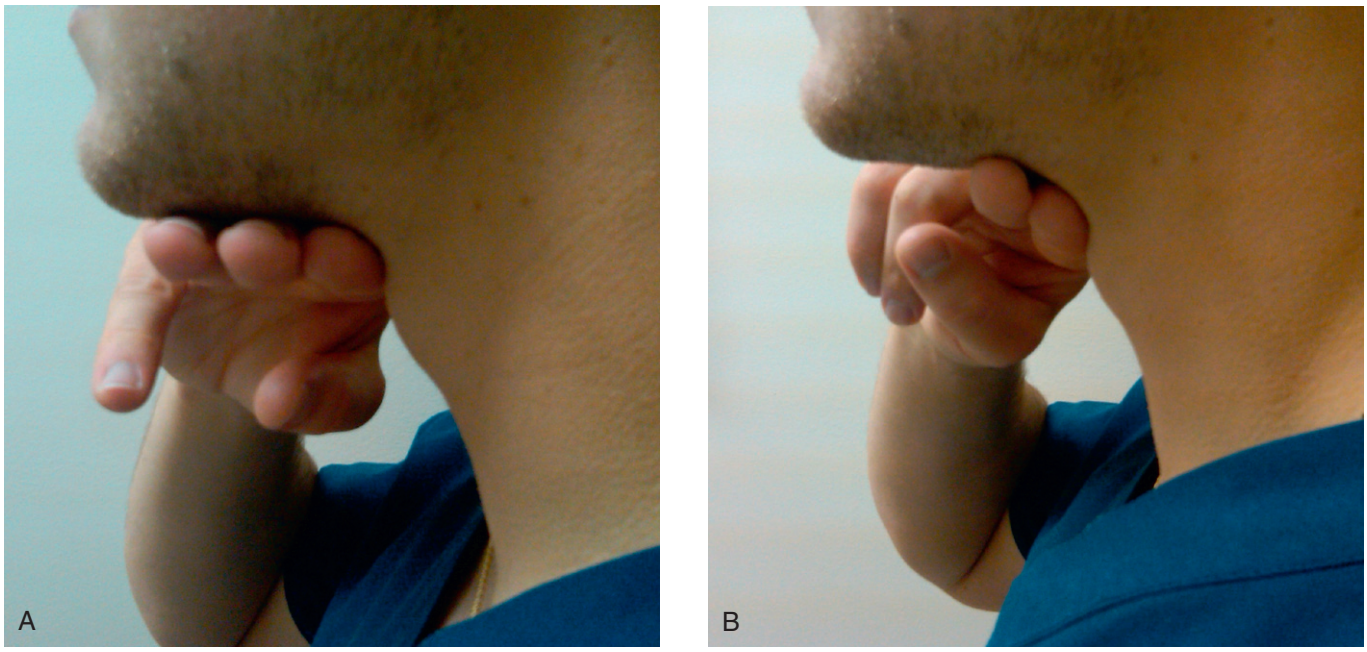


FIGURE 5-4 3-3-2 rule. **A,** Three fingers are placed along the floor of the mouth beginning at the mentum. **B,** Two fingers are placed in the superior laryngeal notch.

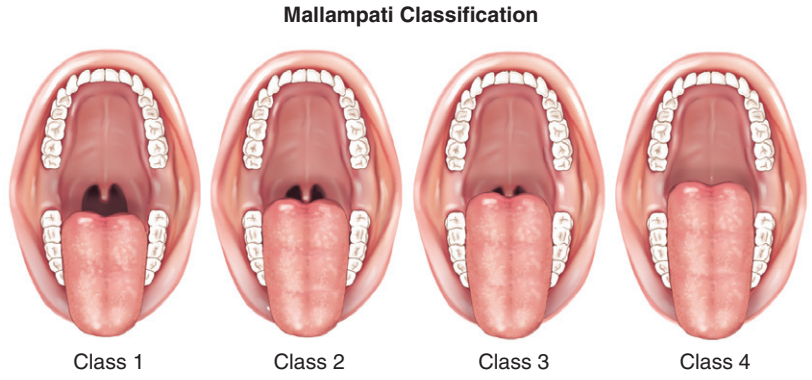


FIGURE 5-5 Mallampati classifications.

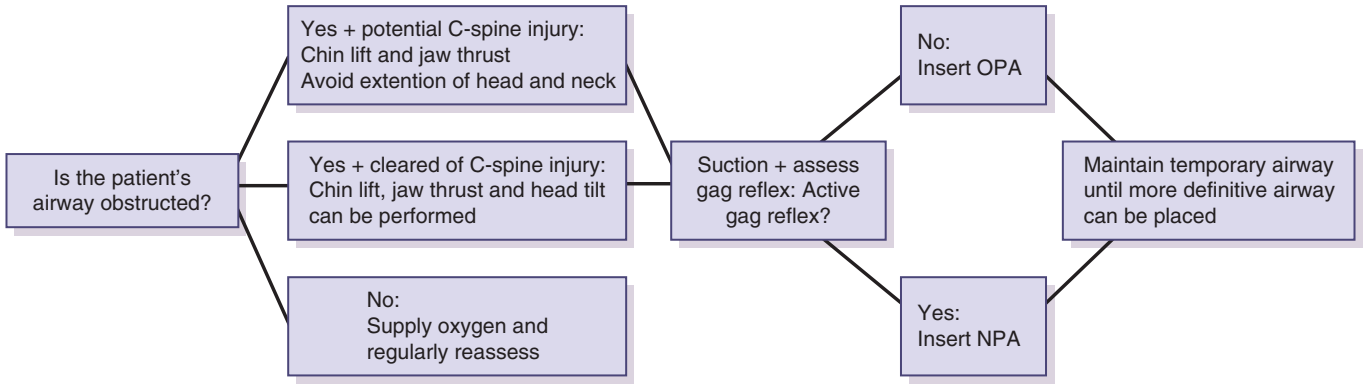


FIGURE 5-6 Basic maneuvers of initial airway management for maxillofacial trauma.

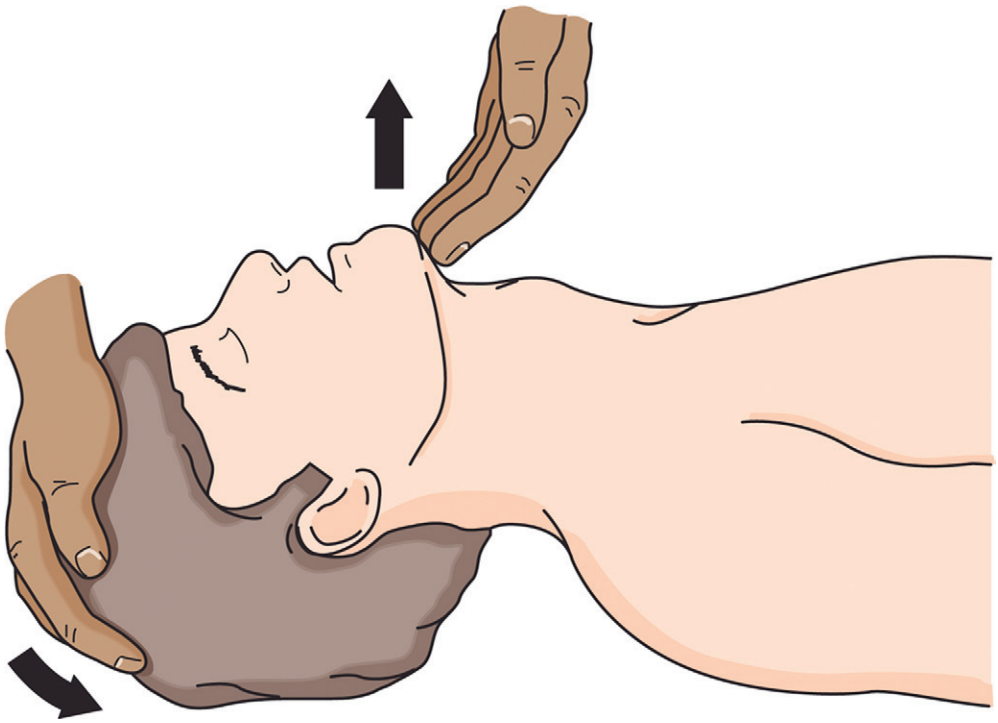


FIGURE 5-7 Chin lift, head tilt. (From Monahan F, Sands J, Neighbors M, et al: *Phipps' medical-surgical nursing*, ed 8, St. Louis, 2007, Mosby.)



FIGURE 5-8 Jaw thrust. (From Malamed SF: *Medical emergencies in the dental office*, ed 6, St. Louis, 2007, Mosby.)

performed with the physician behind the patient. The knuckles of the index fingers are placed behind the angle of the mandible on each side, and the physician's thumbs apply pressure on the cheek bones at the same time that the jaw thrust lifts and displaces the mandible forward. The jaw thrust without head extension is especially important in cases of suspected spinal injury.[6,10,16,18]

The chin lift and head tilt (see Fig. 5-7) can be performed if the patient has been cleared of a cervical spine injury. These can be done by grasping the mandible and lifting up while rotating the occiput caudad.[6]

The suctioning of debris such as broken teeth or bones, other foreign objects, or secretions should be completed once the basic maneuvers have been performed. This is done carefully to avoid gagging a conscious patient, which could lead to vomiting and a more serious situation. [10]

Adjuncts

Oropharyngeal (Fig. 5-9) and nasopharyngeal airway devices (Fig. 5-10) can be effective in temporarily maintaining the airway in a patient, but these adjuncts are not considered definitive airway management. Rather, these devices are used until the patient is ready for intubation.[6,11]

The oropharyngeal airway (OPA) should be used in a patient who lacks an active gag reflex. In adult patients, the size of the OPA is measured so that it reaches from the mouth angle to the ear lobe. When placed in the mouth, the OPA (Fig. 5-11; see Fig. 5-9) is inserted upside down and turned 180 degrees once it reaches the level of the uvula (Table 5-3). This technique helps the device to push the tongue down anteriorly and away from the posterior pharyngeal wall. It should be emphasized that



FIGURE 5-9 Oropharyngeal airway. (From Malamed SF: *Medical emergencies in the dental office*, ed 6, St. Louis, 2007, Mosby.)



FIGURE 5-10 Nasopharyngeal airway. (From Stoy W, Platt T, Lejeune D: *Mosby's EMT—basic textbook*, ed 2, St. Louis, 2007, Mosby.)

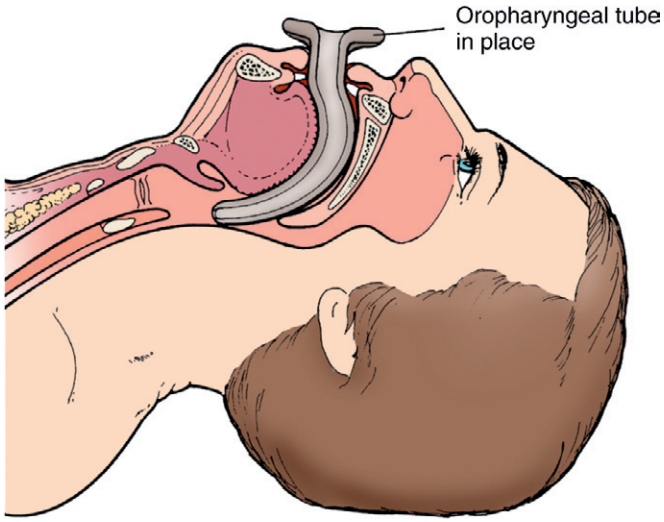


FIGURE 5-11 Insertion of oropharyngeal airway (OPA). (From Marshak AB: Emergency life support. In Wilkins RL, Stoller JK, Scanlan CL, editors: *Egan's fundamentals of respiratory care*, ed 8, St. Louis, 2003.)

TABLE 5-3 Technique for Placement of Oropharyngeal Airway

Step	Instructions
1	Use suction to clear mouth of secretions and assess gag reflex; if no gag reflex present, then continue.
2	Select OPA size; OPA should be measured so that it stretches from the corner of the mouth to the angle of the mandible.
3	Insert device upside down so that it curves upward.
4	Rotate OPA into proper position so that tongue is pulled forward and away from pharynx.

Adapted from references 6, 10, and 11 and American Heart Association: *ACLS advanced cardiovascular life support provider manual: Professional*. Chicago, 2011, American Heart Association.

TABLE 5-4 Technique for Placement of Nasopharyngeal Airway

Step	Instructions
1	Select NPA size.
2	Prepare NPA by applying lubricant with local anesthetic.
3	Gently insert device while passing it along the nasopharynx; slightly rotate NPA during insertion if met with light resistance.

Adapted from references 10 and 11 and American Heart Association: *ACLS advanced cardiovascular life support provider manual: Professional*. Chicago, 2011, American Heart Association.

the most critical part of the assessment is the presence or absence of the gag reflex. If the reflex can be stimulated, do not use the oropharyngeal airway.[6,10,11,16]

An alternative to the oropharyngeal airway is the nasopharyngeal airway (NPA). The NPA provides a passage of airflow between the nares and pharynx and can be used in patients who are conscious or semiconscious, with an intact gag reflex (Table 5-4). The device is prepped with lubricant, ideally one that contains a local anesthetic agent, before it is placed in the larger nasal passage. The length of the NPA should approximate the distance between the end of the patient's nose and the ear lobe (Fig. 5-12). If there is slight resistance during the insertion of the device, it should be rotated slightly to allow it to pass down behind the tongue and into the oropharynx. If there is stiff resistance during device insertion, a smaller NPA should be selected to fit the patient's nasal passage better. Because of its small size, it is not uncommon for the NPA to become obstructed. Consequently, the airway should be regularly reevaluated until an airway is definitively established.[10,11,16]

BAG-VALVE-MASK VENTILATION

If other basic maneuvers and adjuncts are unsuccessful in maintaining a patent airway, bag-valve-mask (BVM) ventilation is performed to achieve oxygenation and ventilation for the patient.[19] Bag-mask devices are the most common method of providing positive-pressure

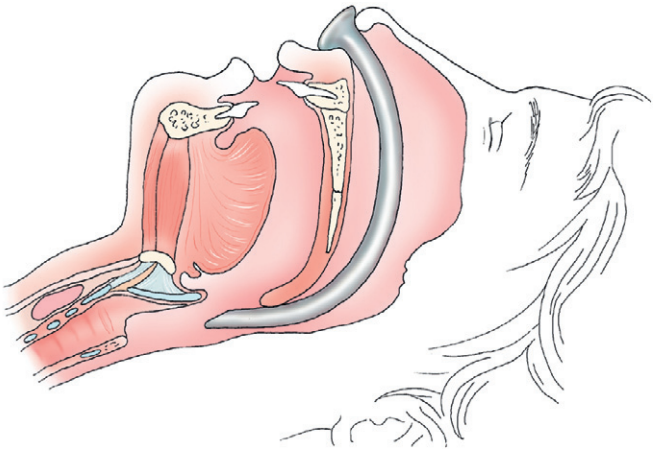


FIGURE 5-12 Inserted nasopharyngeal airway (NPA). (From McSwain N: The basic EMT: *Comprehensive prehospital care*, ed 2, St. Louis, 2003, Mosby.)



FIGURE 5-13 Bag-valve-mask. (Courtesy Sedation Resource, Lone Oak, Texas.)

ventilation[16] (Fig. 5-13). The importance of this skill cannot be understated. If done correctly, it can serve as a definitive airway in many cases. When comparing the survival rates of trauma patients who had prehospital endotracheal intubations to those who had BVM ventilations, it was found that there was no survival advantage to endotracheal intubations when the severity of their injuries were taken into account.[20]

Basic airway adjuncts and maneuvers are often performed in conjunction with BVM ventilation to assist in the maintenance of the airway. This includes the use of oropharyngeal and nasopharyngeal airways, along with the jaw thrust, chin lift, and head tilt techniques. The equipment needed for proper BVM ventilation includes the bag-valve device, masks of different sizes, oxygen source, oxygen connector tubing, pulse oximeter, suction and vacuum power source, OPAs or NPAs, and water-based lubricant.[21]

There are three main techniques for BVM ventilation. Ideally, three individuals should be assigned to this task. Because this is not always feasible, it is imperative that providers are adept at completing this alone, with one



FIGURE 5-14 Two-hand technique for BVM ventilation. (From Thomsen T, Setnik G, editors: *Procedures consult—emergency medicine module*, St. Louis, 2008, Elsevier.)

partner, or as part of a three-person team. The preferred method for BVM ventilation requires two people, and is known as the two-hand or two-provider technique (Fig. 5-14). The patient is positioned so that the ear is in line with the sternal notch.[22] An OPA or NPA should already be in place and the mask size should be chosen. A correctly sized mask fits just below the eyes, does not extend past the chin, and completely covers the nose and mouth.[21] This allows the mask to create a good seal and provide more effective ventilation. With two providers present, one is responsible for delivering the bag ventilations. According to the 2011 Advanced Cardiac Life Support (ACLS) guidelines, this should be done at an approximately 600-mL tidal volume to produce chest rise over 1 second.[16] Gastric inflation is one major risk of BVM ventilation, but the established tidal volume of 600 mL minimizes this risk.[23] The second provider should secure the mask on the patient's face by creating a C shape with the thumb and index finger of each hand and gently pressing down on the mask. By using the remaining fingers to lift up on the mandible, the second provider can simultaneously perform the chin lift and hold the mask in place while the first provider delivers bag ventilations. In rare cases in which a third provider is available, they should supply cricoid pressure to avoid excess air in the esophagus and stomach while remaining aware of the potential for airway obstruction because of the cricoid pressure.[11,21]

An alternative method for the two-hand technique (Fig. 5-15) uses different hand positioning for the provider responsible for securing the mask. Both thumbs face away from the provider and run parallel to each other as the thenar eminences hold the mask against the patient's face and the remaining fingers lift the mandible.[19]

When only one provider is available, the one-hand E-C technique is used (Figs. 5-16 and 5-17). The thumb and index finger of the nondominant hand form a C shape over the mask and apply pressure. The remaining fingers lift up the mandible to perform the jaw thrust, creating the E shape.[19]

The mnemonic MOANS is used to identify potential difficulties with BVM ventilation (Box 5-2). It is not



FIGURE 5-15 Alternate two-hand technique for BVM ventilation. (From Thomsen T, Setnik G, editors: *Procedures consult—emergency medicine module*, St. Louis, 2008, Elsevier.)



FIGURE 5-16 One-hand EC technique for BVM ventilation. (From Malamed SF: *Sedation: A guide to patient management*, ed 5, St. Louis, 2010, Mosby.)

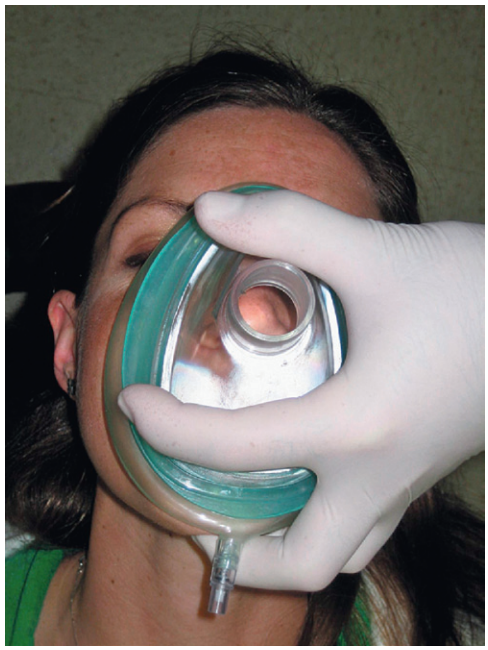


FIGURE 5-17 One-hand E-C technique *(top view)*. (From Malamed SF: *Sedation: A guide to patient management*, ed 5, St. Louis, 2010, Mosby.)

BOX 5-2 MOANS Test for Difficult Bag-Valve-Mask Ventilations

M: Mask seal—facial hair, abnormal facial anatomy, and excessive bleeding or vomiting may lead to an ineffective mask seal. A water-soluble lubricant can be used for patients with facial hair to improve the seal.
O: Obstruction or obesity—an upper airway obstruction or a BMI > 26 can lead to difficulty with BVM ventilation.
A: Age—patients older than 55 years, although not an exact age cutoff, tend to have less elasticity of the tissues and therefore are at higher risk for difficult BVM ventilation.
N: No teeth—teeth provide a natural framework for the mask to rest against. If a patient wears dentures, they should be left in during BVM ventilation to increase the chances for success.
S: Stiffness—this may be a sign of asthma, chronic obstructive pulmonary disease (COPD), or pulmonary edema.

Adapted from references 9 and 15.

uncommon for providers to hyperventilate patients during BVM ventilation and extra care should be taken to avoid this whenever possible. The American Heart Association cautions that the negative impacts of hyperventilation include increased intrathoracic pressure, reduced venous return, diminished cardiac output, and decreased coronary and cerebral perfusion.[24]

ENDOTRACHEAL INTUBATION

According to various studies, between 9% and 28% of trauma patients undergo tracheal intubation.[25] Although the exact percentage may be debatable, the failure to secure a definitive airway in a trauma patient is undoubtedly a major cause of preventable death.[3]

Indications

A definitive airway consists of a cuffed tube that is secured in the trachea.[26] Table 5-5 provides a list of indications that a definitive airway should be promptly secured. Providers should err on the side of caution when deciding whether or not to place a definitive airway to avoid the challenges of securing a crash airway.[14]

There are four major questions outlining the decision to perform an intubation[14:]

1. Is there a failure of airway maintenance or protection? A loss of airway reflexes, pooling secretions signifying the inability to swallow, and tolerance of the placement of nasopharyngeal airway or oropharyngeal airways indicate a patient's inability to maintain and protect their airway.
2. Is there a failure of oxygenation? This is illustrated by clinical symptoms of cyanosis, obtundation, restlessness or agitation, and by low oxygen saturation as measured by a pulse oximeter. A lack of oxygen

TABLE 5-5 Indications for Definitively Secured Airway

Apnea	Glasgow Coma Scale score < 9
Sustained seizure activity	Unstable midface trauma, maxillofacial fractures
Airway injury or obstruction	Cardiac arrest
Persistent hypoxia (SpO_2 < 90%)	High aspiration risk, blood, vomit
Inability to obtain adequate seal with facemask; insufficient ventilation	Shock; systolic BP < 80 mm Hg
Anticipation of impending airway compromise	Hypoventilation
Inability to maintain patent airway via other techniques	Severe hypoxemia
Combative patient	

Adapted from references 11, 12, 26, and 27.

can cause irreversible damage to human tissues in only minutes.

3. Is there a failure of ventilation? This can be observed through the patient's mental status and observing the patient's respirations. The inability to release carbon dioxide from the body adequately leads to altered mental status and respiratory acidosis.
4. Is there an anticipated need for intubation? It is absolutely imperative that providers assess the likely progression of symptoms and anticipate impending airway compromise whenever possible.[14]

Contraindications

Because of the importance of securing an airway, there are few contraindications to intubation.[28] The only major contraindication to intubation is the ability to maintain a patent airway in a less invasive manner.[11] If it is possible to maintain a patent airway through basic adjuncts and maneuvers, advanced airway measures should be avoided. Possible cervical spine injury is not a contraindication for intubation; however, immobilization of the cervical spine should be maintained throughout the procedure in cases in which the patient's spine has not been cleared of injury.[28] There are several contraindications to specific types of intubation (see later).

Types of Endotracheal Tubes

Endotracheal tubes have many shapes and sizes. The differences in shapes may vary slightly and should be selected based on the specific type of intubation. In general, endotracheal tubes have the following characteristics: two open ends, a 15-mm adapter for a bag-valve or ventilator device, measurements on the side, a large distal port and small side port to reduce chances for occlusion, and a low-pressure, high-volume cuff.[11] The cuff is in place to secure a seal with the patient's trachea, helping minimize leakage during positive pressure ventilation. Additionally, the cuff centers the tube to avoid damaging any surrounding mucosa.[29] The cuff can be safely inflated to 10 mL initially but this can be adjusted as needed. Overinflation (>30 mm Hg) of the cuff can cause mucosal necrosis and should be avoided.[6]

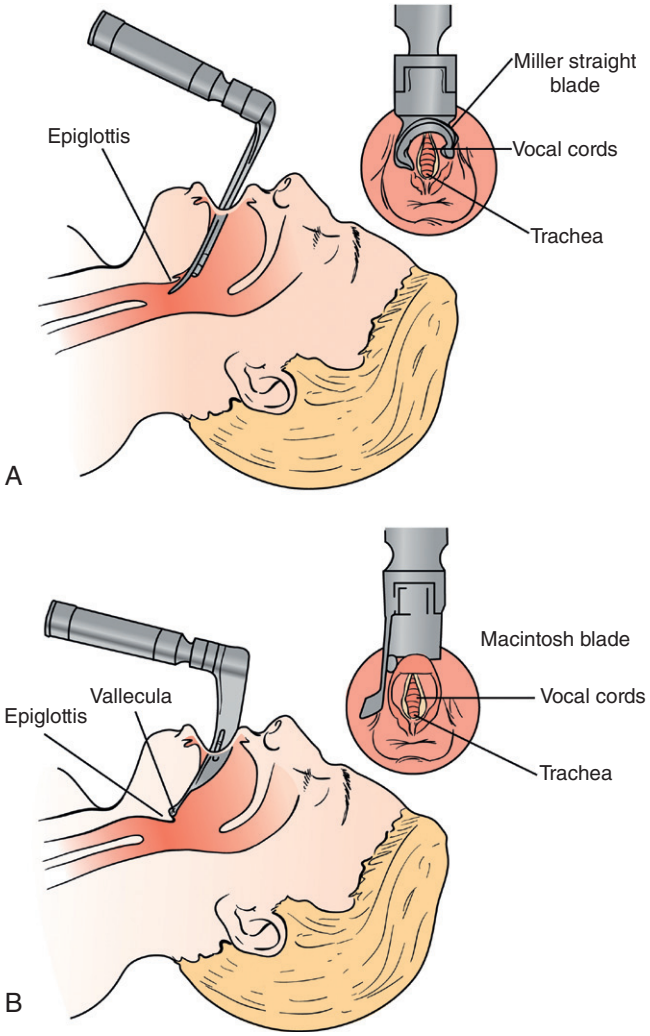


FIGURE 5-18 The epiglottis **(A)** and vallecula **(B)** are important landmarks when performing direct laryngoscopy. (From Stoy W, Platt T, Lejeune D: *Mosby's EMT—basic textbook*, ed 2, St. Louis, 2007, Mosby.)

The average man will tolerate an endotracheal tube with an internal diameter of 7.5, 8.0, or 8.5 Fr. The average woman, however, requires a slightly smaller tube of 7.0 or 7.5 Fr in most cases. Tubes that are met with resistance at the vocal cords should be substituted for a smaller one to prevent tracheal damage.[6]

Direct Laryngoscopy

A direct laryngoscopy is a procedure that uses a short rigid instrument to deflect the tongue and jaw, allowing for a view of the larynx and the placement of a tube within the trachea.[30] The epiglottis and vallecula are important anatomic landmarks to identify before performing a laryngoscopy (Fig. 5-18; see Fig. 5-3). The epiglottis, found at the base of the tongue, covers the underlying glottis; the vallecula is a cleft between the tongue and epiglottis.[31]

To move forward with the procedure, it is important to have the proper equipment available and easily

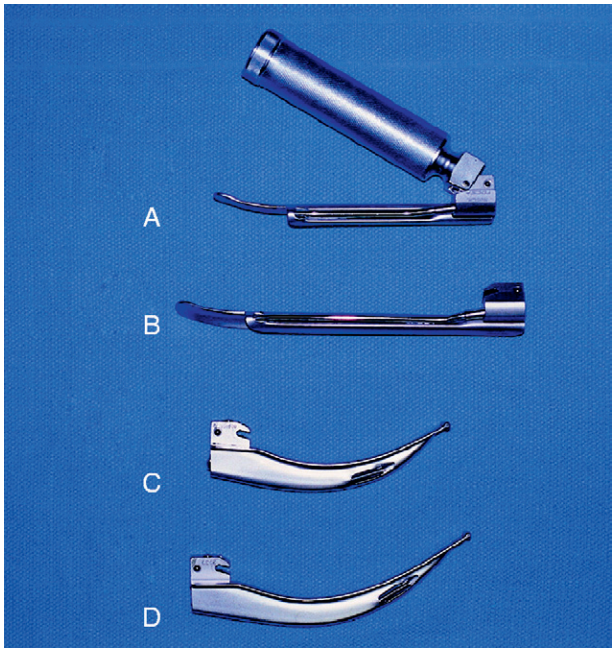


FIGURE 5-19 Laryngoscope blades. The most common laryngoscope blades are the Miller and Macintosh blades. **A,** Miller no. 2. **B,** Miller no. 3. **C,** Macintosh no. 3. **D,** Macintosh no. 4. (From Spiro S, Albert R, Jett J: *Clinical respiratory medicine*, ed 3, St. Louis, 2009, Mosby.)

accessible. All equipment should be checked to avoid the use of any damaged parts. The necessary equipment includes the following[31]:

- Laryngoscope handle with various blades (Fig. 5-19)
- Endotracheal tubes of various sizes
- Stylet
- Introducer
- Oropharyngeal and nasopharyngeal airways
- Rescue airway devices—laryngeal mask airway, Combitube
- Monitors—blood pressure, heart, pulse oximeter, end-tidal carbon dioxide
- Esophageal detector
- Tape

A laryngoscope blade has a light source attached and can be curved (Macintosh) or straight (Miller).[11] If the straight blade is used, it should be inserted under the epiglottis so that the glottis becomes exposed. Alternatively, the curved blade should be placed into the vallecula.[32] The Macintosh blade tends to provide a better view in adult trauma cases.[6]

Positioning. The positioning of the patient is critical during direct laryngoscopy. In a recent study, 10% of the 3400 emergent intubations performed were classified as difficult because they required multiple attempts.[33] Proper positioning gives the clinician the best chance of success on the initial attempt. Experienced clinicians should expect to be successful on the first attempt in most cases. However, in difficult airways, multiple attempts may be required before the intubation is completed. Multiple intubation attempts should be avoided, when possible, because they are associated with increased morbidity in emergent situations or with cardiopulmonary instability.[31]

If the patient has not been cleared of a cervical spine injury, the spine must remain immobilized throughout the procedure. The front of the neck collar is often removed to improve the clinician's view, but an assistant must stabilize the neck manually. The inability to position the patient may create a more difficult laryngoscopy.[11]

In situations in which the patient is cleared of a cervical spine injury, he or she is placed in a supine position, with an elevated head (5 to 7 cm) and flexion of the neck, to align the pharyngeal and laryngeal axes. The patient's head is extended at the atlanto-occipital joint so that the oral axis is in line with the other two[31,34] (Fig. 5-20).

Technique of Direct Laryngoscopy: Cleared Cervical Spine. The STOP MAID mnemonic device can be used as a reminder about the steps and tools of intubations[31]:

S: Suction
T: Tools (e.g., blade, handle.)
O: Oxygen
P: Positioning
M: Monitors (electrocardiogram [ECG], O_2, CO_2, blood pressure [BP])
A: Assessment, airway devices, assistant
I: IV access
D: Drugs

Direct laryngoscopy begins immediately after the initial assessment is made. The assistant positions the patient's body and opens her or his mouth while the clinician grabs the laryngoscope in the left hand. The right hand should be free to suction, manipulate the larynx, and insert the endotracheal tube.[34] The curved blade is then positioned along the right side of the tongue, displacing the tongue out of sight and to the left. This allows the epiglottis to come into view so that it can be lifted to expose the glottis.[32] The tongue and epiglottis are elevated as the clinician pulls the left hand up and away while keeping the elbow in close to the body. When the epiglottis is identified, the curved blade is advanced until it reaches the vallecula.[11] At this time, the endotracheal tube is inserted, its placement is confirmed, and the tube is secured in place.[31] The straight blade is advanced in a similar manner, but it can be advanced deeper into the hypopharynx and may be obscured by the tongue. A paraglossal technique can be performed with the straight blade to displace the tongue properly and create a better view. For this technique, the blade is inserted between the tongue and lower right molars and advanced between the tongue and tonsillar pillar until the epiglottis can be seen. At this point, the tongue is displaced laterally and anteriorly.

Confirming Tube Placement

Proper endotracheal tube (ETT) placement can be confirmed clinically by visualization of the tube passing through the vocal cords, observation and auscultation of the chest, and condensation in the ETT from exhalation.[35] In many cases, this is not possible or there may be false results, so other methods must be used. The gold standard for verifying proper ETT placement is

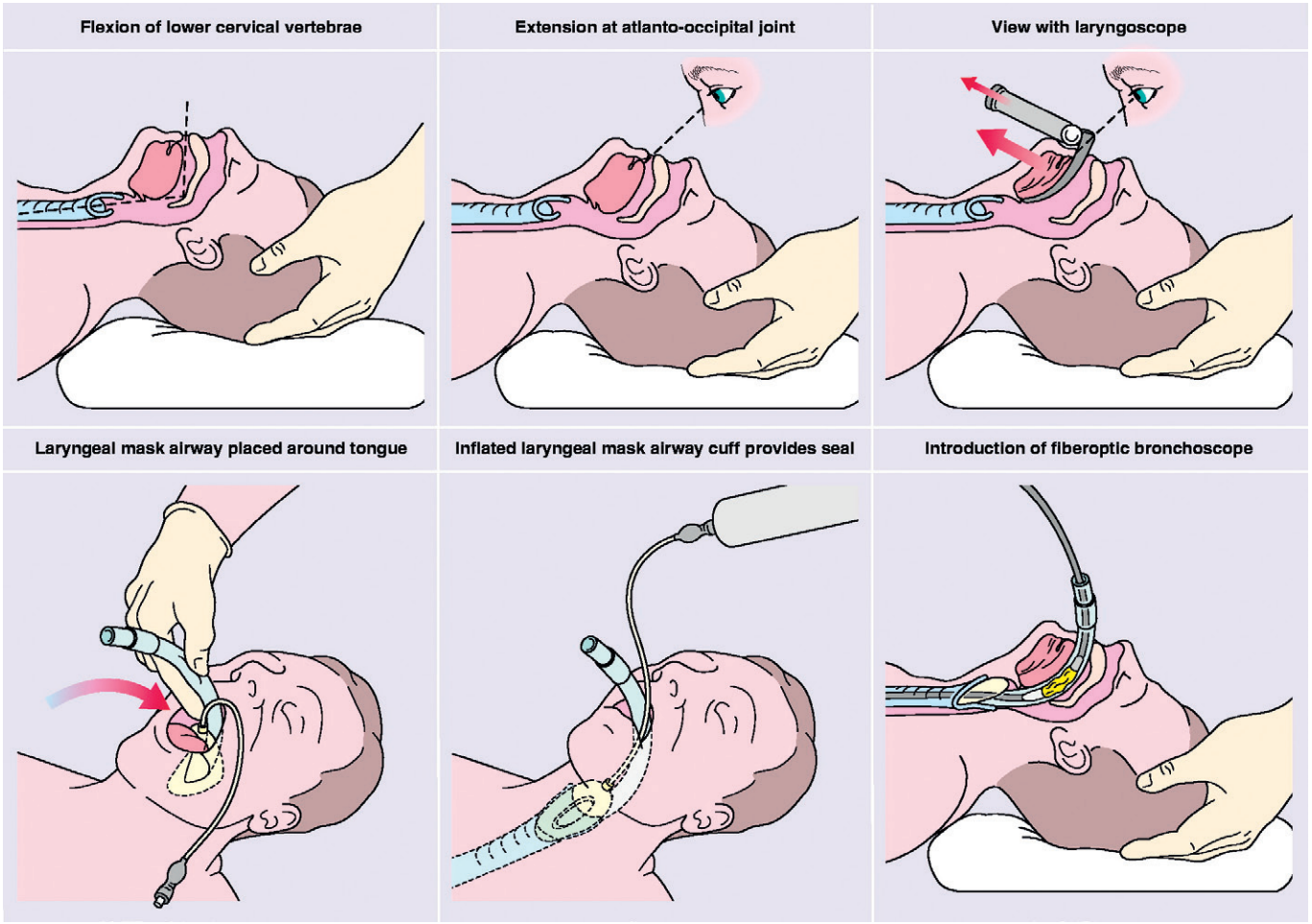


FIGURE 5-20 Positioning during laryngoscopy with cleared C-spine. (From Spiro S, Albert R, Jett J: *Clinical respiratory medicine*, ed 3, St. Louis, 2009, Mosby.)

capnometry, which measures exhaled CO_2 from the tube. When the ETT is positioned in the trachea, the carbon dioxide levels should not drop significantly over time. If positioned in the esophagus, however, these levels will drop continuously to near zero. False-positives can occur if the patient has recently ingested a carbonated beverage, causing carbon dioxide levels in the stomach to be higher than normal.[11]An esophageal detection device can also be used to verify the position of the ETT, although this device is not 100% accurate.[36]

Capnometry and esophageal detection devices provide information immediately, enabling the clinician to perform another intubation if the tube is placed into the esophagus by mistake.[37] The use of an ETT introducer also helps confirm proper placement of the ETT, because the rings of the trachea can be felt as the introducer tip is advanced. The introducer tip will also stop abruptly once it reaches the bronchus. If the ETT is in the esophagus instead, the tracheal rings will not be felt, and there will be no abrupt stopping point.[38] A chest x-ray provides a view of the depth of ETT placement, but is unable to differentiate between the esophagus and trachea.[34] The ideal depth is 5 ± 2 cm above the tracheal carina[35] (Fig. 5-21).

Although all these methods are acceptable, it is important to note that a physical examination is not sufficient for the confirmation of proper ETT placement.[37] Ultimately, confirmation of proper ETT placement should be done using a number of methods to be sure of the results.

Nasotracheal Intubation

In most cases of maxillofacial trauma, nasotracheal intubation is contraindicated because of the potential risk of brain injury. Fractures of the basilar skull and cribriform plate are especially susceptible to this type of injury.[39] Clinicians should also avoid this procedure with pediatric patients, and those showing signs of traumatic brain injuries or rising intracranial pressure.[11] On the other hand, if difficulty is anticipated with other airway techniques, and if the clinician is experienced with this method, nasotracheal intubation should be considered. When a conscious patient needs airway intervention, nasotracheal intubation may be preferred because of the patient's active gag reflex. This technique places less pressure on the tongue and therefore leads to less gagging.[32]

Properly sized nasotracheal tubes are 0.5 to 1.0 sizes smaller than oral tubes. Other necessary materials include a topical vasoconstrictor anesthetic and fiberoptic bronchoscope, which is placed through the nasotracheal tube. The patient is put in the sniffing position with immobilization measures taken in case of cervical spine

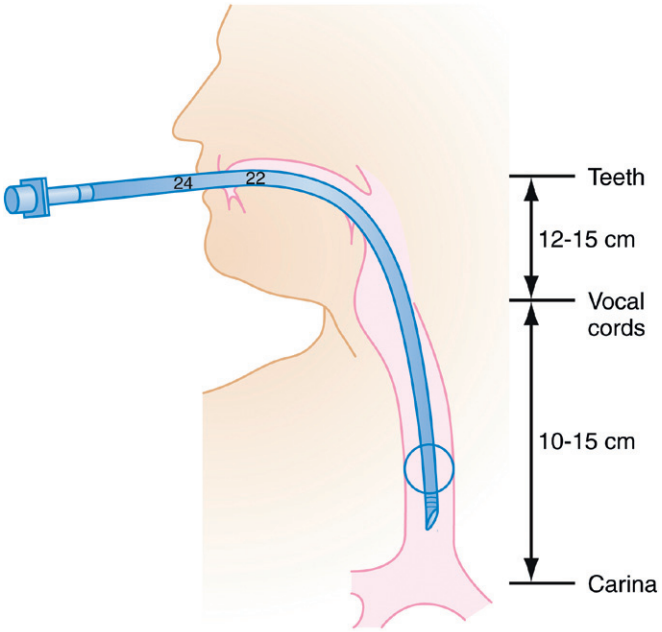


FIGURE 5-21 Endotracheal tube positioning. (From Bogdonoff DL: Airway considerations in the management of patients requiring long-term endotracheal intubation. *Anesth Analg* 74:276, 1992.)

injury. The tube should be warm and lubricated before it is advanced into the larger naris along the nasal floor. To minimize potential damage to the Kiesselbach plexus, the bevel of the tube should face the septum.[34] The tube is advanced past the vocal cords and eventually down into the larynx on inspiration, where a cough indicates proper placement. Slow and steady insertion of the bronchoscope and nasotracheal tube, along with slight rotation when needed, should be sufficient to overcome slight resistance. As with other methods of intubation, if the tube is met with stiff resistance, the tube should not be forced any further. The procedure should then be attempted through the other side or a smaller sized tube may be required.

The ideal depth of placement for a nasotracheal tube is 28 cm for men and 26 cm for women, as measured at the nares. As with orotracheal intubation, proper tube placement should be confirmed. Vocal cord sounds of any type signify a failed attempt at nasotracheal intubation, which may be modified by repositioning or rotating the tube.[34]

Rapid-Sequence Intubation

The standard of care for securing an emergency airway in semiconscious or conscious patients is rapid-sequence intubation (RSI).[38] The success rate of this procedure by properly trained individuals is greater than 97%, according to various studies.[11] RSI uses sedation and paralysis to facilitate intubation and minimize potential risks. The steps of RSI are known as the seven Ps[6]:

1. Preparation
2. Preoxygenation
3. Premedication and induction
4. Paralytic
5. Pressure (cricoid)
6. Placement
7. Postintubation Care

Preparation. All necessary personnel, equipment, and medication should be ready on arrival of the patient. The equipment should be inspected for integrity prior to being used.

Preoxygenation. The patient is placed on 100% oxygen through a rebreather mask for 3-5 minutes to create an oxygen reservoir in the lungs prior to paralysis. This helps KEEP the patient's blood oxygen saturation from dropping to dangerous levels (<90%) during the intubation procedure. If the patient does not respond to the rebreather mask, a bag-valve mask device can be used instead.[40,41]

Premedication and Induction. When time permits, it is optimal to administer premedication to the trauma patient prior to induction and intubation. These agents, given approximately 3 minutes before induction, have the ability to mitigate the physiologic responses associated with intubation.[40] The mnemonic device LOAD is used to identify the premedication step of RSI[42]:

L: Lidocaine
O: Opioids (typically fentanyl)
A: Atropine
D: Defasciculating agent (vecuronium, rocuronium)

Table 5-6 details the four components of premedication. Induction begins a few minutes after the premedication agents are administered. The induction agents have a sedative effect and are given prior to the paralytic agent. The five most common induction agents will be discussed, but only one should be given during RSI. The induction agent is selected based on patient-specific characteristics.[42]

Induction Agents

Etomidate. In the case of maxillofacial trauma, etomidate is the most commonly used induction agent. This hypnotic agent has a very short half-life (≈5 minutes) and is hemodynamically neutral. The proper dosage is 0.3 mg/kg IV for children and adults.[43] However, because of its short half-life, administration of etomidate is typically followed by another sedative agent, such as propofol or midazolam.[6] The major drawback of etomidate is that it can cause adrenal suppression. It should not be used in patients suffering from septic shock.

Ketamine. A dissociative anesthetic agent, ketamine is unique because of its amnestic effects in addition to its sedative and analgesic properties. It stimulates the cardiovascular system and relaxes smooth muscle, making it especially useful for patients suffering from hypotension or bronchospasms.[11] The proper dosage is 1 to 2 mg/kg IV or 3 to 4 mg/kg IM.[6] The major drawback of ketamine is increased intracranial pressure (ICP); therefore, it should not be used in patients with traumatic brain injury.[42]

Propofol. This hypnotic agent is a lipid-soluble, alkyl phenol derivative, which causes sedation and amnesia. Propofol has a short half-life, reduces ICP, and has an antiepileptic effect, making it a desirable induction agent. However, it is also a myocardial depressant and is associated with significant hypotension. The proper dosage is 1 to 2 mg/kg IV bolus for children and adults.[40]

TABLE 5-6 Premedication for Rapid-Sequence Intubation

Drugs	Dosage	Effects	Cautions
Lidocaine	1.5 mg/kg IV	Suppresses airway reactivity; reduces intracranial pressure; reduces hypertensive response	Hypotension
Fentanyl	2-8 µg/kg IV	Sedative and analgesic effects; reduces intracranial pressure; hemodynamically nonreactive	Patients with severe shock Hypotension
Atropine	0.02 mg/kg IV, children 0.5-1.0 mg, adults	Treats bradycardia and asystole; reduces secretions	Only for use in children up to age 5 years Or Adults with Bradycardia
Rocuronium	0.08-1.2 mg/kg (10% of paralytic dosage)	Counteracts increase in ICP caused by succinylcholine; prevents fasciculations	High doses may result in paralytic effect (>0.06 mg/kg)

Adapted from references 40 and 41 and from Muskat PC: Emergency airway management in the traumatized patient. In Fonseca RJ, Turvey TA, Marciani RD, editors: *Oral and maxillofacial surgery*, ed 2, St. Louis, 2008, WB Saunders, pp 25–34.

Midazolam. This benzodiazepine causes sedation and amnesia, but also has antiepileptic properties. The proper dosage is 0.1 mg/kg IV, but it is typically underdosed as a precaution for its major drawback, hypotension.[40] The speed of onset varies by patient, but tends to be slower than etomidate.

Sodium Thiopental. An fast-acting barbiturate, sodium thiopental is known for causing deep amnesia and sedation, although it lacks analgesic characteristics. It is especially helpful for patients suffering from traumatic brain injuries because of its cerebroprotective properties.[11] Drawbacks of sodium thiopental include inhibition of the sympathetic central nervous system (CNS) response, lack of availability, and hypotension. The proper dosage is 3 to 5 mg/kg IV in adults and 5 to 8 mg/kg IV in children.[41]

Paralytic Agents. To achieve optimal intubation conditions, a paralytic agent is administered after the induction agent. Although the induction agent is a sedative with analgesic and amnestic effects, the paralytic agent causes the relaxation of skeletal muscle. Through this relaxation of the facial muscles, the clinician gets a better view during laryngoscopy, which facilitates an easier intubation and reduces the chance of complications.[42,44]

Paralytics are known as neuromuscular blocking agents (NMBAs) and are classified in two distinct groups:

1. Depolarizing—act on motor end-plate to activate acetylcholine receptors
2. Nondepolarizing—act on motor end-plate to inhibit acetylcholine receptors

Succinylcholine. This depolarizing NMBA is the most commonly used paralytic. It is fast-acting, in less than 1 minute, and has a short half-life. The use of paralytics leads to an apneic patient, emphasizing that they should only be used under the administration of appropriately trained clinicians. The proper dosage is 1.5 to 2 mg/kg IV.[11] There are some major contraindications to the use of succinylcholine in RSI (Box 5-3). In the presence of any contraindications, the use of succinylcholine should be avoided.

Rocuronium. When succinylcholine is contraindicated, a nondepolarizing NMBA is used instead. The most common nondepolarizing NMBA used in RSI is rocuronium. Similar to succinylcholine, rocuronium is fast-acting and creates ideal intubation conditions, but its paralytic effects last significantly longer. Although other alternatives such as vecuronium and pancuronium exist, rocuronium is the best alternative to succinylcholine.[42] The proper dosage is 1 mg/kg IV.

BOX 5-3 Contraindications to Succinylcholine

- Personal or family history of malignant hyperthermia
- Denervation of muscles because of neuromuscular disease
- Muscular dystrophy
- Stroke (>72 hr old)
- Rhabdomyolysis
- Burn (>72 hr old)
- Significant hyperkalemia

Adapted from references 40, 42, and 44.

Cricoid Pressure. The literature exploring cricoid pressure and its role in RSI is inconclusive. Some sources include it as a necessary step, whereas others point to a lack of definitive support and consider it optional.[40] When proper technique is not used, cricoid pressure can obscure the view of the glottis, impede passage of the ETT, and prevent adequate ventilation.[38] On the other hand, if it is done correctly, it can facilitate intubation and reduce potential complications.

Cricoid pressure (Sellick maneuver) is performed following induction and is maintained until the ETT is properly placed.[38] One assistant performs the Sellick maneuver by using the thumb and middle or index finger to apply pressure to the cricoid cartilage. It is performed after the anterior portion of the cervical collar is removed while another assistant maintains cervical spine immobilization. There are three reasons to apply cricoid pressure[6]:

1. To avoid insufflation of the esophagus and stomach during BVM ventilation, which reduces the risk of vomiting and passive regurgitation. If active vomiting occurs at any time, cricoid pressure should be immediately released to prevent a ruptured esophagus.

2. To prevent passive regurgitation after induction and sedation agents are administered.
3. To manipulate the larynx for optimal vocal cord visualization and to improve the view of the glottis through use of the BURP method (backward-upward-rightward pressure).

If the Sellick maneuver (Fig. 5-22) is performed, the assistant should be careful to avoid the common error of applying pressure to the thyroid cartilage.

Placement of the Endotracheal Tube. The next step in RSI is the placement of the ETT. This step is addressed earlier in the chapter, where direct laryngoscopy is discussed in more detail.

Postintubation Care and Management. Immediately after placement of the ETT is confirmed, the tube should be secured in place with tape (Fig. 5-23). If the Sellick maneuver was performed, cricoid pressure can be stopped at this time. The cervical collar is put back in place if it was taken apart at any stage of RSI. A chest x-ray is obtained to verify proper ETT placement.[38]

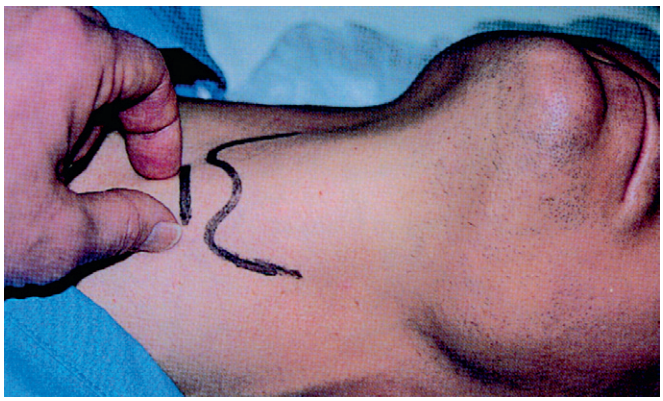


FIGURE 5-22 Sellick maneuver. The cricoid cartilage is identified by palpation below the thyroid cartilage. Firm pressure is placed on this structure to occlude the esophagus. Pressure is maintained until after intubation; airway control is documented by auscultation of the lung fields and end-tidal CO_2. (From Spiro S, Albert R, Jett J: *Clinical respiratory medicine*, ed 3, St. Louis, 2009, Mosby.)



FIGURE 5-23 Secure the ETT with tape. (From Perry A, Potter P: *Clinical nursing skills and techniques*, ed 7, St. Louis, 2006, Mosby.)

Further treatment can be administered at this time, because a patent airway has been secured.

ADJUNCTS TO INTUBATION

When difficult laryngoscopy is expected, adjuncts to intubation are used to facilitate easier placement of the ETT.[31] The tracheal tube introducer, or gum elastic bougie (Fig. 5-24), is inexpensive and easy to use. This plastic wand acts as a guide for the endotracheal tube. It is placed through the vocal cords during direct laryngoscopy. The insertion of the bougie is easier than placement of the ETT because of its smaller diameter.[39]

Introducer. Although it is contraindicated in the presence of maxillofacial trauma because of its blind insertion, the lighted stylet (Fig. 5-25) is another useful adjunct to intubation. Unlike the bougie, the lighted stylet takes significant practice before mastery. Once mastered, the stylet has a high success rate.[11] The advantage of the lighted stylet is that it does not require direct laryngoscopy. The ETT fits over the stylet, which has a bent, illuminated distal end. When properly positioned in the trachea, the stylet illuminates the skin of the neck just above the thyroid notch. If it is placed in the esophagus, the light will not be visible.[32]

Rescue Airway Devices. The laryngeal mask airway (LMA) consists of a curved tube and inflatable cuff. This rescue device is used as a noninvasive way to provide ventilation. Another version of this device, called the LMA Fastrach, allows passage of an ETT blindly through the cuff and into the trachea. The LMA Fastrach can accommodate a no. 3 or 4 ETT for women and a no. 4 or 5 ETT for men.[6]

The LMA is easy to use and has few potential complications. Using the thumb and index finger, the LMA is lubricated, placed into the mouth, and pressed against the hard palate. The LMA follows the curvature of the

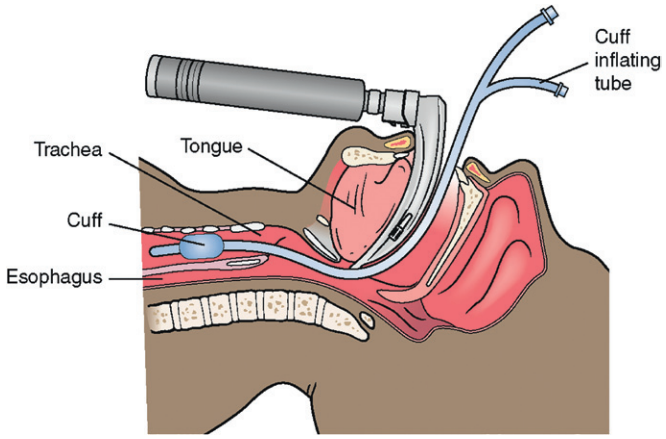


FIGURE 5-24 The tracheal tube introducer. (From Buck C: Step-by-step medical coding St. Louis, 2009, WB Saunders.)

tongue to the back of the mouth and is advanced into the oropharynx with the index finger. The device is then pushed even further down the hypopharynx before the index finger is removed.[32] Once in its final position, the LMA cuff should be inflated to between 20 and 30-mL of air and secured in place[6] (Fig. 5-26). It is important to note that according to ATLS guidelines, the LMA is not a secure airway device and does not protect the patient against aspiration.[11]

The esophageal-tracheal Combitube (Kendall-Sheridan, Argyle, NY) is another rescue airway device that is used as a backup for failed intubation. It is a double-lumen tube that is usually reserved for emergent situations because of the potential for esophageal injury.[39] The presence of supraglottic obstruction is an indication for use of this device; the Combitube is unique because of its ability to provide ventilation from placement in the esophagus or trachea.[32] Limited device sizes are available, so the Combitube can only be used on adults taller than 5 feet. The Combitube consists of two cuffs; the larger, pharyngeal cuff seals the oronasopharynx and the distal cuff seals at the trachea or esophagus (Fig. 5-27). The proximal cuff is sealed first, followed by the distal cuff.[11] The side holes on the tube aid in ventilation. Similar to the LMA, the Combitube is not considered a definitively secure airway and is performed as a precursor to placement of an ETT or tracheostomy.



FIGURE 5-25 The lighted stylet. (From Cote C, Lerman J, Todres ID: *A practice of anesthesia for infants and children*, ed 4, St. Louis, 2009, Saunders.)

Alternative Intubation Techniques

Fiberoptic-Assisted Intubation. This instrument is used for difficult intubations, but may not be available in all emergency departments. Spontaneous breathing in the patient is an indication to try this method.[10] A conscious patient can assist in the procedure by sticking out the tongue to keep it out of the way. The fiberoptic scope is placed within the ETT before it is advanced into the trachea.

Retrograde Intubation. A previous study has shown that patients with maxillofacial fractures are successfully managed at the trauma site during the first attempt at retrograde intubation.[10] This technique involves passing a guidewire through a needle inserted into the larynx and delivering that wire through the mouth or nose to guide the endotracheal tube. Topical anesthesia is applied to the oropharynx as lidocaine is injected at the cricothyroid membrane and into the larynx. The guidewire is passed into the needle, back through the larynx, and up into the oropharynx. Forceps are used to retrieve the guidewire in the mouth. Finally, the ETT follows the path of the guidewire into the trachea.[32]

Awake Laryngoscopy. If there is a risk of both aspiration and a difficult airway, an awake laryngoscopy may be performed. Some slight sedation is used to facilitate patient cooperation by reducing their protective reflexes. The necessary effect for successful laryngoscopy is produced by 30 mg/kg of midazolam in addition to 1.5 mcg/kg of fentanyl .[32] A topical anesthetic may also aid in the facilitation of this procedure. Patients at risk for an expanding hematoma would be candidates for an awake laryngoscopy. Because the patient remains conscious, it is important that the clinician explain the steps of the procedure.

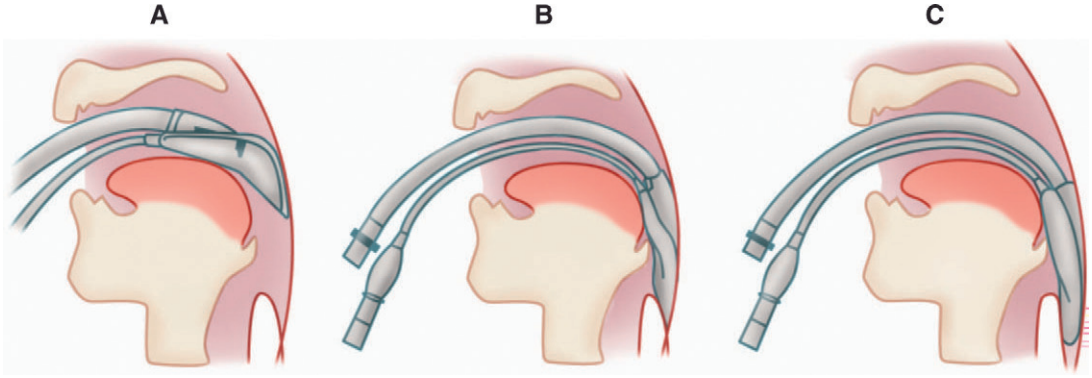


FIGURE 5-26 Insertion and proper placement of the laryngeal mask airway (LMA). **A,** Insertion of LMA. **B,** Proper location of LMA (deflated). **C,** Properly placed and inflated LMA. (From Rothrock J: *Alexander's care of the patient in surgery*, ed 13, St. Louis, 2007, Mosby.)

Extubation

Before the endotracheal tube is removed, the patient's respiratory sufficiency is observed. If the inspiratory capacity of the patient is measured at 15 mL/kg or greater, and the patient is able to protect his or her own airway, the clinician can continue with extubation.[34] During this process, the patient is in an upright position and the ETT and oral cavity are suctioned. The tube is removed in one smooth motion on exhalation of a deep breath. After removal of the tube, the mouth is suctioned again and supplemental oxygen is administered. Immediately after extubation, the patient is monitored for changes in vital signs. Positive-pressure ventilation may be used as a method to prevent laryngospasms and respiratory failure after extubation.[45]

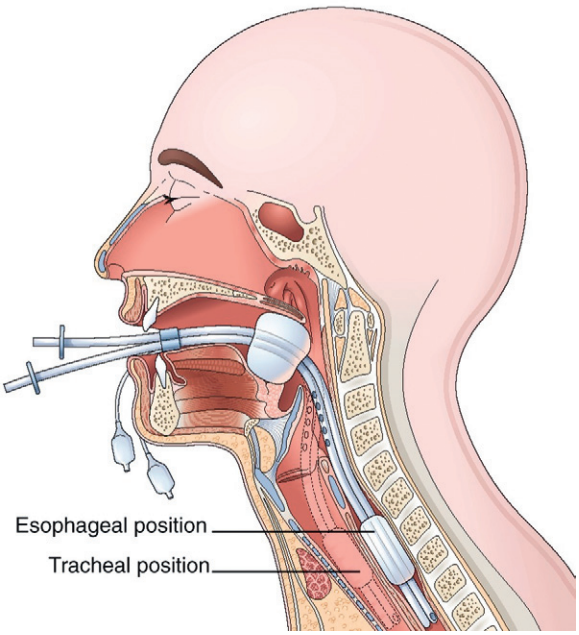


FIGURE 5-27 Esophageal-tracheal Combitube. The distal tube and cuff can be placed into either the esophagus or trachea. The operator must quickly determine where the tip of the device is located to ventilate the correct airway tube. (From Roberts J, Hedges J: *Clinical procedures in emergency medicine*, ed 5, St. Louis, 2010, Saunders.)

Complications of Endotracheal Intubation

The use of proper intubation techniques certainly minimizes the number of complications. Complications may arise from three distinct stages of the process—during direct laryngoscopy and endotracheal intubation, while the tube is in place, and postextubation.[11] Table 5-7 organizes these complications by stage.

SURGICAL AIRWAY

Establishing a surgical airway can be a last resort in response to failed intubation attempts for a critical patient with a compromised airway. When intubation fails, although the Combitube and laryngeal mask airway have been used with positive outcomes, surgically establishing the airway remains the gold standard. In some cases, a surgical airway is indicated prior to less invasive techniques. Typically, these situations consist of extreme facial trauma or a completely compromised airway, because serious brain damage can occur in a short period of time if the airway remains obstructed. When dealing with a compromised airway, delaying treatment can result in irreversible consequences. Therefore, avoiding delay in establishing a patent airway is as important, if not more so, than choosing which method or device to use in the setting of a patient with a compromised airway.[46]

The three accepted methods of obtaining surgical airways in adults are needle cricothyrotomy and translaryngeal jet ventilation, cricothyrotomy, and tracheostomy. In an emergency situation, cricothyrotomy has been shown to be faster and have lower morbidity and mortality rates than tracheostomy, and is therefore indicated over tracheostomy. Additionally, cricothyrotomy

TABLE 5-7 Complications of Intubation

During Laryngoscopy, Endotracheal Intubation	While ETT is in Place	After Extubation
Mucosal trauma	Laryngeal edema	Swallowing impairment
Dislocation of mandible	Cuff leakage	Infection
Dental trauma	Tracheal mucosal damage	Sinusitis
Esophageal Intubation	Infection	Laryngeal ulceration
Soft tissue perforation	Obstruction of ETT	Paralysis of vocal cords
Spinal cord injury caused by hyperextension	Aspiration	Pharyngitis, laryngitis, tracheitis
Induced vomiting	Laryngeal granulomas	Tracheal stenosis
Bronchial intubation	Tube displacement	Tracheoesophageal fistula
Vocal cord damage	Vocal cord damage	
Unable to intubate		
Epiglottis fracture		
Hemorrhage of airway		
Hypertension, tachycardia, bradycardia, dysrhythmias		

Adapted from references 11, 31, 34, and 37 and from Muskat PC: Emergency airway management in the traumatized patient. In Fonseca RJ, Turvey TA, Marciani RD, editors: *Oral and maxillofacial surgery*, ed 2, St. Louis, 2008, WB Saunders, pp 25–34.

should be avoided in infants and approached cautiously in children younger than 10 to 12 years because of the small anatomy of the cricoid cartilage and the associated high complication rates.[47-49]

Needle Cricothyrotomy and Translaryngeal Jet Ventilation

History. In 1967, Sanders first described the percutaneous placement of a tracheal needle with jet ventilation. Spoerel et al and Klain and Smith (1977) described the technique and indication for the procedure.[50-52]

Indications. Endotracheal intubation is contraindicated in certain situations, including severe hemorrhaging of the airway, edema, and some facial fractures or dislocations. In such cases, a needle cricothyrotomy (Figs. 5-28 and 5-29) is a quick and useful technique that provides oxygen on a short-term basis.[53] This should only be used temporarily until a definitive airway can be secured.[54,55] Extended use of this intervention will lead to severely elevated CO_2 levels, resulting in worsened respiratory acidosis.[56] Needle cricothyrotomy is quickly performed and may oxygenate the patient for up to 45 minutes while the physician establishes a more stable airway.[57] Needle cricothyrotomy and translaryngeal jet ventilation are indicated over cricothyrotomy in pediatric settings because of the smaller cricothyroid membrane and narrow airway in children.[47,49,58]

Technique. Needle cricothyrotomy consists of the insertion of a catheter through the cricothyroid membrane (see Figs. 5-28 and 5-29). The use of appropriate equipment increases the efficacy of translaryngeal jet ventilation. There are catheter devices made specifically for this procedure, such as the emergency transtracheal airway catheter (Cook, Bloomington, Ind). The major benefit of using these specific devices is that they tend to kink less frequently than standard angiocatheters, which may be used if the specific needle cricothyrotomy catheters are not available. Most adults require a 12- to 16-gauge standard angiocatheter (internal diameter [ID], 2.8 to1.5 mm) or a 6-Fr transtracheal catheter (ID, 2mm) for adequate oxygenation and enough ventilation to delay acidosis. Infants and small children typically require 16- to 18-gauge catheters. Needleless safety catheters should be avoided because of their inability to connect to a syringe.[57]

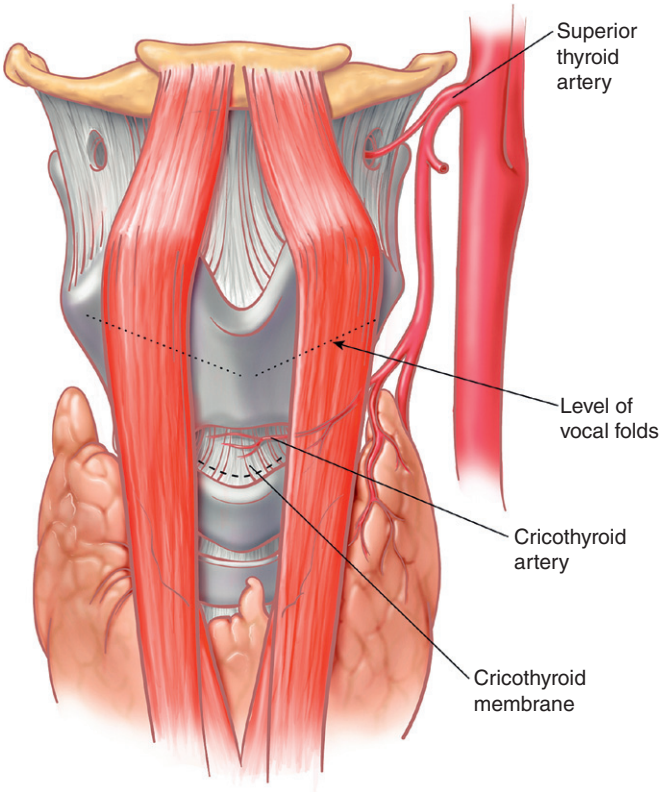


FIGURE 5-28 Cricothyrotomy anatomy. (From Thurnher D, Moukarbel RV, Novak CB, Gullane PJ: The glottis and subglottis: An otolaryngologist's perspective. *Thorac Surg Clin* 17:549, 2007.)

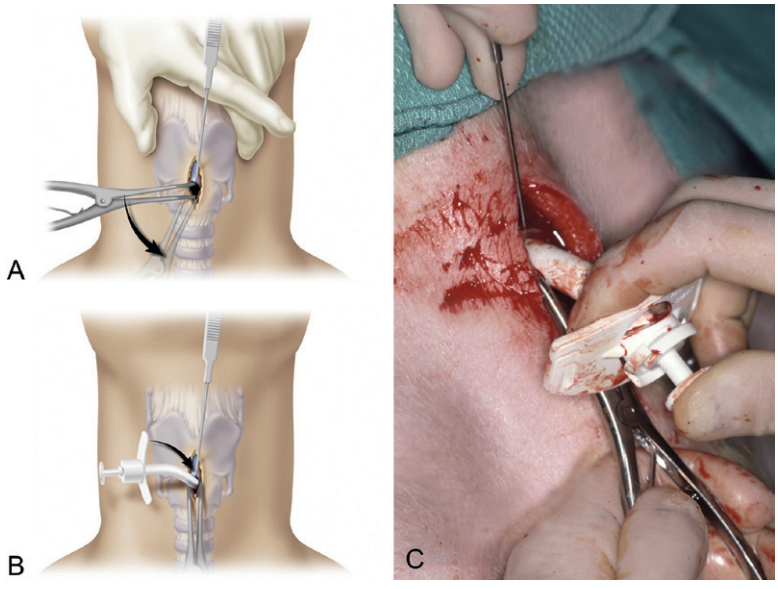


FIGURE 5-29 Needle cricothyrotomy. (From Custalow CB: *Color atlas of emergency department procedures*, Philadelphia, 2005, Elsevier Saunders.)

After identifying the relevant anatomic landmarks, the trachea is stabilized by the thumb and middle finger of the nondominant hand while the index finger locates the cricothyroid membrane. The skin is anesthetized with 1% lidocaine or a similar local anesthetic. A 10-mL syringe filled with 5-mL of saline is attached to the catheter and the needle is directed caudally at the inferior aspect of the cricothyroid membrane. Ideally, the needle enters the skin at a 30- to 45-degree angle to the horizontal and avoids injury to the surrounding vessels. In cases of severe laryngeal trauma, it may be necessary to use a more distal tracheal puncture.[11] Negative pressure is applied to the syringe on insertion of the needle and continues throughout advancement of the catheter. The entrance of air bubbles into the syringe confirms proper tracheal placement and the catheter is advanced until the hub reaches the level of the skin. Once the catheter has been advanced, the needle and syringe are withdrawn.[57,59,60]

Because of the importance of proper placement, the syringe is reattached and aspirated to verify that the catheter remains in place after the removal of the needle. An oxygen source is connected to the catheter and oxygen is delivered at 50 psi, with a flow rate of 15 liters/min. Although some devices are specifically made for this procedure, there are numerous variations of a moonlighter's device, used to connect the oxygen source to the catheter for translaryngeal jet ventilation. One simple variation includes oxygen tubing, a 3-mm endotracheal tube adapter, and a tracheal suction catheter vent.[11] Ventilations occur through the port on the suction device at a ratio of 1-second insufflation for every 4 to 5 seconds of exhalation. Although the ratio of 1:4 or 1:5 provides sufficient oxygenation in many cases, it does not provide adequate ventilation over time.[60]

Throughout the ventilation process, the catheter is manually secured in place until a definitive airway can be maintained.[57] It is important to note that because needle cricothyrotomy and translaryngeal jet ventilation only provide temporary airway control, a direct laryngoscopy may be performed at any time. The air bubbles within the trachea because of the translaryngeal jet ventilation may serve as a helpful guide for laryngoscopy.[11]

The traditional method of direct puncture (Quicktrach, VBM Medical, Sulz am Neckar, Germany), despite being a faster intervention, may involve more complications than the wire-guided tube placement technique (Melker set, Cook).[61] Additionally, a combined cannula-over-needle and wire-guided technique has been proposed.[62] A recent study using a swine model has revealed the potential benefit of using an esophageal detector device to ensure proper IV catheter placement in needle cricothyrotomies.[63] Newer, more flexible techniques have also been reported in the literature, including the use of an apparatus constructed using IV line tubing that has been executed successfully in emergency situations in adult patients.[64,65] Alternatively, the retrograde technique of intubation can be attempted using the translaryngeal catheter, which can be carefully withdrawn and redirected rostrally while still in the trachea.[11]

Complications. The most common complications of needle cricothyrotomy and translaryngeal jet ventilation are barotrauma, subcutaneous emphysema, and pneumothorax. By securing the catheter in place for the duration of the procedure, the risk of these complications can be minimized. Subcutaneous emphysema is often the result of a kinked catheter or multiple puncture sites. The use of a specific needle cricothyrotomy catheter (kink-resistant) helps decrease the chance of subcutaneous emphysema during or after the procedure. This complication may be unavoidable if there is leakage at the original puncture site or if there is significant catheter movement during ventilation causing subcutaneous air and swelling.[11]

Measures should be taken to avoid exceedingly high pressures by adjusting the airflow, depending on the apparatus used.[66] Complete upper airway obstruction is a contraindication to needle cricothyrotomy. It can lead to an inability to expel gas from the trachea and may cause additional air leakage at the puncture site, as well as increased pressure overall. Preventive measures should be taken to avoid excessive insufflation of oxygen, minimizing the risk of pneumothorax.[67] Some of the less common complications of needle cricothyrotomy and translaryngeal jet ventilation include infection, damage to surrounding tissues and structures, and bleeding.[57]

Percutaneous Cricothyrotomy. Percutaneous cricothyrotomy differs slightly from the traditional needle cricothyrotomy. Many commercially made kits come with all the necessary equipment to perform the procedure including needles, catheters, syringes, no. 15 scalpel blade, airway catheters of varying sizes, dilators, airway devices, flexible guidewire, and tracheal ties. It is important to note that some of these kits come with cuffed devices and others do not. Those kits containing uncuffed devices do not provide definitive airway management. The Melker emergency transcricothyrotomy catheter kit (Cook) and the Pertrach kit (Pulmodyne, Indianapolis) both contain cuffed devices.[60]

The procedure for the percutaneous cricothyrotomy begins just as the traditional needle cricothyrotomy does, with the identification of anatomic landmarks, stabilization of the trachea with the nondominant hand, and insertion of the needle into the cricothyroid membrane. If necessary, a no. 15 blade scalpel can be used to make the initial cut, followed by placement of the catheter over the needle with the attached syringe. Negative pressure is kept on the syringe until proper placement is confirmed by the presence of air bubbles. The needle and syringe are removed and the Seldinger technique, or placement of a guidewire through the catheter and into the trachea, is then used. The catheter is removed and extra care is taken to grasp the guidewire firmly so that it is not released into the airway. The dilator and airway device are placed over the guidewire and gently advanced into the trachea. Use of the dilator makes the opening larger for the catheter and device, leading to better ventilation.[60]

Despite their advantages on paper, some studies have suggested a higher complication rate and increased placement times for percutaneous cricothyrotomy with the kits when compared with the traditional surgical cricothyrotomy.[68-79] The results of these studies have indi-

cated that there is no substitute for experience of technique and proper use of equipment.

Cricothyrotomy

History. The history of cricothyrotomy (also known as cricothyroidotomy) is complicated and somewhat uncertain because of the numerous terms used to describe this procedure. The similarities of tracheostomy, tracheotomy, laryngotomy, high tracheostomy, and coniotomy make it difficult to differentiate between the histories of each procedure in cases in which the medical literature did not specify the important anatomic landmarks used. The first published descriptions of laryngotomy and cricothyrotomy occurred in the seventh century by Paul of Aegina and French surgeon Vicq d' Azyr, respectively.[80-82] The European diphtheria epidemic in 1833 gave rise to the popularity of the high tracheostomy, although Bretonneau and Trosseau originally described the procedure as an incision through the cricoid cartilage.[80,81]

In 1909, Chevalier Jackson first described the surgical techniques of the cricothyrotomy, even though he also referred to the procedure as a high tracheostomy. In the early 1920s, Jackson had 200 patients develop tracheal stenosis after the original procedure. When reviewed in further detail, it was found that 158 of the patients actually had a high tracheostomy done, whereas the remaining 32 patients had true cricothyrotomies. In the aftermath of these complications, Jackson decided that cricothyrotomies were unsafe and should no longer be taught as a form of surgical airway management.[11,83]

In 1976, Brantigan and Grow published their findings of a study that included cricothyrotomy data from 655 patients. Their results illustrated a low complication rate of 6.1%, in addition to no cases of subglottic stenosis.[52] In the years since they published their paper, numerous studies have supported their findings and provided validation that the cricothyrotomy is a safe and effective emergency surgical airway method.[54,56,68-70,84] The role of the cricothyrotomy in elective airway management, however, remains controversial.[71-74,85]

Indications and Contraindications. Cricothyrotomy (Table 5-8) is indicated in cases in which orotracheal and nasotracheal intubation are unsuccessful. If a patient is unable to get oxygen through simple methods and also cannot be intubated, an airway device such as the LMA may be used until the proper equipment and personnel are in place to perform a cricothyrotomy.[86] In an emergency medicine study that reviewed the most common conditions requiring cricothyrotomies, facial fractures were responsible for 32% of the cricothyrotomies performed and blood or emesis in the airway were responsible for another 32%.[87] Oral and maxillofacial trauma is one of the most important indications for cricothyrotomy.[11] Trismus, which may occur during cardiac arrest, is another indication for cricothyrotomy to maintain oxygenation and ventilation.[88] Contraindications include complete or partial transection of the airway, significant injury to the cricoid cartilage, and a severely fractured larynx.[83] In young children, cricothyrotomy is contraindicated because of the size of the cricothyroid membrane and narrowing of the pediatric airway. As a result, needle cricothyrotomy with translaryngeal jet ventilation is the preferred surgical airway method for children.[5,49]

TABLE 5-8 Cricothyrotomy

Indications	Contraindications
Maxillofacial trauma—obstructed airway	Age < 11 yr
Oropharyngeal obstruction—edema, infection, allergic reaction, heat injuries, foreign body, lesions, failed intubation	Crushed larynx—may result in tracheal separation because of direct trauma
Unsuccessful tracheal intubation caused by congenital malformations, hemorrhaging, vomiting, laryngospasms	Preexisting laryngeal or tracheal pathology

Adapted from reference 11.

Surgical Anatomy and Procedures. The cricothyroid space and its relevant anatomic landmarks can be identified visually and are palpable from the normal surface anatomy. Inferior to the mandible, the hyoid bone is located at the midline of the anterior neck and lies superior to the laryngeal prominence (Adam's apple). The laryngeal prominence is located at the superior edge of the thyroid cartilage, which is 2 to 3 cm above the cricoid cartilage.[89] It is most clearly seen in thin men, and is less likely to be visible or palpable in women, children, or obese patients.[90,91] The widest portion of the cricoid cartilage is found posteriorly.[83] The cricothyroid membrane is found in the space just below the thyroid cartilage but above the cricoid cartilage, and is also enclosed by the lateral cricothyroideus muscles on each side. This is the most important anatomic landmark because it is the location of the cricothyrotomy. Typically, the cricothyroid membrane measures between 2.7 and 3.2 cm wide and between 0.5 and 1.2 cm high (see Fig. 5-28).

Depending on the patient and case specific circumstances, the procedure can be done under local or general anesthesia. The head and neck should be slightly extended, unless a cervical spine injury is suspected or has not been ruled out. When working on a conscious patient, a local anesthetic (1% lidocaine) is administered to the skin and subcutaneous tissues after the anterior neck is prepped with an antiseptic solution. The positioning of the surgeon is a key element in obtaining a successful surgical airway. A right-handed surgeon performs the procedure by standing on the patient's right side and a left-handed surgeon stands on the patient's left side.[83] Using the same technique as in the needle cricothyrotomy, the thumb and middle finger of the nondominant hand are used to immobilize the larynx while the index finger locates the cricothyroid membrane. Stabilization of the larynx should be maintained at all times during the procedure.[11]

A 2-cm horizontal incision is made through the skin and subcutaneous tissue. (There is some debate among

experts about the benefits of a 3- to 5-cm vertical midline incision. These benefits include easy extension of the incision if the initial cut is too high or too low, as well as the ability to avoid the vascular structures located along the lateral aspect of the structure.)*

Regardless of the type of incision, care should be taken to avoid damaging the surrounding vessels by making the incision just superior to the cricoid cartilage. The incision is carried down through the cricothyroid membrane without going through the posterior wall of the airway, and is directed caudally to avoid the vocal cords. The nondominant index finger is used to hold the incision open and to minimize the bleeding. A Trousseau dilator or a large hemostat is inserted to spread the incision vertically. This increased opening in the cricothyroid membrane eases placement of the tracheal hook.[95-97] In some cases, the scalpel may remain in the incision until the tracheal hook is in place and can be used to retract the thyroid cartilage superiorly and anteriorly. The cephalad traction on the thyroid cartilage against the dilator stabilizes the airway and allows the surgeon to release the grasp of the larynx with their nondominant hand. A properly sized tracheostomy tube (no. 6 Shiley for average men, no. 4 for average women) is inserted into the opening and advanced into the trachea. An ETT can be placed if a tracheostomy tube is not immediately available.[98,99] The efficacy of certain ballpoint pen tubes to serve in place of a tracheostomy tube in emergency out of hospital situations has been documented.[100]

The dilator and tracheal hook are carefully removed to avoid causing any damage. The obturator is then removed before the inner cannula is inserted and the cuff or balloon of the tracheostomy tube is inflated. The tube is attached to a bag-valve device or a mechanical ventilator and is secured with umbilical tape that is tied around the neck before ventilation begins.[60,83] Because of the potential for the development of subcutaneous emphysema and pneumomediastinum, especially during mechanically supported respirations, the skin is not sutured as a method for securing the tube in place.[11] Additional bleeding from the insertion site is not uncommon, and gentle pressure is applied to stop it. After the cricothyrotomy is complete, a chest x-ray should be obtained to verify proper positioning of the tube and to exclude pneumothorax.[99,101]

An analysis of different cricothyrotomy devices (cuffed: Quicktrach II [VBM Medical], Portex cricothyroidotomy kit [Smiths Medical, Dublin, Ohio], Melker cuffed cannula [Cook]; uncuffed: Airfree (VBM Medical), 4.0-mm ID Quicktrach, 6.0-mm ID Melker, 13-gauge Ravussin cannula [VBM Medical]) has demonstrated the superiority of cuffed devices during controlled, manual, and spontaneous ventilation.[102]

The traditional cricothyrotomy procedures have been described, but a simplified four-step technique can also be used. The only equipment needed for the rapid four-step technique (RFST) is a no. 20 scalpel, hook, and tracheostomy tube. The procedural steps are as follows:

1. Palpate and identify the cricothyroid membrane.
2. Using the no. 20 scalpel, make a 1- to 2-cm horizontal incision through the skin, subcutaneous tissue, and cricothyroid membrane.
3. Place the tracheal hook (before removing the scalpel) and direct it inferiorly to provide caudal traction.
4. Insert the tracheostomy tube.[83,103]

Based on the fewer number of steps used, the RFST is typically quicker than the traditional method, as confirmed by a cadaver study.[104] Although previous cadaver studies explored the potential increase in complications for using the rapid technique, other clinical studies have suggested a higher complication rate associated with the traditional technique based on retrospective data.[105,106] For patients who have well-defined anatomic landmarks with little fat or inflammation, the RFST of cricothyrotomy is likely to be successful. However, in patients with less obvious anatomic landmarks or significant swelling, the traditional technique should be followed.[11]

Similar to the rapid four-step technique is the recently developed bougie-assisted cricothyrotomy technique.[107] The major difference is the insertion of a bougie and endotracheal tube instead of a Shiley tracheostomy tube. In animal studies, the bougie-assisted cricothyrotomy technique was demonstrated as a faster technique with similar complication rates compared with the standard cricothyrotomy technique.[108]

When the patient is confined to a small area, the limited space may make an intervention difficult. A study analyzing the execution of emergency cricothyrotomy in confined spaces has revealed that the medical residents preferred a particular kit, the Quicktrach kit (VBM Medical). The Quicktrach kit was placed correctly in the airway and had the fastest placement time because of the quality and simplicity of the kit when compared with the multipart Melker kit (Cook).[109]

Complications. Complications of cricothyrotomy vary greatly depending on clinical circumstances—most notably, whether the procedure was elective or emergent. Typically, emergency cricothyrotomy has a higher complication rate than elective cricothyrotomy. This is expected because patients who undergo emergency cricothyrotomy are often critically ill, have difficult airways, and need to be oxygenated immediately. The higher complication rate is acceptable in emergent cases because of the importance of securing an airway. Studies have shown complication rates of up to 8% for cricothyrotomy, although for emergency procedures, the rate is between 28% and 32%.[92,110,111] In these emergent cases, poor outcomes are usually because of the original trauma and not directly attributed to the cricothyrotomy. Another study, conducted in 2001, suggested a low rate of minor long-term complications.[112] Complications from a cricothyrotomy can be divided into two groups, perioperative and postoperative.

Perioperative Complications. The most common perioperative complications include hemorrhage, improper placement of the tube, and prolonged execution time. Bleeding during the procedure is not usually severe, and can be controlled by ligation, cautery, or packing with gauze. Clamping should be avoided, if possible, to protect

*References 11,60,83,86,91-94.

the surrounding structures. By correctly positioning the patient, using adequate light and suction, selecting the proper instruments, and placing the tube under direct visualization, the potential for complications caused by improper tube placement can be minimized. This complication occurred in 1.3% of elective cricothyrotomies and in 36% of emergent cricothyrotomies.[11,98,99,113] Although an uncomplicated cricothyrotomy can be performed in as few as 30 seconds, 3 minutes is the acceptable standard of time for the procedure. Some of the more severe complications are associated with longer procedure times and the airway should therefore be secured as quickly and efficiently as possible.[110]

Less common perioperative complications include injury or laceration to the thyroid or cricoid cartilage, injury to the esophagus or laryngeal nerve, pneumomediastinum, perforation of the posterior trachea, and subcutaneous emphysema.[11,83]

Postoperative Complications. Postoperative complications of cricothyrotomy include hemorrhage, infection, aspiration, tube occlusion, paralysis of the vocal cords, persistent stoma, dysphonia and hoarseness, and subglottic stenosis. Infection is a rare complication of cricothyrotomy. In the report by Brantigan and Grow in the 1970s, only 3 of 655 patients studied experienced postoperative infections.[92] Voice changes, including dysphonia, hoarseness, and changes in pitch or volume, are some of the more common postoperative complications of cricothyrotomy. Some studies have shown that the rate of this complication is approximately 40%.[110,112,114,115] In most cases, the voice changes correct themselves over time, although a follow-up surgery to remove granulomas may be performed, if necessary, to correct any serious complications.[11] The most severe complication of cricothyrotomy is subglottic stenosis, which is experienced by from 1.2% to 2.6% of patients according to various studies. When compared with the subglottic stenosis complication rates of other surgical airway methods, such as tracheostomy (18% to 20%) or even long-term endotracheal intubation, these rates are extremely low.[91,111,116,117] Postoperative stenosis caused by cricothyrotomy is typically found at the cuff site or just inferior to the vocal cords. This condition can be treated with steroids, chronic cannulation, dilation, surgical removal of granulation tissue, or tracheal resection.

Proper postoperative care, including the monitoring of cuff pressure, is crucial for the prevention of secondary complications. These complications may include hemorrhage, tracheomalacia, and cellulitis.[11]

Few deaths have been identified as a result of complications from cricothyrotomy. Of those published cases, the causes of death ranged from loss of airway during surgery, to the inadvertent tube removal and inability to reinsert due to tube occlusion, and asystole during tube change.[98,99,110,118]

Tracheostomy

History. The tracheostomy is one of the oldest documented surgical procedures, dating as far back as 2000 BC, when it was described in the Rig Veda, a sacred Hindu text. Information on the procedure was also found in Egyptian documents written more than 3500 years ago. Galen, Antyllus, and Aretaeus wrote about the elective tracheostomy during the second century AD.[90,119-122]

Prior to the nineteenth century, the tracheostomy (laryngotomy or bronchotomy) was not often performed because of the belief that it was a dangerous and futile procedure. The techniques of tracheostomy were first described by Hieronymus Fabricius in 1617 and Habicot in 1620. Although Heister introduced the term *tracheotomy* in the early 1700s, Negus did not introduce the term *tracheostomy* until 1938; tracheostomy became an acceptable surgical technique in 1833 in response to the findings of Bretonneau and Trousseau.[90,119-122] They found that tracheostomy was successful in treating the airway obstruction of children suffering from diphtheria or croup. From there, Chevalier Jackson increased the depth of understanding procedural techniques and complications through papers published in 1909 and 1921.[123,124] As a more detailed understanding of tracheostomy evolved, indications for its use increased.

Because of the safety, speed, and relative ease of RSI by properly trained individuals, in addition to the choice of needle or open cricothyrotomy as the preferred surgical intervention when an emergency surgical airway is indicated, tracheostomy has been relegated to limited use in emergency situations.[47,125,126] However, a recent study by Bobek et al has revealed the use, safety, and efficacy of tracheotomies in elective, urgent, and emergent situations as an acceptable alternative to cricothyrotomy.[127]

Indications and Contraindications. In general, the best results from tracheostomy occur when it is carefully performed in the operating room. Tracheostomy in the controlled environment of the operating room has been shown to have a complication rate of less than 3%.[128] It is also important to note that early tracheostomy placement—within the first week of mechanical ventilation—in the patient with an anticipated mechanical ventilation time of longer than 10 days results in significantly less nosocomial pneumonia, decreases time on mechanical ventilation, and shortens ICU stay.[129-131] However, studies focused on the timing of tracheostomy placement in relation to ICU survival rates have produced varying results.[132-134] Hospitalized critically ill patients who received a tracheostomy had a lower mortality rate (13.7%) than those who did not (26.4%).[135] Needle or open cricothyrotomy remains the surgical procedure of choice in most emergency settings because it tends to be less difficult and time-consuming and has lower complication rates. However, in some rare situations, an emergency tracheostomy is the recommended or only course of action.[111,114,118,136]

Blunt neck trauma, including laryngotracheal trauma, is the one condition for which a tracheostomy is the undisputed method of establishing the airway. Although this particular type of trauma is rare, usually occurring in high-speed accidents when the anterior neck strikes a cord or rope, people who experience it are almost certainly found in some form of respiratory distress.[137] Distortion of the larynx makes anatomic identification and proper tube placement difficult in a cricothyrotomy.[126,138] Blunt neck trauma can also lead to fractures of the thyroid or cricoid cartilage, vessel damage, or fracture of

the hyoid bone. These injuries may prevent access to the cricothyroid membrane, making cricothyrotomy difficult.

If endotracheal intubation or cricothyrotomy is performed in the presence of a tracheal transection, there is a chance that the trachea may retract into the mediastinum. To avoid this, a tracheostomy is indicated in the presence of tracheal transection.[139] Other indications for tracheostomy include upper airway obstruction, need for prolonged mechanical ventilation, complex facial fractures, large or expanding neck hematomas, edema, deep space neck infections, and lacerations to the floor of the mouth.[137,140] Attempts to establish an airway by cricothyrotomy through a hematoma could result in exsanguination. As a result, the airway must be accessed lower in the trachea, indicating tracheostomy.[141] Because of improved intubation techniques and use of the fiberoptic laryngoscope and bronchoscope, it has become less common to use upper airway obstruction as an indication for tracheostomy.[11]

It should be reinforced that tracheostomy is contraindicated if an airway can be secured by any other method. If endotracheal intubation fails or cannot be performed, cricothyrotomy is the preferred surgical airway. The difficulty level, amount of time needed, and potential complications are greater for tracheostomy than for other definitive airways, emphasizing that this is the method of last resort.[11]

Surgical Anatomy and Procedures. To gain access to the trachea, a thorough knowledge of neck anatomy is needed to avoid unnecessary complications. Figures 5-30 and 5-31 (also see Figs. 5-3 and 5-28) present a variety of neck anatomy illustrations. The hyoid bone attaches to the base of the skull, tongue, and mandible by muscles. It is the most stable portion of the airway and can be a useful anatomic landmark throughout the procedure.[137] The thyroid cartilage is palpable superficially and superiorly. The cricothyroid membrane and cricoid cartilage are identified inferior to the thyroid cartilage. Proceeding inferiorly, the tracheal rings are palpable initially, but palpation becomes increasingly difficult as the trachea descends into the chest. The suprasternal or jugular notch is the visible dip located where the neck and chest come together. Once the trachea reaches this level, it is 1 to 1.5 cm deeper than the cricoid cartilage, emphasizing the technical expertise required to perform a tracheostomy safely.[142]

The superficial layers are made up of skin and subcutaneous tissue, followed by the investing fascia, which above the sternum divides into two layers attached to the anterior and posterior sternum, forming the suprasternal space of Burns. Loose connective tissue and fat fill this space, which also contains the anterior jugular veins and their anastomosis—the jugular venous arch. Sometimes, the anterior jugular veins come together to form the median jugular vein. The infrahyoid muscles and their fascia, found laterally to the midline, form the posterior wall of the space of Burns. The infrahyoid muscles do not cross the midline, resulting in the formation of a linea alba at the midline.[11,143,144]

The pretracheal fascia, which overlies the trachea with its palpable rings, and the thyroid isthmus are located

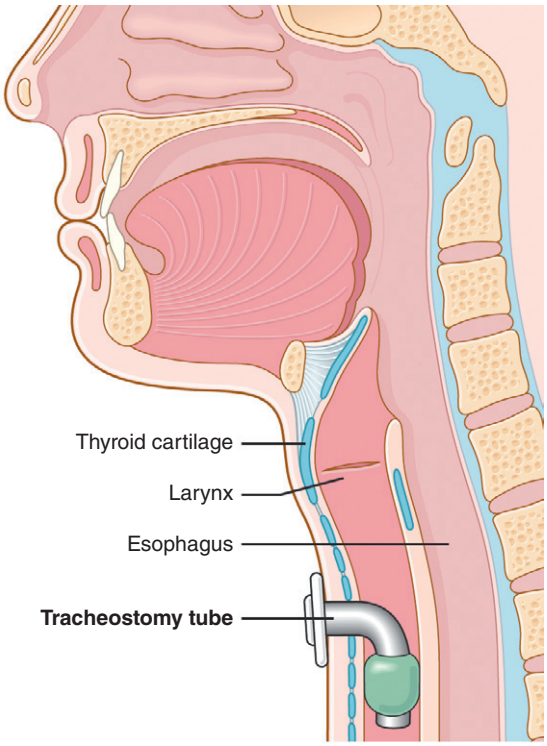


FIGURE 5-30 Tracheostomy. (From Black JM, Hawks JH, Keene AM: Medical-surgical nursing: Clinical management for positive outcomes, ed 6, Philadelphia, 2001, WB Saunders.)

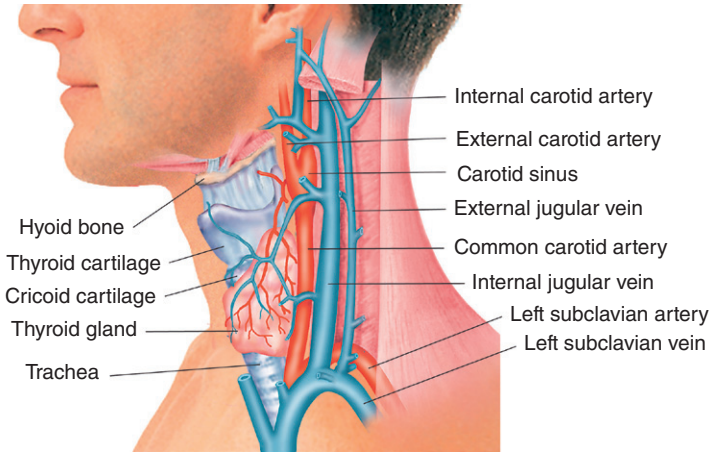


FIGURE 5-31 Neck vasculature. (From Seidel HM, Ball JW, Dains JE, et al: *Mosby's guide to physical examination*, ed 6, St. Louis, 2006, Mosby.)

beneath the infrahyoid fascia and muscles. The anterior portion of the pretracheal fascia is thin; however, it is thicker laterally as it blends with the carotid sheath. Posteriorly, the fibroelastic wall of the trachea lies against the esophagus.[11,143,144] The thyroid gland, which has a rich blood supply, is difficult to avoid during tracheostomy because of its location directly anterior to the second, third, and fourth tracheal rings.[137] The complex neck vasculature is illustrated in Figure 5-31. Although the major blood vessels do not typically cross the midline of the neck, many of the major vessels anastomose anterior

to the thyroid gland. This plexus of veins includes the infrahyoid veins, which empty into the brachiocephalic vein, the infrathyroid artery from the thyrocervical trunk, and the thyroid ima artery. The brachiocephalic and left common carotid arteries are in the same layer inferiorly. Although these arteries are not usually exposed in adults, they can be exposed in children because their hearts and great vessels sit at a higher level.[145] Ultrasound guidance may prove to be beneficial for the localization of anterior neck anatomy, especially the vasculature, in surgical tracheostomies.[146]

In preparation for the procedure, the skin incision should be marked while the patient's head is in a normal position. Marking the incision spot before extension of the neck should ensure that the incision is located in the proper tracheal opening. This also prevents skin irritation, skin tension against the tube, and dislodging of the tracheostomy tube. Unless contraindicated because of potential cervical spine injury, a rolled towel is placed under the patient's neck and shoulders. Full extension of the neck fixes the airway in position and enlarges the surgical field.[137,142]

Depending on the particular circumstances regarding each individual patient, general or local anesthesia may be necessary to establish the airway by tracheostomy. If a patient is hypoxic, uncooperative, or difficult to restrain, a sedative medication may be indicated. The sedative of choice is typically one that leaves the gag reflex intact, preserves spontaneous respirations, and does not have extensive hemodynamic consequences. If a patient is conscious and cooperative, the procedure can be done under local anesthesia. The equipment should be checked to ensure that all needed parts are available, properly sized, and working effectively. The average man requires a no. 7 or 8 Shiley tracheal tube; the average woman requires a no. 5 or 6 Shiley tracheal tube.[137] Appropriately sized tracheostomy tubes are selected to occupy from two thirds to three quarters of the tracheal lumen diameter.[11]

The neck is prepped with an antiseptic solution and a local anesthetic, such as 1% lidocaine, is injected into the incision site. Additionally, 2 mL of the local anesthetic is inserted into the cricothyroid membrane and injected into the trachea. This blunts the cough reflex. The airway should be stabilized with the nondominant hand, similar to the procedure for the other surgical airways. Both vertical and horizontal incisions can provide adequate access to the airway. In an emergency tracheostomy, the vertical incision maintains midline dissection and reduces the potential for anatomic damage when the direction of the incision is changed.[126] The 3- or 4-cm vertical incision is made through the skin, subcutaneous tissue, and platysma muscle. It begins just below the cricoid cartilage and extends to the suprasternal or supraclavicular notch. An incision that follows these landmarks will result in proper placement over the second through fourth tracheal rings.[137,142,147]

In an elective tracheostomy, the horizontal incision is preferred for better cosmetic results. A 4- to 5-cm horizontal incision is made approximately 2 cm below the cricoid cartilage. Like the vertical incision, it is carried through subcutaneous tissue and platysma muscle until the superficial layer of the deep cervical fascia is identified.[143,145,144]

As the space of Burns is entered bluntly, the inferior thyroid veins are identified, clamped, and tied before cutting them to minimize bleeding. The infrahyoid fascia, which overlies the sternohyoid muscles, is bluntly dissected through its linea alba. By vertically retracting the midline tissue away from the trachea, injury to major vessels, nerves, and glandular tissue can be avoided. Retraction and dissection now expose the pretracheal fascia and thyroid isthmus. The second through fourth tracheal rings are located underneath these structures. Because of the richly vascular nature of this region, blunt dissection is done to prevent significant hemorrhaging. The thyroid gland is retracted out of the field, exposing the tracheal rings. If the thyroid isthmus cannot be retracted out of the field, it must be transected, which can be done by cutting the suspensory ligament. On each side of the midline, clamps are used and the isthmus is cut and oversewn. At this point, the tracheal rings should now be visible. The trachea has an external diameter of 1.5 to 2.0 cm in the adult and 3 to 4 mm in the newborn. It is composed of 16 to 20 hyaline cartilage rings and a fibroelastic tissue connects it posteriorly. The dissection should be clear, making it easy to identify the cricoid cartilage and the first four tracheal rings (see Fig. 5-30).[11,137]

Although there are various techniques for surgical entrance into the trachea, two principles must be followed no matter which technique is used. First, the cricoid cartilage and first tracheal ring must not be cut or injured. Second, the incision into the trachea must stop at or above the fourth tracheal ring.[11]

A tracheostomy hook is placed just below the first tracheal ring. This acts to immobilize and elevate the trachea. The tracheal incision can be made by the following techniques—U, inverted U, T flap, and cruciform. In emergent situations, a vertical midline incision between the second and fourth tracheal rings is recommended. In nonemergent situations, the inverted U offers some distinct advantages; it prevents the cannula from being inserted anterior to the trachea, the patient can breathe more easily through the stoma if the cannula is lost in the first few days, and changing of the cannula is facilitated.[148,149] A traction suture of 2-0 silk is placed through the flap tip and inferior margin of the skin and is tied. This secures the position of the flap tip forward and down. Traction on the suture helps position the trachea and stoma anteriorly, leading to better visualization and access. The inverted U incision is made through the second and third tracheal rings. Once the incision is made, a Trousseau dilator or Kelly hemostat is inserted and spread vertically. The tracheal lumen and an ETT, if present, should be visible. The tracheostomy tube should be inserted under direct vision once the Trousseau dilator is in place. While the tracheostomy tube is inserted, the ETT, if present, is carefully and slowly withdrawn to expose the tracheal lumen. The cuff and tip of the tube are advanced into position, just inferior to the vocal cords.[11,137]

Proper tube location and fit should be assessed and the patient's chest should be monitored for movement.

The cuff is then inflated and the skin can be left open or loosely sutured. If the skin is sutured too tightly, subcutaneous emphysema may result from not allowing air to escape during forced expiration or continuous positive-pressure ventilation.[11]

Once it has been determined that the tube is in the right location, a tracheostomy gauze dressing should be placed under the tracheostomy tube phalanges and around the cannula. The tube should be secured with cloth tape tied around the patient's neck to prevent inadvertent extubation. Typically, a chest x-ray is obtained to verify tube placement and to check for pneumothorax, a complication of special concern in the pediatric population.[101,141,150] In one 2012 study, however, Tobler et al argued that there may be no benefit to obtaining a chest x-ray unless clinically indicated.[151]

Complications. Although it was once a commonly used surgical procedure, the historical results of complications caused by tracheostomy are not favorable. In fact, the literature has identified only 28 successful procedures performed before 1825.[152] Throughout the years, advances in surgical technique, instrumentation, postoperative care, and surgical training have moved tracheostomy into the realm of an acceptable, predictable surgical procedure, especially in a controlled environment.[11] Although recent advances have improved the postoperative outcomes of this procedure, complications following tracheostomy still occur. Reports of the morbidity rate of tracheostomies range from 6% to 58%. Emergent procedures tend to have higher morbidity rates.[137]

Overall, the incidence of complications from tracheostomy ranges from 2.7% to 48%.[73,111] Rogers has reported rates of 9.6% and Chew and Cantrell have reported rates of 15.8% in larger sample sizes.[153,154] One factor that greatly affects the morbidity incidence and severity of tracheostomy is the duration of tube placement. An increase in duration from 7 to 30 days causes an increase from 20% to 50%.[155] Obesity is another factor that increases the chances of tracheostomy complications.[156] One study, which reviewed the frequency of complications related to tracheostomy placement and management in morbidly obese (body mass index [BMI] > 40 kg/m^2) patients to be 25% compared with nonobese patients, at 14%.[157] In general, the complications for tracheostomy are similar to those associated with cricothyrotomy. Hemorrhage, pneumothorax, subcutaneous emphysema, pneumomediastinum, hypoxia through false passage, obstruction, and extubation are perioperative complications common to both procedures. Complications specific to tracheostomy include recurrent laryngeal nerve injury, tracheoesophageal fistula, and death. Postoperative complications caused by tracheostomy are also similar to those associated with cricothyrotomy and include hypoxia, infection, hemorrhage, tracheal stenosis, tracheal erosion, tracheomalacia, and unsatisfactory cosmesis.[11]

Of the perioperative complications listed, acute hemorrhage is the most common complication because of the rich vasculature of the neck, specifically the tracheostomy site.[158,141,159,160] The vessels in this area include the anterior jugular veins and their midline anastomoses, inferior thyroid plexus of veins, and superior thyroid artery. Other potential sources of hemorrhage are the thyroid gland isthmus if it is improperly clamped, cut, or oversewn and the carotid sheath, found lateral to the trachea, which contains the common carotid artery, vagus nerve, and jugular veins. This location makes it susceptible to injury by the scalpel if it is not properly controlled, especially in infants.[11] Approximately 5% of tracheostomy cases result in some form of hemorrhage. It is important to recognize and treat hemorrhaging as soon as it is discovered to avoid the more serious complications associated with major bleeds or exsanguinations. If delayed hemorrhage presents itself during the postoperative period, it may be a sign of a major bleed.[161,162] Visible pulsation of the placed tracheostomy tube also signifies a potential great vessel bleed.

Regarding postoperative hemorrhages, most occur in the first 2 to 4 weeks after the procedure. By the end of the third postoperative week, almost 72% of hemorrhages that will develop have already occurred. In some rare cases, hemorrhage can occur months after the procedure in patients with long-existing tracheostomies and a thoracic surgery consult is indicated.[163]

Cases that involve a tracheostomy placed below the third or fourth tracheal ring could lead to excessive cuff or tube tip pressure. This can cause erosion of the trachea or major vessels in the neck and mediastinum. Breakdown of tissue barriers because of infection or inflammation can contribute to an environment in which a catastrophic bleed can occur. The innominate artery is located superior to the manubrium; formation of a tracheoinnominate artery fistula and subsequent hemorrhage is a rare but serious complication.[164] Jones et al[163] have conducted a review of 9415 tracheostomies and a tracheoinnominate artery fistula occurred with an incidence of 0.6%. A surgically repaired fistula has a survival rate of approximately 25%. However, if the fistula is not operated on, there is almost a 100% mortality rate.[154] Procedural interventions for tracheoinnominate artery fistula include endovascular stenting and endovascular embolization of the innominate artery.[165,166]

If a major bleed occurs, overinflation of the tracheostomy tube can tamponade the vessel until surgical control can be achieved. This maneuver has been found to be effective approximately 85% of the time. In cases in which the originally placed tube was noncuffed, it must be replaced with an ETT or cuffed tracheostomy tube. If cuff pressure fails to slow the bleeding, digital compression of the artery against the sternum should be attempted.[163,167,168]

Infection is the second most common complication of tracheostomy. Potential postoperative infection includes surgical site infection, tracheitis, mediastinitis, and pneumonia.[137] Stomal infection can be caused by contaminants from the skin, nasopharynx, and oral cavity. Mediastinitis is a more severe and devastating complication; it occurs because the infection of the stoma travels into the chest. The pathogens most commonly isolated from tracheostomy infections are *Pseudomonas aeruginosa*, *Staphylococcus aureus*, hemolytic streptococci, and *Candida*.[154,155,159]

Tracheal stenosis is one of the most common delayed complications of tracheostomy. Results about the rate of

occurrence for this complication vary greatly. Although one study reported a 98% chance of stenosis, another study, which reviewed autopsy reports, showed a much lower incidence rate of 65%.[137,152,169] Stenosis is not considered clinically significant until the normal adult trachea is compromised between 25% and 50%. Systematic stenosis occurred in 2% to 20% of tracheostomy cases.[160,170] With the help of clinical investigations and experimental studies, the causes of stenosis have been documented. One potential cause of the complication is cuff pressure that exceeds the mucosal capillary perfusion pressure (≈30 mm Hg). This causes an interruption in the mucosal blood supply, leading to tissue death, ulceration, resultant granulation tissue formation, and scarring.[11] Currently, the highest acceptable cuff pressure is 20 to 30 mm Hg.[171-173] Another possible contributing factor to stenosis is the location of the incision. Cartilage loses its ability to regenerate after it becomes infected or is damaged because of ischemia, resulting in granuloma formation, tracheomalacia, or stenosis.

The occurrence of pneumothorax after tracheostomy is higher than after cricothyrotomy; it may occur in as many as 5% of adult tracheostomies.[152] Infants, children, and those with chronic obstructive pulmonary disease also have an increased chance of pneumothorax complications after tracheostomy. The risk is as high as 17% for children and infants because their pleural domes are higher in the neck.[174] Furthermore, the loose connective tissue in children allows for air tracing, which can lead to pneumomediastinum and rupture of the pleura.[175]

Aspiration is a serious postoperative tracheostomy complication that is especially important in patients older than 70 years. Younger patients are more likely to have a successful swallow post-tracheostomy.[176]

Recurrent, bilateral laryngeal nerve injury may cause severe dyspnea because of collapse of the vocal cords and may be noticed only when attempts at decannulation are unsuccessful. Permanent tracheal cannulation may be necessary in the presence of paralysis. If the damage is only affecting a single nerve, it may result in hoarseness and dyspnea on exertion, or the patient may be completely asymptomatic. Through proper maintenance of the midline dissection and careful use of blunt dissection techniques, with adequate light and proper instrumentation, the risk of laryngeal nerve damage can be limited.[144]

Because of the difficulty of emergency tracheostomy, technical errors are not uncommon and complications can occur. Mortality rates from tracheostomy are reported in the range of 2.5% to 10%, with higher rates in children and infants.[169,177] It is imperative that when reviewing the statistics on complications and mortality rates after tracheostomy, it is understood that the patients undergoing these procedures are critically ill or have experienced incredible trauma. It is highly likely that some of the complications are directly related to the underlying condition, and not actually because of the placement of the surgical airway.[11] The most common fatal complications (listed in descending frequency) are hemorrhage, displaced tube, infection, obstruction, aspiration, and tracheal erosion, with resultant stenosis or fistula.[153,178,179]

Postoperative Care. The importance of proper aftercare for tracheostomy patients should not be understated. In the days and weeks following the procedure, numerous complications can potentially arise. Some of those complications may be avoided with proper care. Tracheal cannula occlusion caused by hemorrhage or mucous secretions can lead to serious complications.

Regular monitoring and proper suctioning can reduce the chances of this postoperative complication. "Trach care," which describes the specific techniques for proper tube care, consists of tube aspirations and frequent suctioning in the days and weeks after surgery. If blood, mucus, or other secretions build up in the airway and cause occlusion of the tube, the patient will lose the ability to breathe. If this is not immediately recognized, the consequences can be fatal.

To aspirate the tracheostomy tube effectively, it is recommended that that patient's lungs be filled with 100% oxygen for 2 or 3 minutes before suctioning occurs; then 5 mL of sterile saline is injected into the tracheal tube, immediately followed by 2- to 3-second suctioning intervals. The steps should be repeated as long as notable secretions are removed from the airway. Trach care is completed once every hour for the first 48 hours. The following 2 days, it should be completed once every 2 hours. After the first 4 days, it should be completed every 4 hours.[11]

In addition to cleaning the original tracheostomy tube, it is recommended that the tube be changed as needed. There is no exact time frame for when tubes should be changed, but guidelines vary, from 5 days to 1 week to 7 to 14 days after the original tube placement.[11,180,181] Tube changes are often indicated by clinical circumstances such as patient discomfort, improper tube positioning, patient-ventilator asynchrony, cuff leak, tube fracture, need for a different tube type, or bronchoscopy.

Other important aspects of postoperative tracheostomy care include keeping the surgical wound clean and dry by frequently changing the sterile gauze dressings and maintaining proper cuff pressure (≈20 mm Hg). When a tracheostomy tube is in place, the patient is unable to humidify inspired air because the nasopharynx is bypassed in the respiration process. As a result, 40% humidified air should be administered during postoperative care to prevent drying of mucosal tissues and to aid in secretion removal. If humidified air is not provided in sufficient amounts, secretions become more viscous from the effects of dehydration.[11]

Coughing, deep breathing, and pulmonary toilet are all encouraged in conscious patients. The presence of a weak cough, or an inability to cough, predisposes the patient to aspiration, leading to serious complications. As noted, tracheostomy leads to a decrease in the amount of humidified air present, which leads to decreased ciliary function. This makes the cough mechanism even more important.[125,142]

Percutaneous Tracheostomy

The need for a more efficient and safe tracheostomy technique led to the development of the percutaneous tracheostomy. Although not indicated for emergency airway management, it should be mentioned as a method

of elective airway management because of its increasing popularity in ICU settings.[182] The technique was originally developed based on Seldinger's description of arterial catheterization in 1953.[183] Shortly after, a needle-guided trocar was described by Shelden et al for access into the trachea.[184] Many variations of the technique have since been developed. Most of the techniques incorporate slight changes to the procedure originally described by Ciaglia et al in 1985. This percutaneous dilational tracheostomy, based on the Seldinger technique, uses sequentially sized dilators prior to placement of the tracheostomy tube.[185] There are numerous commercial kits available; most are based on the Ciaglia technique.

There are many similarities between the percutaneous dilational tracheostomy (PDT) and the emergency percutaneous cricothyrotomy. The first procedural step of the PDT is to make a horizontal incision (≈2 cm long) at the level of the second tracheal ring. This is followed by blunt vertical dissection. At this point, the five major steps of percutaneous cricothyrotomy are similarly followed:

1. A needle with a saline-filled syringe attached is inserted into the trachea. The presence of air bubbles in the syringe confirms proper tracheal placement.
2. The syringe is removed and the guidewire is introduced through a cannula into the trachea. The needle is also removed.
3. Dilators that increase sequentially in size are introduced into the surgical site over the guidewire. Each dilator should be lubricated to ease entry into the stoma.
4. The dilators are removed and the tracheostomy tube is lubricated and placed.
5. Proper positioning and airway control are confirmed.

Although many of these steps are consistent from technique to technique, some variations include the use of a flexible bronchoscope or type of skin incisions.[11]

Some advantages to the PDT over surgical tracheostomy are as follows: the procedure is less time-consuming, it may have a lower complication rate (depending on the specific method and expertise of the person performing the procedure), and there is increased flexibility as to when it can be performed.[182,186] It has also been reported that the PDT technique reduces patient cost.[187] The results of a 2006 study confirmed a reduction in complications regarding wound infections for PDT patients.[180] Other studies have been inconclusive about some of the potential PDT advantages. Rosenbower et al,[188] in 1998, had comparable complication rates, but found the percutaneous method to be more cost-effective at their institution.[11] In 1999, Dulguerov et al found that although surgical tracheostomy had a higher postoperative complication rate, PDT led to higher rate of perioperative complications.[189] A multi-institutional analysis of 1,175 tracheostomies has revealed a significantly higher rate (6.6%) of postoperative bleeding in percutaneous tracheostomies than open tracheostomies (1.9%).[160]

Percutaneous dilational tracheostomy should not be performed in patients younger than 15 years. Neck distortion caused by hematoma, tumor, thyromegaly, or scarring is another relative contraindication to the procedure, along with obesity, short neck anatomy, infection of the soft tissues, tracheomalacia, and cervical spine instability. Surgical tracheostomy may be indicated over PDT in some of these cases.[180,190]

A 2011 study by Jackson et al has demonstrated the potential benefit of using bronchoscopic guidance for percutaneous tracheostomy in elected cases of patients with difficult anatomy.[191] The use of ultrasound guidance in percutaneous tracheostomy to ensure proper tube placement and avoidance of vascular structures has been shown to increase the accuracy and safety of this intervention.[192,193] Similar to open tracheostomy, the formation of tracheoinnominate artery fistulas and subsequent life-threatening hemorrhage following percutaneous tracheostomy have been documented.[164] The results from one study reported that critically ill patients undergoing PDT have a high short-term mortality, with 11% of patients dying within the first 2 weeks postprocedure.[194]

In general, advocates of the percutaneous tracheostomy believe that it is quicker because it can be performed at the bedside, safer as it omits the transportation to and from the operating room, and more cost-effective because there is no operating room, equipment, anesthesiologist, or surgeon fees charged to the patient. Based on this information, it is not surprising that percutaneous tracheostomy is more widely used than surgical tracheostomy.[179] Critics of PDT believe that financial cost should not be a deciding factor and that the benefits of proper lighting, equipment, and qualified personnel outweigh the monetary difference between the procedures.[11]

Until surgeons have obtained sufficient experience performing this procedure, Powell et al have recommended guidelines to maximize the chance for success.[195] As with all the surgical airways, it is imperative that the surgeon be familiar with the relevant anatomy so that major complications can be avoided. Because the rate of complications for PDT fluctuates greatly depending on the method chosen and expertise of the surgeon, it is important that this procedure only be done under the supervision of experienced personnel.

PEDIATRIC CONSIDERATIONS

Treatment of the pediatric airway in the presence of maxillofacial trauma is similar to that of the adult airway. There are however, a few important differences that need to be addressed. Pediatric trauma patients are especially susceptible to cervical spine injury and these precautions should be taken seriously. Clinicians should maintain proper immobilization at all times, until the child has been cleared of a cervical injury.[196]

ANATOMY

The anatomy of children varies slightly from that of adults. In general, their airways are smaller and tend to become obstructed more easily. Foreign bodies, secretions, or even edema can cause an obstructed airway.[197] Furthermore, the tongue and tonsils of a child are large in relation to the rest of the oral cavity. Because of their relatively large size, they have a tendency to get in the

way during airway interventions.[198] The larynx is located higher and more anteriorly in children than in adults. This is important to note because hyperextension of the neck may further obstruct the airway.

Signs of inadequate ventilation for pediatric trauma patients are similar to the signs in adults. Wheezing, stridor, nasal flaring, grunting, retractions, and tachypnea are all signs of difficult ventilation. As with adults, noisy breathing is a sign of airway obstruction.[196]

TIPS FOR SPECIFIC INTUBATION PROCEDURES ON CHILDREN

1. Direct laryngoscopy: The straight blade is used with younger children; the curved blade is used for older children.[199]
2. ETT selection: Proper tube size can be determined by the following equation[199]:

$$\text{Tube size} = 4 + (\text{child's age}/4)$$

   a. Newborn, no. 3.0 or 3.5 ETT
   b. 6-year-old child, no. 5.5 ETT
3. Needle cricothyrotomy with transtracheal jet ventilation: This is the preferred surgical airway method in children because it is straightforward and provides a patent airway for close to an hour.[199]
4. Cricothyrotomy: This procedure has a high complication rate and should not be done in children younger than 10 years.[199]
5. Tracheostomy: This should also be avoided in children because it is a time-consuming procedure. However, in emergent cases, if needle cricothyrotomy with transtracheal jet ventilation has failed in a very small child, a tracheostomy may be performed.[200]

When performing RSI in children, it is important that the clinician be fully aware of the side effects of each medication on pediatric patients.[199]

SUMMARY

When dealing with the airway management of a maxillofacial trauma patient, it is essential that the clinician possess the skills and techniques not only to provide an emergency airway, but to adapt in critical situations. A variety of methods, maneuvers, and techniques were described throughout this chapter to review different methodologies to obtain a definitive airway. Managing a traumatic airway is an incredibly dynamic situation, making it necessary to reevaluate and assess the situation regularly. In general, clinicians should attempt the least invasive procedure that is expected to be successful based on the individual patient's current diagnosis and symptoms. Securing a traumatic airway could save the life of a patient. Because of the importance of this skill, clinicians should regularly practice as many methods as possible.

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CHAPTER

6 Management of Nonpenetrating Chest Trauma

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OUTLINE

History of Chest Trauma
Motor Vehicle Accidents and Thoracic Injuries
General Management of Blunt Chest Trauma
Pulmonary Dysfunction
Extrathoracic Injuries
Process for Treating Chest Wall Injuries
Arterial Blood Gas
Radiographic Examination
Advanced Trauma Life Support
Pneumothorax
Closed or Noncommunicating Pneumothorax
Tension Pneumothorax
Simple Pneumothorax
Open Pneumothorax
Hemothorax
Diagnosis
Management
Hemopneumothorax
Pulmonary Contusion
Treatment
Rib Fractures
Diagnosis
Treatment
First Rib Fractures
Flail Chest
Pathophysiology
Diagnosis
Treatment
Fractured Sternum
Cervical Tracheal Disruption
Tracheobronchial Injuries
Esophageal Injuries
Penetrating Neck Trauma
Diaphragmatic Injuries
Rupture of the Aorta
Injuries of the Descending Thoracic Aorta
Diagnosis
Treatment
Myocardial Contusion
Treatment

HISTORY OF CHEST TRAUMA

Trauma is the leading cause of morbidity and mortality in Americans younger than 45 years and the third highest overall cause of death in the United States.[1] Thoracic trauma can cause significant mortality, because 25% of traumatic deaths are secondary to thoracic injuries.[2] The written history of chest trauma can be traced back to 1600 BC in Egypt; 48 cases of trauma were reported, and four of these involving the chest were described. One detailed flail chest, which at that time was deemed hopeless. In 400 BC, Hippocrates described wrapping of chest with linen to stabilize fractures for 20-40 days. This practice continued until the twentieth century. In 1773, William Bromfield performed the first thoracentesis for traumatic pneumothorax and described tension pneumothorax.

Over the last 100 years, most of what we have learned about chest trauma was taken from wartime. During WWI, 37% of deaths were related to chest injuries.[2] In WWII, 34% of deaths were related to chest injuries. However, the overall mortality from WWI to WWII decreased from 24% to 8%. Advances in antimicrobials, anatomy, and physiology have helped improved morbidity and mortality associated with chest trauma.

MOTOR VEHICLE ACCIDENTS AND THORACIC INJURIES

The number of motor vehicles on the roads worldwide has increased steadily since World War II and, with the increase in high-speed motor vehicle travel, there has been a correlated increase in the number of motor vehicle accidents (MVAs). MVAs are leading cause of death for people age 5 to 34 years in the United States.[3] Each year, more than 6 million people are injured in MVAs. Of those, 45% to 50% have thoracic injuries and approximately 150,000 of these victims die. A total of 25% of these 150,000 deaths are caused entirely by thoracic injuries and in another 50% of victims, injuries of the thorax are a major contributing factor to mortality[4-6] The male-to-female ratio is 6.5:1. Also, nonfatal chest injuries, such as rib fractures and pneumothorax or hemothorax, are frequently seen in victims of MVAs. MVAs are the most common cause of blunt chest trauma, accounting for 70% to 80% of blunt thoracic injuries. The costs of crash-related deaths and injuries among drivers and passengers in 2003 totaled $70 billion dollars.

The mortality rate from isolated blunt chest trauma 25 years ago approached 50%. Fortunately, improvements in prehospital care and the development of rapid

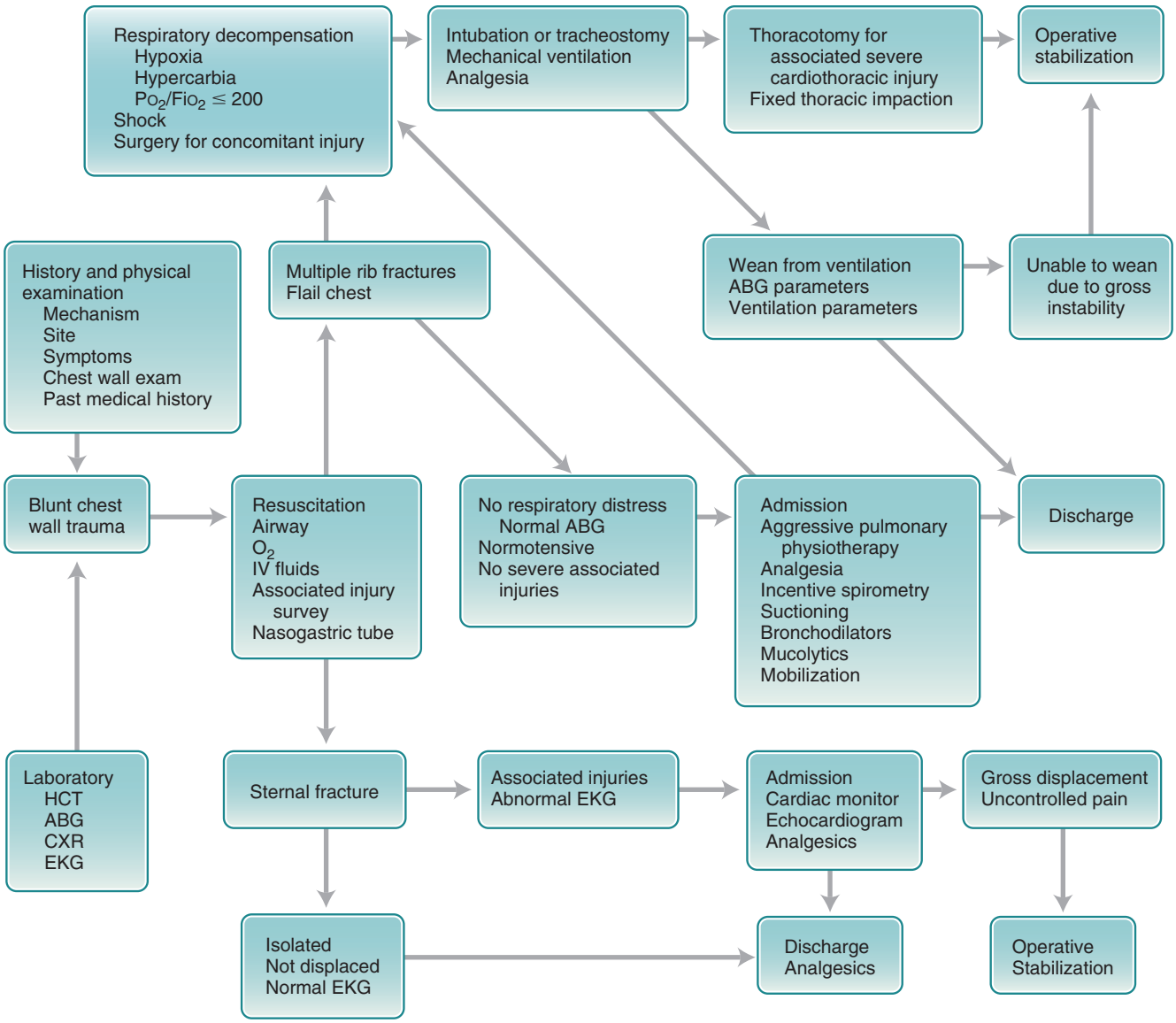


FIGURE 6-1 Chest wall trauma algorithm. (Adapted from Mattox KL, Feliciano DV, Moore EE, editors: *Trauma*, ed 4, New York, 2000, McGraw-Hill.)

transport systems have lowered the mortality rate to less than 10%. Less than 10% of the patients suffering blunt chest trauma require a thoracotomy. Prevention of closed chest injuries associated with MVAs has been the focus of improved highway construction and design, as well as vehicular safety features and innovations such as air bag systems, safety belts with shoulder harnesses, antilock brakes, use of energy-absorbing materials, and improved design. Enforcing the use of safety belts and impaired driver legislation have also decreased the severity of MVAs and associated injuries.[7] It is hoped that once the patient reaches the hospital, the prompt recognition of chest injuries, coupled with an improved understanding of their pathophysiology and management, will further reduce the number of deaths caused by nonpenetrating thoracic trauma.[8]

GENERAL MANAGEMENT OF BLUNT CHEST TRAUMA

The most common injury associated with blunt chest trauma is rib fracture, which occurs in 56% of patients reported in a study of chest trauma victims, followed by pneumothorax in 40% of patients evaluated, pulmonary contusion (35%), flail chest (13%), and injury to the heart and major vessels in 5% of cases.[9] Initial management of patients with blunt chest trauma should focus on performance of the ABCs of trauma care,[10] including establishment of a patent airway, initiation or support of adequate ventilation, and treatment of shock (Fig. 6-1).

Treatment of severe chest wall injury must focus on the identification and care of specific injuries after establishing adequate ventilation and oxygenation. Chest

BOX 6-1 Injuries Associated with Nonpenetrating Chest Trauma

Cardiovascular
- Myocardial concussion
- Myocardial contusion
- Cardiac rupture
- Air embolus
- Traumatic aortic injury
- Aortic fistula
- Great vessel injury
- Traumatic asphyxia

Pulmonary
- Tracheal tear
- Bronchial tear
- Pneumothorax
- Pneumomediastinum emphysema
- Subcutaneous emphysema
- Hemothorax
- Lung laceration
- Pulmonary contusion
- Acute respiratory distress syndrome

Chest Wall
- Rib fractures
- Flail chest
- Sternum fractures

Diaphragm
- Diaphragmatic rupture
- Esophageal injuries

trauma can cause mortality from airway obstruction, tracheobronchial injuries, pulmonary secretions, hemorrhage, loss of oxygenation, ventilation, exsanguination, or cardiac failure. Blunt forces inflicted on the chest wall will cause injury by three mechanisms—rapid deceleration, direct impact, and compression.[11] Rapid deceleration is usually associated with MVAs and falls from heights. It is important to recognize that serious intrathoracic injury can occur without obvious external chest injuries or rib fractures. The degree of injury to the chest wall is usually not indicative of the severity of the injury to the internal tissue and/or organs. The possibility of injury to the heart, lungs, vessels, and other tissue should be taken into account (Box 6-1). Deceleration produces traction on the great vessels and trachea, with disruption and tears near points of anatomic fixation, such as the ligamentum arteriosum with the aorta and the carina with the trachea.[9]

When the chest wall is exposed to a significant enough force that can fracture ribs, the semirigid chest wall of older adults absorbs energy that would otherwise be transmitted to the lung parenchyma and great vessels. However, the flexible chest wall of the younger patient transmits essentially all the kinetic energy to the thoracic contents. Thus, the absence of rib fractures does not exclude the possibility of life-threatening injuries because injuries to the lung parenchyma, cardiac contusion, or pneumothorax are possible without external signs. Almost 50% of all patients with traumatic aortic injury[12-14] and up to one third of patients with myocardial contusion[15,16] have no external signs of trauma. If the injury is not recognized early, death frequently results. Those caring for patients who have sustained blunt trauma to the chest must not be deceived by initial clinical stability and must always be alert for the likelihood of later deterioration. Some patients with life-threatening injuries may appear clinically well on arrival to the emergency room.

PULMONARY DYSFUNCTION

Most of the pulmonary dysfunction associated with nonpenetrating chest trauma is secondary to the underlying pulmonary contusion or lung injury rather than disruption of chest wall expansion and respiratory mechanics caused by fractured ribs and/or sternum, pain, or muscle splinting.[17-19] Compression of the chest wall by heavy objects (e.g., automobiles, rocks, with a trapped victim) prevents respiration and may result in significant increase in pressure within the veins of the upper thorax and traumatic asphyxia. Frequent observation of any patient with blunt chest injury is as important as the initial evaluation to detect progressive changes in function. A pneumothorax may develop several hours after an injury and a small hemothorax may continue to increase in size over a period of several hours. A flail segment not apparent on an initial examination may become obvious from 1 to 10 days after an injury.[11,20]

EXTRATHORACIC INJURIES

Patients with thoracic injuries frequently sustain multiple extrathoracic injuries. The physician's attention must not be diverted from thoracic injuries by obvious extrathoracic trauma, such as abdominal, maxillofacial, or central nervous system (CNS) injuries. Associated injuries occur in 66% to 80% of patients with chest trauma and usually involve the head, abdomen, pelvis, and extremities.[21]

PROCESS FOR TREATING CHEST WALL INJURIES

After any immediate life-threatening emergencies have been treated, the physician should undertake a careful history, if possible, and should methodically examine all systems of the injured patient. Knowledge of the patient's past medical history, allergies, and medications may influence subsequent therapy. Clues to likely injuries may be gained from knowledge of the circumstances under which the injuries occurred. The chest wall should be inspected for wounds, lacerations, contusions, abrasions, or other evidence of trauma and splinting, paradoxical movement of the chest wall with breathing caused by rib or sternum fractures. The neck should be evaluated for distention of the veins or tracheal deviation. The physician should listen to the lung fields and heart for the presence, absence, or alteration of breath or cardiac sounds, and heart murmurs or bruits that may be indicative of a developing cardiac, lung, or vessel injury. A nasogastric tube should be placed, unless contraindicated because of injury, to prevent acute gastric dilation, which can exacerbate respiratory compromise in injured patients.[11]

ARTERIAL BLOOD GAS

Arterial blood gas (ABG) determinations are a means of indirect measurement and serial assessment of the

TABLE 6-1 Indications for Respiratory Support

Function	Value
MECHANICS	
Respiratory rate (breaths/min)	>35
Vital capacity (mL/kg body weight)	<15
FEV_1 (mL/kg body weight)	<10
Inspiratory force (cm H_2O)	<–25
OXYGENATION	
PaO_2 (mm Hg)	<60 (supplemented with O_2)
$AaDO_2$ (mm Hg)	>450 (on 100% O_2)
VENTILATION	
$PaCO_2$ (mm Hg)	>55
V_D/V_T	>0.60

$AaDO_2$, Alveolar-arterial difference in partial pressure of oxygen; *FEV_1*, forced expiratory volume in 1 second; *V_D*, volume of dead air space; *V_T*, tidal volume.

TABLE 6-2 Arterial Blood Gas

Condition	pH	$PaCO_2$	HCO_3
Respiratory acidosis	Decreased	Increased	Normal
Respiratory alkalosis	Increased	Decreased	Normal
Metabolic acidosis	Decreased	Normal	Decreased
Metabolic alkalosis	Increased	Normal	Increased

Parameter	Definition	Normal Range
pH	Measurement of acidity or alkalinity based on the hydrogen (H^+) ions present	7.35-7.45
PaO_2	Partial pressure of oxygen that is dissolved in arterial blood	80-100 mm Hg
SaO_2	Arterial oxygen saturation	95%-100%
$PaCO_2$	Partial pressure of carbon dioxide in arterial blood	35-45mm Hg
HCO_3	Amount of bicarbonate in the bloodstream	22-26 mEq/liter
Base excess	Indicates the amount of excess or insufficient level of bicarbonate in the system	–2 to +2 mEq/liter

functional impairment associated with pulmonary injury. All patients with suspected flail chest or pulmonary contusion should be observed, including taking serial blood gas measurements. Often, the first sign of impending respiratory failure will be marked hypoxia (Table 6-1). The ratio of arterial oxygen tension to the fraction of inspired oxygen (PaO_2/FiO_2 ratio) yields an estimate of the extent of an intrapulmonary shunt and may be used as a parameter indicating the need for mechanical ventilation in patients with pulmonary contusion and flail chest.[22] Serial ABG determinations, compared with baseline data obtained while the patient breathes room air, may be useful in predicting trends in respiratory function (Table 6-2). Using a fractional concentration of inspired oxygen (FiO_2) of 0.21 permits consistent serial comparisons and gives a more accurate estimation of the severity of the respiratory impairment. Patients should be followed initially with ABG determinations every 4 hours for 48 hours to detect subtle changes that might prompt modification of treatment. Noninvasive measurement of oxygen saturation by pulse oximetry can be used as an indicator of the degree of functional pulmonary injury and response to treatment.

RADIOGRAPHIC EXAMINATION

Radiographic examination using standard supine chest radiographs, with inspiratory and expiratory views and a lateral projection, should be obtained early in the examination of patients with nonpenetrating chest wall trauma to evaluate the sternum, spine, ribs, heart, lungs, and other structures of the neck, thorax, and abdomen for the presence of a pneumothorax, hemothorax, widened mediastinum, subcutaneous air, pulmonary contusion, ruptured diaphragm, and other abnormal findings (see Box 6-1). It is important to obtain quality chest x-rays quickly in the initial management of patients with blunt chest trauma, even when the patient has serious associated injuries, such as head trauma or compound fractures, because there may be developing life-threatening injuries that have not been previously detected or clinically apparent. It is also important to obtain baseline radiographs during the initial examination of the patient to evaluate the patient's clinical progress or changes in clinical signs and symptoms further. Chest films should be delayed only if obvious therapeutic measures are immediately required, such as for significant bleeding, shock, and airway and/or ventilatory problems.

Chest computed tomography (CT) has proven to be of great use in distinguishing chest wall injury from parenchymal or mediastinal injury. Also, by CT scanning, more injuries are detected and consequently treated. Therefore, every patient who has sustained severe thoracic trauma and is hemodynamically stable should get a CT scan of the chest on admission to a hospital.[23-26]

ADVANCED TRAUMA LIFE SUPPORT

Appropriate patient management of nonpenetrating chest wall trauma involves the correction of alterations of gas exchange and ventilation and support of respiratory function. Blunt thoracic injuries may quickly develop into life-threatening circumstances. Consequently, each patient should be assessed according to Advanced Trauma Life Support (ATLS) principles, including establishment of a patent airway with cervical spine support until cleared, initiation or support of adequate breathing and ventilation, and treatment of shock. Lives can be saved if each patient is assessed according to ATLS principles. Initial assessment with the ABCDE algorithm—***a***irway, ***b***reathing, ***c***irculation, ***d***isability, and ***e***xposure—is followed by a thorough secondary survey. At all times, lifesaving interventions should be made when identified, followed by reevaluation and stabilization, and finally transfer to a definitive care hospital.

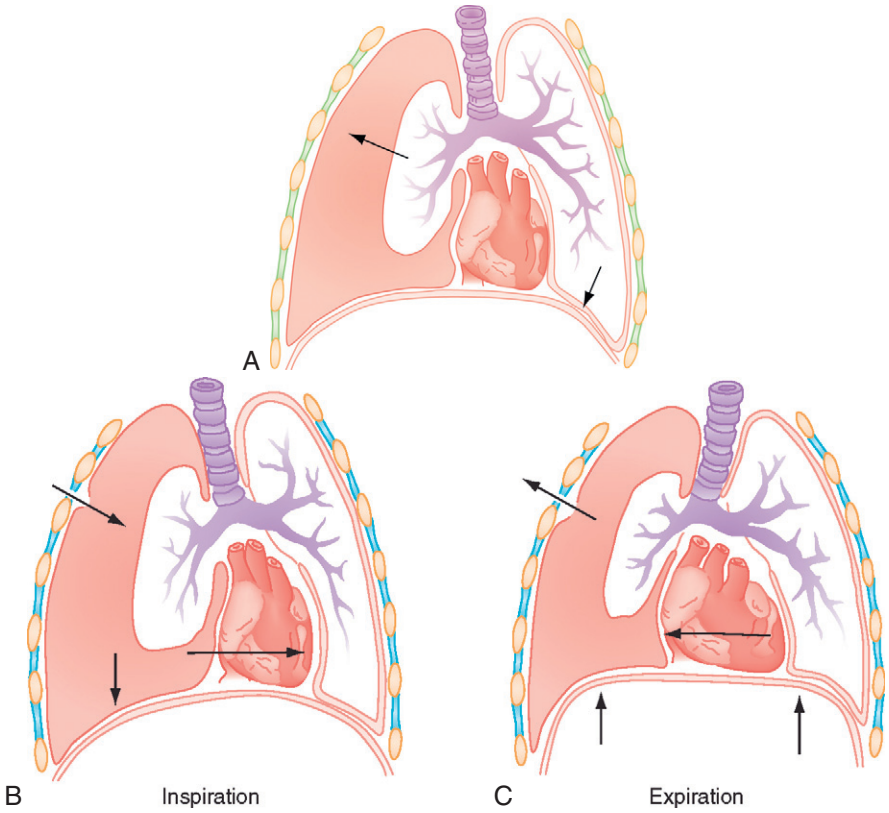


FIGURE 6-2 **A,** A pneumothorax develops from damage to the chest wall or laceration of the lung pleura, with a resulting loss of negative intrapleural pressure. A pneumothorax may be graded as small (<15%), moderate (15% to 60%), or large (>60%). Usually, only patients with moderate or large pneumothoraces are symptomatic. **B, C,** With an open wound in the chest wall, air can be often heard moving in and out of the wound during respiration, known as a sucking chest wound. An open pneumothorax may be converted to a simple pneumothorax with the use of an occlusive dressing over the chest wall wound. Care must be taken not to create a trapdoor effect that does not allow the air from the pleural cavity to escape and create a tension pneumothorax. (From Marx JA, Hockberger RS, Walls RM: *Rosen's emergency medicine*, ed 7, St. Louis, 2010, Mosby.)

PNEUMOTHORAX

The thoracic cavity is normally filled by the expanded lung. A potential space exists between visceral and parietal pleura, blood, or air in the space is pathologic. If there is air present in the pleural space, one of two events has occurred: (1) communication between alveolar spaces and pleura; or (2) direct or indirect communication between the atmosphere and pleural space.[27] Pneumothorax is a common complication of penetrating chest trauma caused by a defect in the chest wall that allows air to be moved in and out of the pleural cavity with each respiration or a sudden increase in intrathoracic pressure (Fig. 6-2). An equilibrium develops between the intrathoracic pressure and atmospheric pressure, which affects the ability of the lung to remain inflated. The involved lung collapses on inspiration and slightly expands on expiration. This causes the air to be drawn out of the wound in the chest wall with an open pneumothorax or from a wound in the lung from a fractured rib. Inspired air escapes into the pleural cavity in a closed pneumothorax injury (Fig. 6-3). Pneumothorax is usually unilateral; however, it may be bilateral in 10% to 15% of cases.[8] Most of these injuries are associated with MVAs, but blast injuries, falls, direct blows to the chest, or automobile-pedestrian collisions may also be associated with a pneumothorax. Pneumothoraces are categorized as open or closed and are further defined as simple or complicated. A simple pneumothorax is air in the pleural space that does not communicate with the atmosphere or distort the mediastinum. The two possible mechanisms include laceration of the pleura or lung parenchyma caused by a fractured rib and compression of the chest against a closed glottis that ruptures the alveoli.[28]

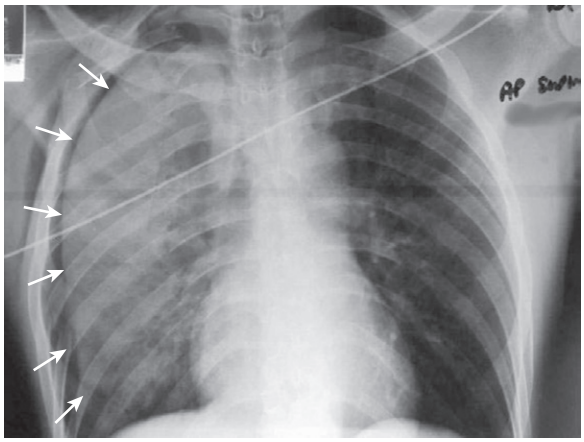


FIGURE 6-3 A wound in the lung from a fractured rib allows inspired air to escape into the pleural cavity in a patient with a closed pneumothorax injury. The involved lung collapses on inspiration (as outlined by the *arrows*) and slightly expands on expiration, causing the air to be drawn out of the wound in the lung.

CLOSED OR NONCOMMUNICATING PNEUMOTHORAX

A closed pneumothorax, the most common type, usually results from a rib fracture, causing a parenchymal laceration (Fig. 6-4). It may also occur without an accompanying rib fracture. In general, this type of pneumothorax

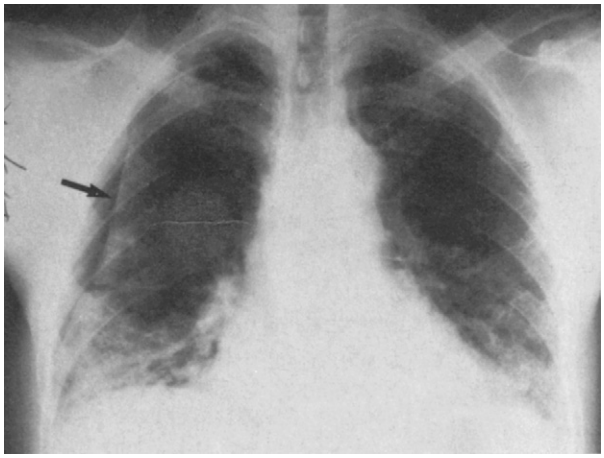


FIGURE 6-4 A closed pneumothorax, the most common type of injury to the lung, is demonstrated in the right lung (*arrow*) on this expiratory chest radiograph. (From Rosen P, Doris PE, Barkin RM, et al, editors: *Diagnostic radiology in emergency medicine*, St. Louis, 1992, Mosby.)

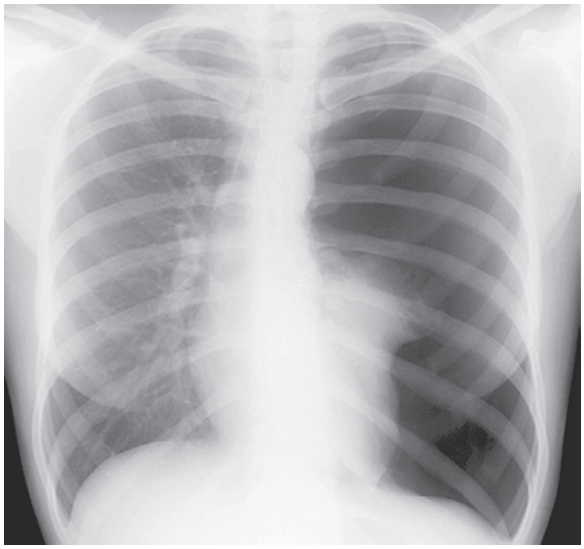


FIGURE 6-5 A tension pneumothorax resulting from trauma. This upright chest radiograph demonstrates the shift of the mediastinum, with compression of the opposite lung and resultant hypoxia.

tends to be self-limited because the resultant pulmonary collapse usually seals the air leak (simple pneumothorax). However, if a large parenchymal disruption exists, air from the damaged lung continuously is allowed to enter the pleural cavity. Because the air cannot escape from the pleural cavity, it tends to accumulate under pressure, producing a tension pneumothorax. Air from the lung to the pleural space equalizes the pressure within the chest cavity and the lung collapses. A ventilation-perfusion deficit occurs because the blood circulated to the affected lung is not oxygenated. With a pneumothorax, percussion of the chest shows hyperresonance and breath sounds are usually distant or absent.

TENSION PNEUMOTHORAX

A tension pneumothorax that develops as a result of trauma exists when the tissues surrounding the opening into the pleural cavity act as a valve, allowing air to enter the pleural cavity but not to escape (Fig. 6-5). This results in a shift of the mediastinum, with compression of the opposite lung and resultant hypoxia. The positive intrapleural pressure produces compression of the vena cava, thereby interfering with diastolic filling of the heart and resulting in low cardiac output.

Patients with tension pneumothoraces are acutely ill and in respiratory distress with chest pain. They are restless, agitated, dyspneic, and at times cyanotic. On physical examination, the patients are hypotensive, tachycardiac, and tachypneic. Breath sounds are absent and percussion is hyperresonant over the involved hemithorax. The cervical trachea is displaced to the side opposite the pneumothorax, and distended neck veins may be present.

Tension pneumothorax is life-threatening and demands immediate treatment following a clinical diagnosis based on the above-mentioned signs and symptoms. If untreated, tension pneumothorax quickly results in death. If a developing tension pneumothorax is suspected, the positive intrapleural pressure should be released as soon as possible, and not delayed for radiographic confirmation. The pressure can be released with insertion of a large angiocatheter (14 to 16 gauge) in the midclavicular line in the second intercostal space. This quickly converts the tension pneumothorax into a pneumothorax, which can then be treated with a thoracostomy in the anterior axillary line at the level of the nipple and placement of a chest tube. If the clinical diagnosis of a tension pneumothorax is missed clinically, the chest radiograph will demonstrate a large collection of air in the involved hemithorax, with a shift of the mediastinum toward the unaffected side. The lung on the involved side need not be completely collapsed.

Positive pressure ventilation is the major cause of tension pneumothorax; however, a tension pneumothorax may be associated with invasive medical procedures such as central line placement. The development of a tension pneumothorax occasionally occurs with spontaneous breathing, but is more likely in patients who undergo bag-valve-mask or mechanical ventilation. Positive-pressure ventilation may also convert a simple pneumothorax to a tension pneumothorax. As the intrapleural pressure increases, the mediastinum shifts and the venous return to the superior vena cava is impaired. Tension pneumothorax causes hypotension, increases the central venous pressure, decreases cardiac output, and ultimately produces cardiovascular collapse.[29,30]

SIMPLE PNEUMOTHORAX

Simple pneumothoraces are usually classified according to the volume of the hemithorax occupied by air or the degree of pulmonary collapse. A pneumothorax that occupies 15% or less of the pleural cavity is classified as a small pneumothorax. One that occupies between 15% and 60% of the pleural cavity is classified as moderate, and one that occupies more than 60% is classified as large. Usually, only patients with moderate or large pneumothoraces are symptomatic. However, in patients with significant underlying parenchymal disease, a small pneumothorax may compromise pulmonary function

sufficiently to cause symptoms and require immediate treatment.

The most frequent symptoms in patients with pneumothoraces are chest pain and shortness of breath. On physical examination, the breath sounds are distant or entirely absent over the involved hemithorax. In an attempt to decompress the thoracic cavity, air frequently escapes into the subcutaneous tissues, resulting in subcutaneous emphysema. Approximately 25% of patients with pneumothorax have associated injuries of the chest wall.

Diagnosis

The diagnosis of a simple pneumothorax is suggested by the history and clinical findings and is confirmed by the chest radiograph. The chest radiograph should be obtained at the earliest possible moment, but it should not take precedence over emergency treatment if the clinical condition suggests that a pneumothorax is present.

Treatment

Most pneumothoraces associated with trauma require only simple treatment, with thoracostomy drainage with chest tube. Only rarely, in asymptomatic patients who are otherwise healthy and have no associated injury, should a pneumothorax be treated with observation. If observation is chosen as a method of treatment, a repeat chest film should be obtained 6 to 8 hours after admission and daily thereafter until complete reexpansion of the lung occurs. If the patient is breathing spontaneously, supplemental oxygen should be delivered by a face mask. The exchange of air does not guarantee adequate ventilation. The chest wall of a patient with a pneumothorax, flail chest, or hemothorax may move but not ventilate effectively. Also, shallow breaths with minimal tidal volumes do not ventilate the lungs effectively. The patient should at least be monitored by pulse oximetry. If the patient requires mechanical ventilation, operative intervention for other injuries, or transportation to another hospital, a chest tube must be placed in the patient to prevent development of a tension pneumothorax.

The indications for closed tube thoracostomy (Fig. 6-6) include the following: (1) pneumothorax of moderate to large size; (2) presence of respiratory symptoms regardless of the size of the pneumothorax; (3) increase in size of the pneumothorax that initially was treated conservatively; (4) recurrence of pneumothorax

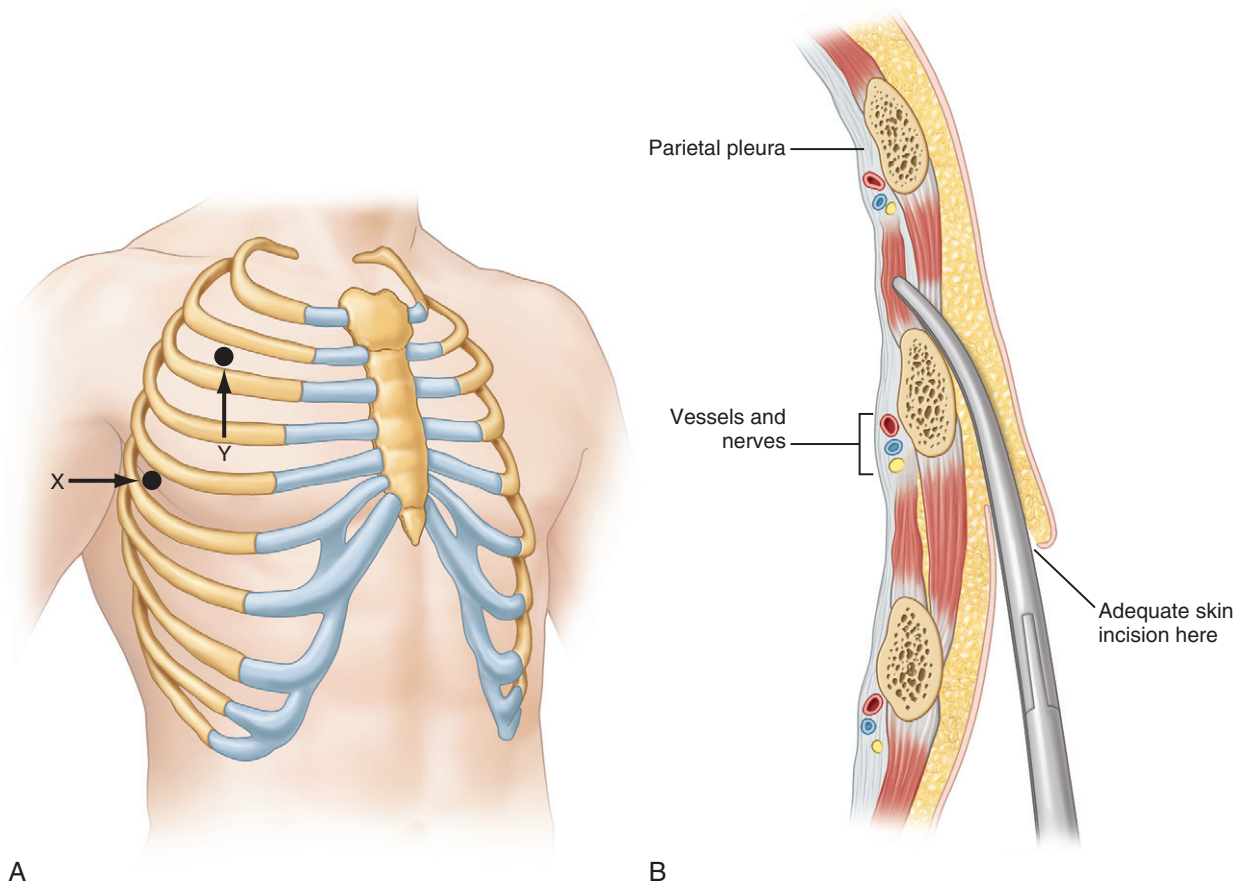


FIGURE 6-6 Closed tube thoracostomy. **A,** Place a moderate-sized chest tube (32 to 40 Fr in adults or 26 to 30 Fr in children) either anteriorly in the fourth or fifth intercostal space midaxillary line (X) or in the second intercostal space, midclavicular (Y). The midaxillary line is generally preferred for cosmetic reasons; if the tube is positioned properly, superiorly toward the apex of the lung, it can effectively remove both fluid and air. **B,** Make a skin incision of approximately 3 cm in length through the skin along the fifth intercostal space. Use a Kelly clamp to create a pocket along the anterior and superior aspect of the rib to avoid the neurovascular bundle, which runs inferior and slightly medial to the rib.

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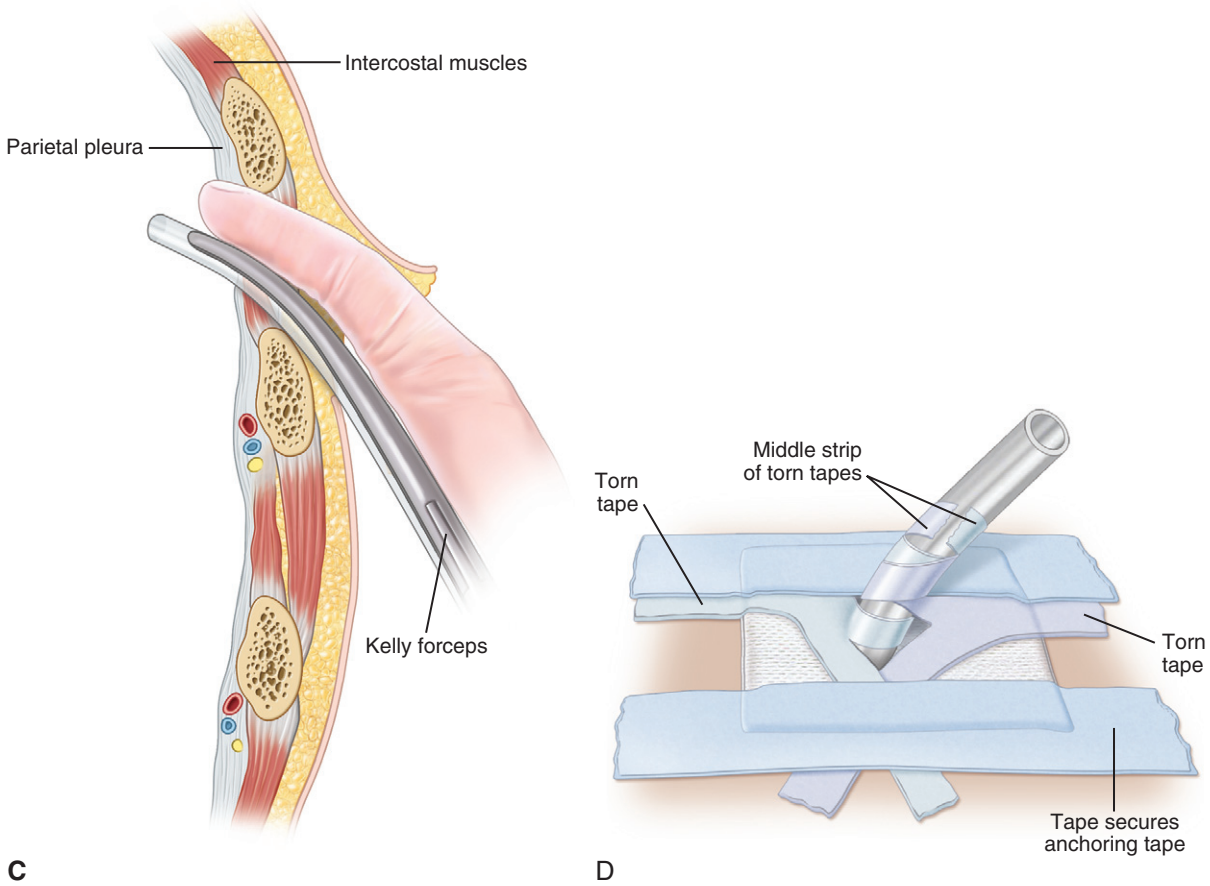


FIGURE 6-6, cont'd **C,** With a gloved finger, tunnel transversely through the subcutaneous tissue to the inferior margin of the fourth rib. Separate the intercostal muscles with a large Kelley clamp and insert the chest tube superiorly and posteriorly into the pleural cavity. **D,** Secure the tube to the skin with sutures, and use an occlusive dressing to cover the defect around the tube. A wide piece of tape is longitudinally split into three pieces; the two outside pieces are placed on the skin on either side of the tube, and the center strip is wrapped around the chest tube. A similar piece of tape can be secured to the tube at a 90-degree angle to the first piece. The tape is secured to the skin with an anchoring piece of tape. The tube is then connected to an underwater sealed drain to remove the air or fluid. Upright posteroanterior and lateral chest radiographs should be taken to confirm the position of the chest tube, the position of the last drainage hole on the tube, and the position and amount of air or fluid remaining in the pleural cavity.

after the initial chest tube was removed; (5) any pneumothorax in a patient who requires ventilatory support; (6) any pneumothorax in a patient who is undergoing general anesthetic; (7) associated hemothorax; (8) bilateral pneumothoraces; and (9) tension pneumothorax.[31]

OPEN PNEUMOTHORAX

An open pneumothorax, or sucking chest wound, that results from blunt chest trauma is associated with a large defect in the chest wall, and atmospheric air has direct access to the pleural cavity through the wound. Because of the loss of chest wall integrity, equilibrium develops between the intrathoracic pressure and atmospheric pressure. Complete expansion of the lung is impossible, because continued communication with the atmosphere is always present. The patient will have signs and symptoms of respiratory distress. During respiration, a sucking sound is often heard. The involved lung collapses on inspiration and slightly expands on expiration, causing air to be sucked in and out of the wound. This is referred to as a sucking chest wound. Also present is subcutaneous emphysema, which is usually extensive and involves both hemithoraces, cervical region, head, and anterior abdominal wall.

If the wound in the chest wall is approximately two thirds the diameter of the trachea, air will pass through the chest wall defect preferentially to the trachea. With the collapse of the involved lung and loss of negative pleural pressure, the expired air from the normal lung passes to the involved lung instead of out of the trachea, and the expired air returns to the normal lung on inspiration. Eventually, a large functional dead space develops in the normal lung and, combined with loss of the involved lung, may develop into a severe ventilation-perfusion problem.

Sucking chest wounds demand immediate surgical treatment. Patients may require intubation and ventilatory support. The wound should be covered with a sterile occlusive dressing secured on three sides of the dressing to the chest. The unsecured side of the dressing acts as a one-way valve, allowing air to escape the pleural cavity on expiration. Secure taping of all edges of the dressing

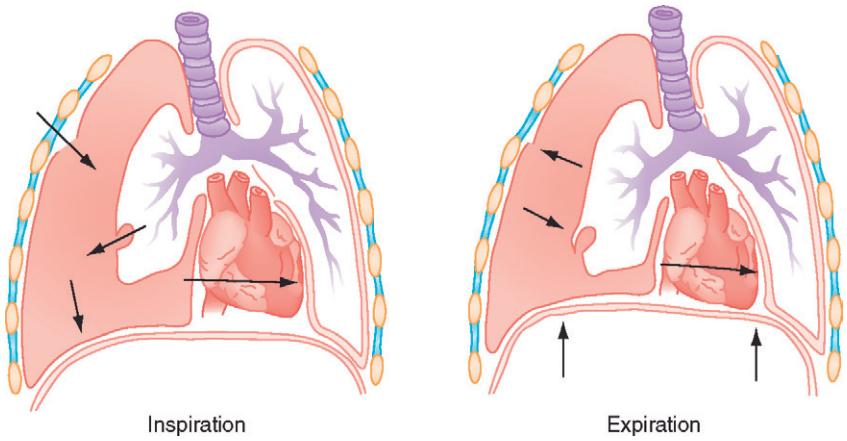


FIGURE 6-7 **A,** A tension pneumothorax develops as air enters into the pleural cavity on inspiration but cannot be removed during expiration. **B,** As the air builds in the pleural cavity, a progressive increase in intrapleural air pressure develops. The injury in the chest wall or trachea acts like a one-way valve and the increasing intrapleural pressure results in a shift of the trachea and mediastinal structures away from the injury. (From Marx JA, Hockberger RS, Walls RM: *Rosen's emergency medicine*, ed 7, St. Louis, 2010, Mosby.)

results in an accumulation of air within the thoracic cavity and a subsequent tension pneumothorax. Occlusive dressings such as petrolatum gauze may be used as a temporary measure during initial examination or over large defects. A chest tube must be placed in a distant site on the affected chest wall to prevent development of a tension pneumothorax, and the wound must eventually be closed in the operating room. If the lung does not expand after closure of the defect or if signs of poor ventilation persist, the patient should be placed on a ventilator with positive end-expiratory pressure (PEEP) to expand the lung. The patient should be carefully monitored and have a chest tube in place to prevent the development of a tension pneumothorax caused by a tear in one of the bronchi or in the lung parenchyma (Fig. 6-7). Signs of a tension pneumothorax in patients on ventilators include increased airway resistance and diminished tidal volume.



FIGURE 6-8 Hemothorax. The collection of blood in the inferior aspect of the pleural cavity blocks visualization of the diaphragm, ribs, and other structures in the upright chest film.

HEMOTHORAX

Hemothorax, a common sequela of blunt chest trauma, is usually unilateral; however, bilateral hemothoraces may occur in 10% to 20% of victims of blunt chest trauma (Fig. 6-8). The source of bleeding may be the lung, heart, great vessels or their branches, intercostal artery or vein, mediastinal veins, or vessels of the diaphragm and chest wall. Bleeding from the lung results if the lung is torn from a fractured rib driven inward toward the lung. A large amount of blood may be present without significant respiratory compromise and may be apparent only on the chest radiograph. More often, a large hemothorax under pressure can interfere with ventilation by causing a mediastinal shift, collapse of the lung, and resultant hypoxia. The shifting of the mediastinum further aggravates venous return and ventilation. This combination of hypovolemia and hypoxia can be lethal unless appreciated and corrected in a timely fashion.

DIAGNOSIS

If less than 400 mL of blood is lost, there may be little or no change in the patient's appearance or vital signs and no physical findings of intrapleural fluid may be present. With larger losses (>1000 mL), the findings of internal hemorrhage may be present—pallor, restlessness, tachycardia, and hypotension. The patient may complain of dyspnea or of a peculiar tightness in the chest. On the chest radiograph, the pleural fluid is best visualized with the patient in the upright position.

MANAGEMENT

The greatest problem with hemothorax is obtaining proper drainage. Although the initial hemothorax can be treated with thoracentesis, adequate drainage is of the utmost importance, necessitating placement of large-bore (32 or 36 Fr) thoracostomy tubes. Tube thoracostomy allows constant monitoring of continued blood loss and continued drainage for better reexpansion of the lung. Only 10% of patients require a thoracotomy (Box 6-2) for hemothorax from blunt trauma. Inadequate drainage (residual clot occupying more than one third of the thorax), sepsis, multiloculation, and hemodynamic or respiratory compromise are appropriate indications for operation. Improperly drained hemothoraces result

BOX 6-2 Radiographic Clues Suggestive of Thoracic Vascular Injury

Widening (>8 cm) of the upper mediastinum
Loss of aortic knob contour
Left apical pleural hematoma
Fractured sternum
Fractured first rib
Massive left hemothorax
Depression of the left mainstem bronchus more than 140 degrees
Calcium layering in the aortic knob (double-shadow sign)
Loss of aortopulmonary window on lateral chest film
Deviation of the trachea to the right
Deviation of the nasogastric tube in the esophagus away from the midline
Blurring of aortic contour

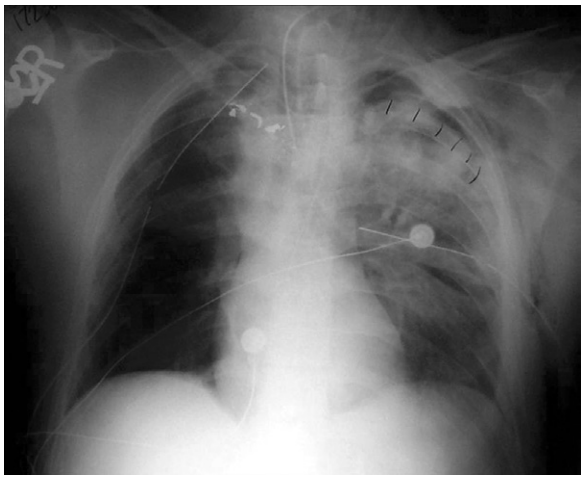


FIGURE 6-9 A pulmonary contusion, damage to the lung parenchyma that results in hemorrhage and edema without accompanying pulmonary laceration.

in a fibrothorax, restrictive lung disease, and possible respiratory compromise. Restoration of the circulating blood volume should be attempted with transfusion of fluids, volume expanders, and blood or blood products through large-bore IV lines.

A persistent hemorrhage requires surgical exploration. In a few cases, emergency thoracotomy in the emergency room may be necessary for control of blood loss. However, mortality from this procedure is very high.

The indications for thoracotomy for hemothorax are the following:

1. Drainage of 1500 mL of blood on placement of a chest tube
2. More than 300 mL/hr for 3 hours
3. More than 150 mL/hr for 3 hours in older or compromised patients
4. Lack of lung reexpansion
5. Bleeding with hemodynamic instability
6. Increasing hemothorax on chest radiographs despite tube thoracostomy

HEMOPNEUMOTHORAX

A hemothorax is usually associated with a pneumothorax secondary to the injury of the lung, chest wall, and associated vasculature. With a developing hemopneumothorax, the loss of blood causes hypotension, decreased cardiac output, and metabolic acidosis, which when combined with ventilatory compromise results in hypoxia and respiratory acidosis. Patients with hemopneumothorax, as identified by an air-fluid level on the chest radiograph, should have two chest tubes inserted, one through the fifth or sixth intercostal space at the midaxillary line and the other through the second or third intercostal space at the midclavicular line. The patient should be evaluated and treated as discussed in the earlier sections on pneumothorax and hemothorax.

PULMONARY CONTUSION

Pulmonary contusion is defined as damage to the lung parenchyma that results in hemorrhage and edema, without accompanying pulmonary laceration (Fig. 6-9). It is a common finding in patients who have sustained blunt chest trauma. A chest radiograph may expose signs of a pulmonary contusion, but a multislice CT scan provides better visualization of the extent of the contusion.[32] The incidence of pulmonary contusion in victims of nonpenetrating chest trauma varies between 30% and 75%.[33] Although often mild and frequently masked by other more dramatic injuries, such as flail chest, pneumothorax, and hemothorax, pulmonary contusion deserves special consideration. It is now well recognized that when undiagnosed and consequently untreated, pulmonary contusion is frequently progressive and may be fatal because of respiratory insufficiency. Pulmonary contusion has played a major role in 25% of deaths resulting from chest injuries sustained in MVAs.

Most victims of MVAs sustain a pulmonary contusion when the chest strikes the steering wheel during deceleration. The precise pathogenesis of pulmonary injury in these patients is unknown, but most believe it is analogous to the mechanism thought to operate in a blast injury—a forceful high-pressure wave that compresses the thoracic cavity. The force is also transmitted to the lung by virtue of its continuity with the tissue of the chest wall. The increase in intrathoracic pressure compresses the lung by diminishing the size of the thorax and results in parenchymal hemorrhage and edema. When the force of compression is removed, decompression occurs and the distorted thorax springs back, creating an instant of negative intrathoracic pressure that leads to additional injury in the areas ruptured during compression. Among the factors that influence the severity of lesions produced are the amount of padding of the chest wall and flexibility of the chest. If the ribs are sufficiently elastic, severe compression of the lung can occur without a break in the costal cartilage. Some of the most severe pulmonary contusions occur in patients who exhibit neither rib fractures nor discontinuity of the chest cage.

Pulmonary contusion is frequently associated with multiple injuries and is often subject to incidental

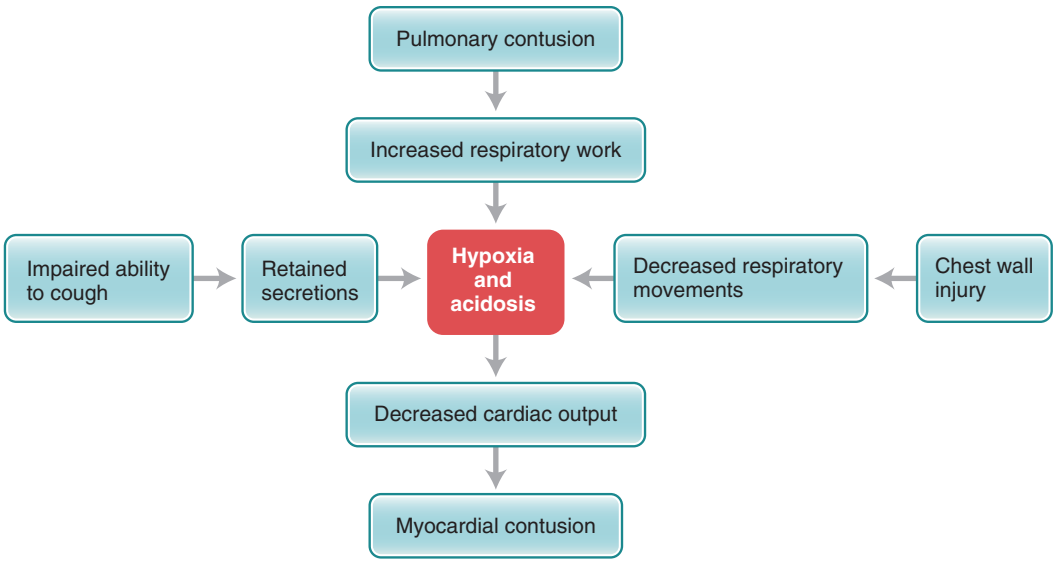


FIGURE 6-10 Pathophysiology of untreated pulmonary contusion.

resuscitative measures, such as administration of large volumes of fluid, which are directed at other injuries. The rapid administration of large volumes of noncolloidal fluid has been shown in experimental and clinical studies to have an adverse effect on the already damaged lung. Figure 6-10 examines the pathophysiology of an untreated pulmonary contusion.

TREATMENT

It should be emphasized that the keys to successful management in patients with pulmonary contusions are as follows:

1. Early and vigorous therapy (the first 24 hours of treatment are the most important)
2. Restoration and maintenance of oxygenation
3. Intense pulmonary hygiene, including intratracheal suction and physical therapy

The following steps should be taken in the treatment of pulmonary contusion:

1. A percutaneous radial artery catheter should be inserted for frequent ABG determinations.
2. Crystalloid solution should be administered based on hemodynamic measurements.
3. A nasogastric tube should be inserted for gastric decompression and nutritional support.
4. Adequate pain relief should be achieved with small frequent doses of narcotics, epidural catheter, or intercostal nerve blocks.
5. When appropriate, broad-spectrum antibiotics should be started for documented infection.
6. Indications for ventilatory support by means of endotracheal intubation are listed in Figure 6-1. The lowest FiO_2 possible should be used to maintain a PO_2 of 60 mm Hg. Positive end-expiratory pressure should be used if a PO_2 of at least 60 mm Hg or an FiO_2 of 60% cannot be obtained. The hematocrit should be kept at a level greater than 30% to optimize mixed venous oxygen and oxygen use.
7. The pH should be maintained at a level higher than 7.35, because an alkaline pH allows for greater oxygen dissociation (shifting the curve to the left reduces the ability of hemoglobin to release oxygen).

It is imperative that nutritional support be adequate in these patients. Impairment of host defenses secondary to malnutrition may result in bacterial translocation from the gut and multiple organ system failure from sepsis.

The course of pulmonary contusion is determined by the severity of the initial injury. In patients with mild pulmonary contusion who do not require ventilatory support, the course of the illness is characterized by rapid resolution within 72 hours. In patients who require ventilatory support, gradual improvement occurs, with weaning from the ventilator in 12 to 14 days. In any patient with a pulmonary contusion and delayed improvement, one should suspect superimposed processes, such as pneumonia, fat embolism, and pulmonary embolism. Despite optimal therapy, approximately 15% of patients with pulmonary contusion die of progressive respiratory insufficiency.

RIB FRACTURES

Rib fractures are a common injury that can affect 350,000 people each year. Rib fractures lead to respiratory complications prolonged hospitalizations, pain, disability, nosocomial pneumonia, and increased morbidity.[12] The incidence and location of rib fractures appear to be related to the age of the patient. Fractures of the ribs are more common in adults than in children because the resilient chest wall of the child can absorb more of an impact without fracture. In contrast, the ribs of older adults are brittle and can be broken by even minor stresses, including those associated with coughing. Rib fractures usually occur at the posterior angle, the weakest point in the rib structurally, and the area that tends to be under the most pressure during impact. Rib fractures associated with blunt trauma occur most commonly because of chest compression and most often involve the fourth through ninth ribs laterally, because these are

more exposed. The compression type of fracture usually results in an outward break, in contrast to the fractures resulting from direct trauma. One should be suspicious for abdominal injuries if the tenth to twelfth ribs are broken.

The number of rib fractures, degree of displacement of the rib fragments, and injury to the underlying lung are dependent on the force and direction of the impact and the area of its distribution. Rib fractures can be complete or incomplete, or have varying degrees of overlap. If a fracture is displaced more than 1 to 2 cm, the likelihood of healing is diminished.[12] If the injuring force is applied over a wide area, especially in the anteroposterior projection, the ribs buckle outward and break at the midshaft position, without injuring the underlying pulmonary parenchyma. A direct force applied over a small area tends to push the rib fragments inward and can cause lacerations of the pleura, pulmonary parenchyma, and intercostal vessels, producing hemothorax, pneumothorax, or both.

One of the most significant complications associated with rib fractures, especially in patients with preexisting chronic lung disease, is pneumonia and possibly respiratory failure. Rib fractures are invariably accompanied by pain that results in splinting, reducing effective deep breathing and ventilation, and causing alveolar collapse, secretion accumulation, and atelectasis. As atelectasis develops, a vicious cycle is set up that leads to hypoventilation and further collapse. Hypoventilation can result in pulmonary infection from retained secretions. Therefore, it is important to supply supportive care and adequate pain control so that deep breathing, coughing, and clearing of secretions can be continued.

DIAGNOSIS

The physician can arrive at the diagnosis of rib fractures by noting the history of trauma and by eliciting pain on palpation. The presence of fractures is confirmed by chest radiograph. Because anterior or lateral rib fractures may not have been seen on an anteroposterior chest radiograph, it is necessary to obtain left anterior and right anterior oblique views as well, so that all regions of the ribs may be seen. It may be important to obtain serial x-rays in all patients with rib fractures, because delayed pneumothorax or hemothorax can develop after the initial injury, along with atelectasis or pneumonia from hypoventilation.

TREATMENT

When pneumothorax or hemothorax complicates rib fractures, these are treated promptly before the treatment of the rib fractures. Usually, tube thoracostomy drainage is needed on the appropriate side. Treatment of rib fractures is dependent on the severity of the injury, age of the patient, extent of preexisting lung disease, and pain threshold of the patient. In older patients, a rib fracture is a serious injury; these patients should be hospitalized regardless of the initial appearance to evaluate possible underlying injury and monitor for subsequent hypoventilation. An older patient's mortality risk increases with the number of fractured ribs. Patients older than 65 years have a twofold increase in mortality compared with patients younger than 65 with more than five rib fractures. Similarly, patients with poor cardiovascular reserve or underlying chronic lung disease should be hospitalized, because the additional insult of the rib fracture may result in pulmonary or cardiac decompensation. Narcotics should be given immediately to control pain and should be repeated as often as necessary. Epidural anesthesia or an intercostal nerve block may be beneficial and should include two segments above and two segments below the fractured site. With adequate analgesia and physiotherapy, the patient should be able to cough and breathe deeply, and thus bronchial secretions can be effectively expelled, which limits atelectasis and prevents pneumonia. Older methods of adhesive chest strapping limit respiratory excursion and result in further decompensation of respiratory status. Consequently, the use of chest strapping should be condemned.

FIRST RIB FRACTURES

At one time, first rib fractures were thought to be of significant consequence, because the first rib lies low in the neck as a short, broad, and relatively thick structure requiring extreme force to be fractured. Interestingly, the mechanism of injury is important, rather than the actual first rib fracture itself. Usually, in these cases, major chest, abdominal, and cardiac injuries are relatively infrequent; however, a high incidence of serious maxillofacial and neurologic injuries has been noted. Associated injuries of the subclavian, carotid arteries, and brachial plexus have been reported in 5% to 15% of patients with first rib fractures.[33] The following are indications for an arteriography in patients with first rib fractures:

1. Absent or decreased upper extremity pulses
2. Evidence of brachial plexus injury
3. Marked displacement of fragments, especially posterior displacement
4. Altered serial chest radiographs
5. Subclavian groove fracture seen anteriorly

No specific therapy exists for first rib fractures. The only significance of these fractures is that they alert the physician to the possibility of associated intrathoracic and extrathoracic injuries, particularly maxillofacial and neurologic injuries.

FLAIL CHEST

Flail chest may result when each of three or more contiguous ribs is fractured at two points (Fig. 6-11). Flail chest may also be generated by anterior dislocation of the costochondral junction coupled with a lateral or posterior fracture. Multiple fractures in several ribs result in a damaged segment of chest wall that no longer maintains its bony continuity with the remainder of the chest and becomes flail. Because the flail portion of the chest wall is subject to changes in intrathoracic pressure, it begins to move independently of and paradoxically to the intact portion of the chest wall. A flail segment may occur anteriorly, laterally, or posteriorly on the chest. In a flail chest injury, the unstable segment of the normally

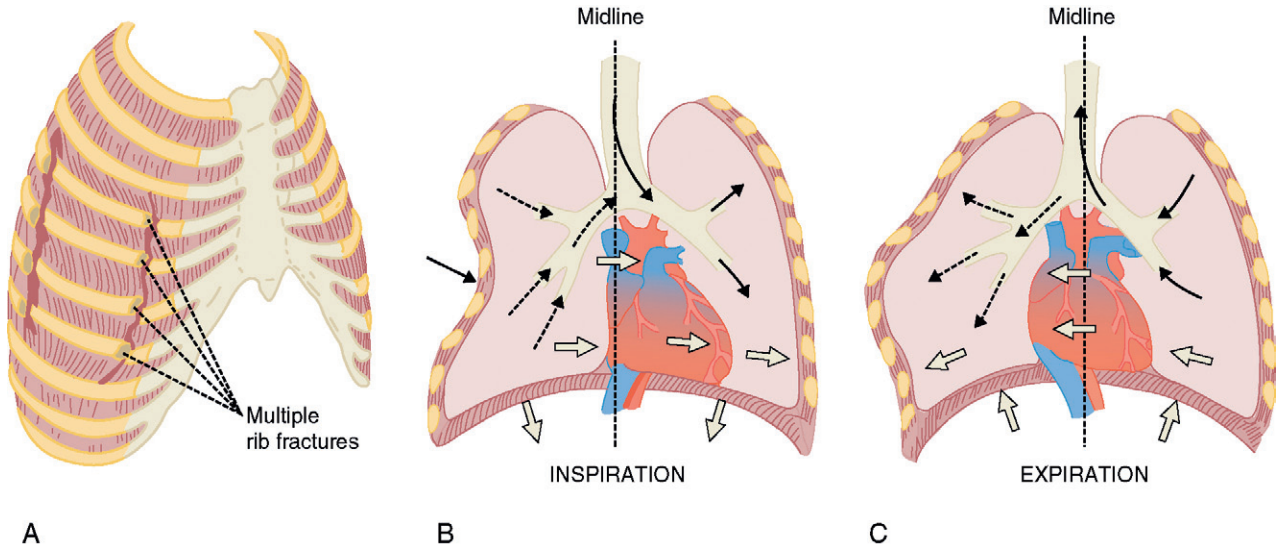


FIGURE 6-11 **A,** Flail chest occurs when three or more adjacent ribs are fractured in at least two locations, resulting in a freely moving segment of chest wall during respirations. The chest wall moves paradoxically during inspiration and expiration because of the flail segment. **B,** On inspiration, the flail segment sinks inward as the chest wall expands, impairing the ability to produce negative intrapleural pressure. The heart and other contents of the mediastinum shift toward the noninjured side. **C,** During expiration, the flail segment is pushed outward, the chest wall cannot efficiently force air from the lungs, and air may shift uselessly from lung to lung. (From Black JM, Hawks JA: *Medical-surgical nursing*, ed 8, St. Louis, 2009, Saunders.)

rigid chest wall moves separately in the opposite direction from the rest of the thoracic cage as the patient attempts to inhale and exhale. The lateral type of flail chest is the most common. The anterior type of flail results when the ribs become separated at the costochondral junction, with or without an associated fracture of the sternum. The posterior type of flail occurs when the posterior ribs are fractured. In this case, paradoxical motion is minimal because of the support provided by the scapula and associated musculature.

The underlying problem with a flail chest is not so much that of chest wall deformity as the underlying parenchymal injury. A significant amount of force is required to cause multiple rib fractures, and this force is transferred internally to the underlying lung. During inspiration, the flail segment moves inward and during expiration the flail segment is pushed outward. Other significant injuries, intrathoracic and extrathoracic, are associated with a flail chest. The incidence of pulmonary contusion with a flail chest is almost 70%.[34,35] In these patients, hemothorax is seen in 50% and pneumothorax in 30%. More than 20% of patients have significant abdominal injuries, neurologic injuries, or both.

PATHOPHYSIOLOGY

The physiologic alterations that occur with flail chest result not only from the disruption in chest wall mechanics but also from the associated pulmonary injury. As a consequence of the chest wall injury, the bellows action is reduced in the chest wall. During inspiration, the intact portion of the rib cage expands, drawing air into the lungs. However, the flail portion does not expand, because it is no longer in continuity with the normally expanded portion. Atmospheric pressure is exerted on the unstable segment, forcing it inward. The negative intrapleural pressure enhances this inward motion. A reverse relationship develops on expiration in that the intrathoracic pressure exceeds atmospheric pressure and the flail segment is pushed outward as the remainder of the thorax contracts normally. Thus, the loss of the structural integrity of the thoracic cage does not permit generation of sufficient negative intrapleural pressure and respiratory work increases in an attempt to overcome this abnormality.

The associated pulmonary parenchymal injury produces additional physiologic derangement. As a result of the lung damage (contusion and atelectasis), lung compliance is notably reduced, airway resistance is increased, pulmonary diffusion is decreased, and the ventilation-perfusion ratio is altered. These changes lead to an even further increase in respiratory work.

DIAGNOSIS

The diagnosis of flail chest injury is usually made by physical examination with careful inspection of the unclothed patient. Observation of chest wall excursion demonstrates paradoxical respiration of the involved chest segment. Excursions of the chest wall are best observed while the physician stands by the patient's side and puts his or her hand on the injured hemithorax. Paradoxical motion can be accentuated by having the patient taking a deep breath or cough. The chest radiograph is of limited value in establishing the presence of paradoxical respiration, but is useful in demonstrating chest wall fractures, pulmonary contusion, atelectasis, hemothorax, or pneumothorax. Although flail chests are usually readily identifiable clinically, a specific search should be made for double fractures of three or more adjacent ribs, a rib fracture associated with sternal fracture, or a costochondral separation.

BOX 6-3 Indications for Treatment of Flail Chest with Mechanical Ventilation

Respiratory failure manifested by one or more of the following criteria:

- Clinical signs of progressive fatigue
- Respiratory rate > 35 breaths/min or <8 breaths /min
- PaO_2 < 60 mm Hg at FiO_2 > 0.5
- $PaCO_2$ > 55 mm Hg at FiO_2 >0.5
- PaO_2 /FiO_2 ratio < 200
- Vital capacity < 15 mL/kg
- FEV < 10 mL/kg
- Inspiratory force > –2cm H_2O
- Alveolar-arterial oxygen gradient ($AaDO_2$ [in mm Hg] at FiO_2 1.0) > 450
- Shunt fraction (Qs/Qt) > 0.2
- Dead space tidal volume ratio (V_{DS}/V_T) > 0.6
- Clinical evidence of severe shock
- Associated severe head injury with lack of airway control or need to ventilate
- Severe associated injury requiring surgery
- Airway obstruction

FEV, Forced expiratory volume in 1 second.

BOX 6-4 Elements of Treatment of Flail Chest without Mechanical Ventilation

Supplemental oxygen administration
Humidification of inspired air
Active physical therapy
Incentive spirometry
Nutritional support
Oscillating or rotating bed
Analgesia
Nonsteroidal anti-inflammatory drugs (NSAIDS)
Parenteral narcotic administration
Patient-controlled analgesic devices
Continuous epidural analgesic
Intermittent positive-pressure breathing
Upper airway and endobronchial suctioning
Early stabilization of long bone fractures
Continuous reassessment
Physical examination
Serial chest x-rays
Serial arterial blood gas determinations
Oximetric monitoring
Serial spirometric testing
Surveillance for pulmonary morbidity
Scheduled outpatient follow-up

Arterial blood gas measurements are of value in estimating the severity of the patient's condition, even in the absence of obvious symptoms. These measurements may provide crucial information if the patient's clinical course deteriorates and can be used as the basis for instituting ventilatory support. Most patients with a flail chest have a low partial pressure of oxygen (PO_2) while breathing room air. A normal or elevated pH in association with an elevated partial pressure of carbon dioxide (PCO_2) indicates the presence of severe parenchymal injury.

TREATMENT

The treatment of flail chest depends on the underlying parenchymal injury. It includes aggressive pulmonary physiotherapy, effective analgesia, selective use of endotracheal intubation, and close observation for alterations in clinical signs and symptoms. The flail segment disrupts the mechanics of breathing, prevents lung inflation, and produces significant ventilatory compromise and hypoxia. The underlying pulmonary contusion, not the mechanical disruption of the chest wall, causes the respiratory insufficiency. Specific indications for endotracheal intubation depend on objective evidence of respiratory failure (Box 6-3), such as measurements of ABG levels, respiratory rates, intrapulmonary shunting, and pulmonary mechanics. Indications for intubation include a PO_2 less than 60 mm Hg while the patient breathes room air or less than 80 mm Hg while the patient breathes supplemental oxygen, a tidal volume (V_T) less than 10 mL/kg of body weight, hypercapnia (PCO_2 > 55 mm Hg), respiratory rate greater than 35 breaths/min, or the inability to clear secretions and protect the airway.[36] Not all patients with flail chest injury require intubation (Box 6-4). Patients without respiratory impairment do not require ventilatory assistance and tend to have lower morbidity and mortality than patients who require intubation.[17-19,22] Tracheostomy is not routinely indicated because many patients with flail chest injury require only brief periods of ventilatory support. Tracheostomy should be considered in patients who have significant craniofacial, maxillary, and mandibular injury—especially with an unstable airway or evidence of upper airway obstruction—and in those intubated patients in whom ventilatory support is expected to exceed 7 to 10 days.[37]

Patients with unilateral paradoxical motion, a small volume of chest wall paradox, mild to moderate pulmonary contusion, and an arterial partial pressure of oxygen (PaO_2) greater than 60 mm Hg while breathing room air (or >80 mm Hg while breathing supplemental oxygen), or with a tidal volume greater than 10 to 15 mL/kg of body weight, can be treated without mechanical ventilation. The treatment regimen is the same as that discussed for pulmonary contusion. Patients undergoing therapy in this manner should be observed carefully and have frequent ABG measurements. If signs of ventilatory insufficiency develop, endotracheal intubation must be carried out promptly and mechanical ventilation instituted.

Adjunctive treatment for flail chest and pulmonary contusion has historically included diuretics, fluid restriction, steroids, and prophylactic antibiotics. However, there has been limited evidence that these measures enhance outcome or prevent pulmonary morbidity, and they are not routinely recommended.*

Effective pain management and aggressive chest physiotherapy, including suctioning, incentive spirometry,

*References 11, 27, 29, 30.

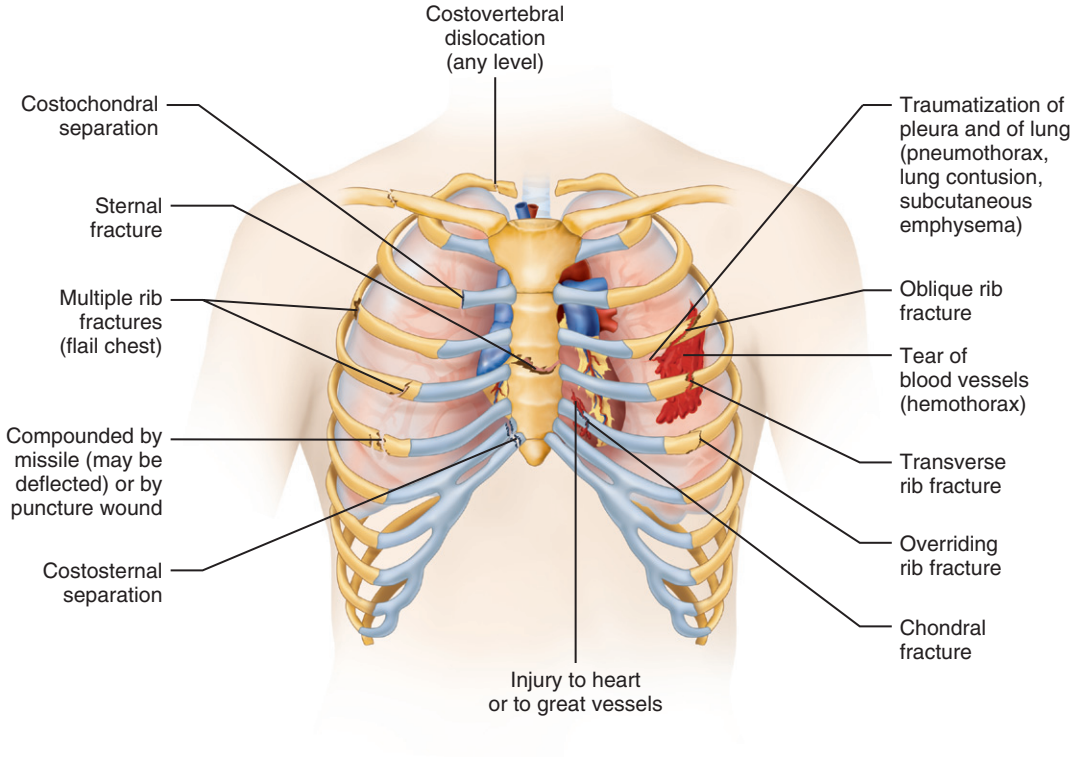


FIGURE 6-12 Blunt chest trauma can cause fractures of several components of the thoracic cage, including the ribs, clavicle, sternum and, importantly, result in significant damage and complications to the heart, lungs, and other structures of the thoracic cavity.

humidification of air, and patient mobility, are useful and important to enhance outcome and minimize morbidity. Coughing is difficult and extremely painful for patients with flail chest because it is difficult for the patient to generate a cough with several fractured ribs. The explosive force of the cough is limited secondarily to the paradoxical movement of the chest wall. Because these patients are limited in their ability to cough, bronchial secretions cannot be effectively removed by the patients and secretions may accumulate, with possible atelectasis and pneumonitis. Current treatment relies on supportive measures; however, fixation of the fractured rib segments has been advocated to restore chest wall mechanics. Two small studies have conducted randomized trials suggesting that flail chest would benefit from open reduction and internal fixation.[12] They have found decreases in the number of days a person is in the intensive care unit (ICU) and on the ventilator.

The patient must be provided with adequate means of pain control. Chest wall injuries can be painful, limiting the patient's ability to endure aggressive chest physiotherapy. Pain management techniques include nerve blocks of the intercostal nerves, intrapleural local anesthetics administered via the chest tube, nonsteroidal antiinflammatory drugs (NSAIDs), narcotic and synthetic opioids, epidural and/or systemic analgesics, continuous and patient-demand local anesthetic or narcotic infusion systems, and other means of local or systemic pain management.

The mortality rate from flail chest varies from 15% to 89%, with death directly related to the age of the patient, severity of the underlying parenchymal injury, and number of associated injuries.[9,19] In Symbas' series,[38] a flail chest combined with pulmonary contusion was associated with a 40% mortality rate. The mortality rate for patients younger than 30 years or for those whose sole injury is a flail chest is less than 3%. However, the mortality rate approaches 40% in patients with head injuries and 60% in patients older than 60 years.

FRACTURED STERNUM

Blunt chest trauma associated with steering wheel injuries is common (Fig. 6-12). However, fractures of the sternum are relatively rare, occurring in only 5% of all victims of blunt trauma. These fractures are seen primarily in older patients, rather than in children and young adults, because of the acquired inelasticity of the chest wall. Almost all fractures occur in a transverse plane and most occur in the body of the sternum, near its junction with the manubrium. Fractures of the xiphoid process rarely occur because of its protected position between the flare of the costal margins bilaterally. The diagnosis is confirmed by visualization of the fracture site on a lateral or oblique chest radiograph or a radiograph of the sternum. A significant force is usually necessary to produce a sternal fracture; thus, associated injuries are common. It is therefore important to evaluate all patients with sternal fractures for associated cardiac injuries or other intrathoracic trauma. Injuries associated with a fractured sternum include the following:

1. Myocardial contusion
2. Pulmonary contusion
3. Flail chest
4. Pneumothorax
5. Hemothorax
6. Hemopericardium
7. Pericardial laceration
8. Ruptured bronchus
9. Cardiac rupture
10. Ruptured thoracic aorta
11. Abdominal visceral injuries
12. Spinal injuries

Most sternal fractures are nondisplaced and treatment is directed only toward pain relief. Analgesics usually control significant sternal pain but, if they are not sufficient, they can be supplemented with anesthetic injection into the fracture site. Although sternal fractures rarely require any surgical intervention, open reduction and stabilization should be carried out in all patients with severely displaced fractures, especially in those who exhibit paradoxical motion, because nonunion is common. These repairs should be delayed until the other associated injuries have been evaluated and treated because the mortality associated with sternal fractures is directly attributed to the associated injuries and not to the fracture itself.

CERVICAL TRACHEAL DISRUPTION

Cervical tracheal injuries are caused by direct trauma to the cervical area. Rupture of the cervical trachea may occur without any external evidence of trauma. There are a number of symptoms of cervical tracheal disruption, including subcutaneous emphysema, inspiratory stridor, hoarseness, hemoptysis, dyspnea, coughing, localized pain or tenderness, and cyanosis. The most common early sign of cervical tracheal disruption is subcutaneous emphysema, which develops rapidly and spreads extensively over the chest and anterior aspect of the neck (Box 6-5). Pneumothorax, a common complication, is usually mild and responds promptly to chest tube insertion. Disruption of the cervical trachea must always be considered in any victim of blunt chest trauma who manifests respiratory distress in association with extensive subcutaneous emphysema. If rupture of the cervical trachea is suspected, an initial attempt at intubation is warranted, with a flexible scope. If intubation is not possible, a tracheostomy below the level of the injury will be necessary. Performing an acute tracheostomy can be a time-consuming and difficult procedure when done in the emergency room. In such cases, surgical cricothyroidotomy might be an option.

Bronchoscopy is an important additive to ensure appropriate tracheal placement. Associated injuries to the cervical spine, esophagus, and great vessels of the neck must be avoided, especially if a developing cervical hematoma is present. As soon as the patient's airway is established and stabilized, the tracheal disruption should diminish. Prior to removal of the tracheostomy tube, the functional status of the vocal cords and presence or absence of a previously unrecognized laryngeal fracture should be determined.

BOX 6-5 Causes of Subcutaneous Emphysema

- Perforation of trachea, bronchial tree, esophagus
- Lung injury
- Injury to face tracking inferiorly through fascial spaces
- Injury to retroperitoneal space that tract through diaphragmatic hiatus
- Introduced from outside as a result of penetrating injury

TRACHEOBRONCHIAL INJURIES

Tracheobronchial injuries are relatively rare but can be a devastating consequence of blunt chest trauma if unrecognized or improperly treated. Prior to reaching the emergency department, 75% of patients die.[39] They represent 1% to 3% of all thoracic injuries associated with blunt chest trauma and are frequently associated with other major thoracic injuries, such as cardiac, great vessel, or esophageal injuries. Blunt trauma that causes tracheobronchial injury has a high incidence of associated injuries, and one third to one half have concomitant facial trauma. Although every level of the tracheobronchial tree can be involved, more than 80% of the injuries are within 2.5 cm of the carina. Most injuries are to the intrathoracic trachea or right mainstem bronchus. Cervical tracheal injuries represent 4% of injuries, left bronchus injuries 17%, distal tracheal injuries 22%, and right bronchus injuries 27%.

The mechanisms of injury causing tracheobronchial injuries include forceful compression of the trachea and thoracic cage, increased airway pressure, rapid deceleration injuries with shear forces at the point of relative fixation of the trachea (i.e., carina), and direct injuries.

In patients with tracheal or major bronchial disruption, two clinical patterns exist, depending on the relationship between the disrupted airway and pleural space. Patients who have a free communication between the site of disruption and pleural cavity will have a large pneumothorax. Insertion of a chest tube results in continuous bubbling of air and failure of the lung to reexpand with suction. The usual signs and symptoms in this group of patients include hemoptysis, dyspnea, and extensive subcutaneous emphysema. In patients in whom there is no communication between the site of disruption and pleural cavity, usually little or no pneumothorax exists.

Chest and cervical spine plain films can be most useful in showing disruption of the tracheobronchial tree. Bronchoscopy is the single definitive diagnostic study, direct or fiberoptic.[39] Securing the airway is the highest priority for tracheal bronchial injuries. Flexible bronchoscope may facilitate intubation or oral intubation with cervical spine stabilization. The necessity of a tracheotomy can be up to 4%. A cricothyroidectomy is not as useful because the injury is usually distal.

If a pneumothorax is present, suction will reexpand the lung. However, the surrounding granulation tissue gradually obstructs the site of transection and delayed

atelectasis occurs. Nonoperative therapy is recommended only for those patients with partial tears, complete reexpansion of the lung, and no prolonged air leak. All other patients with transection of the tracheobronchial tree should undergo repair to avoid disastrous complications, such as bronchiectasis or obstruction with secondary infection and lung destruction after they have been initially stabilized. If the injury is proximal, a low cervical collar approach can be used. The injury should be repaired with interrupted sutures. The operator should be sure to examine the esophagus at the same time. If the injury is more distal (intrathoracic), a sternotomy or thoracotomy approach may be indicated. Any devitalized or necrotic tissue should be débrided. Most injuries can be closed primarily and the injured area is resected; primary repair of up to 50% of the trachea can be closed primarily with the use of neck flexion. A guardian suture can be placed, which helps connect the chin to the sternum and prevent disruption of the repair.

ESOPHAGEAL INJURIES

Blunt injury to the esophagus is extremely rare because of the tremendous force required to cause injury and its position in the posterior mediastinum. Lack of tethering of the esophagus helps protect the esophagus from tearing or disruption during deceleration injuries, unlike the aorta.[40] The diagnosis is frequently made late in the patient's course of treatment. Signs and symptoms of esophageal injury include acute dysphasia, or the inability to swallow saliva; 92% of patients have dysphasia and 60% have neck tenderness. A total of 90% of these injuries are intrathoracic, occurring just above the level of the gastroesophageal junction. Of blunt esophageal injury victims, 19% have cervical emphysema. If suspected, a biplane esophagogram, esophagoscopy, or both should confirm the diagnosis. Esophageal injuries are diagnosed by lateral radiographs of the cervical or upper thorax. An examination of the radiograph for emphysema in the mediastinum can indicate esophageal perforation. If the patient's signs or symptoms lead to a strong suspicion, a Gastrografin swallow study or open exploration in the acute setting or sepsis is the gold standard evaluation. An increase in the inflammatory response because of esophageal injuries such as leukocyte increase with a left shift, low PO_2, or acidosis can be seen. Most of these injuries can be repaired coupled with wide mediastinal drainage to prevent empyema from developing. Tracheoesophageal fistula can be a long-term consequence. The patient should be kept NPO, have a nasogastric tube placed, and be given IV antibiotics.

PENETRATING NECK TRAUMA

Vascular injury is the serious consequence of penetrating neck trauma. Signs and symptoms of neck injury include impending airway compromise of garbled or loss of speech, hoarseness or dysphonia, persistent bleeding from oropharynx, and respiratory distress.[16] A secure airway is the highest priority in penetrating neck injuries by endotracheal intubation, cricothyrotomy, or tracheostomy, with as little manipulation of the neck as possible, protecting from cervical injuries. After initial primary and secondary surveys have been conducted, the patient can be definitively managed. The neck is divided into three zones to help communication among surgeons and help plan treatment of possible injuries associated with injury to each zone (Table 6-3)[16]:

TABLE 6-3 Contents of Neck

Zone 1	Zone 2	Zone 3
Clavicle to inferior cricoid cartilage	Inferior border of cricoid cartilage to angle of mandible	Angle of mandible to skull base
Great vessels of neck, aortic arch, subclavian vessels, trachea, esophagus	Great vessels, aerodigestive tract, cervical spine	Cranial cavity, great vessels, cervical spine, larynx, pharynx

Adapted from Kesser BW, Chance E, Kleiner D, et al: Contemporary management of penetrating neck trauma. *Am Surg* 75:1, 2009.

Zone 1: Sternal notch or clavicles to inferior border of cricoid cartilage. Structures in zone 1 include the great vessels of the neck, aortic arch, subclavian vessels, trachea, and esophagus.

Zone 2: Inferior border cricoid cartilage to angle of mandible. Structures in zone 2 include the great vessels, aerodigestive tract, and cervical spine.

Zone 3: Structures in zone 3 include the angle of mandible to the skull base, cranial cavity, great vessels, cervical spine, larynx, and pharynx.

Accessing zone 1 and 3 is difficult. Previously, injuries to zone 2 that penetrated the platysma were mandated to be explored surgically.[16] Because of a high incidence of negative neck explorations, surgical exploration is no longer mandatory. Also, a negative neck rate as high as 75% had been seen. Current recommendations include surgical intervention in the unstable patient (e.g., hemodynamic instability, airway, shock, hematoma, active brisk bleeding, air sucking in or out of the wound). The ABCs are performed first. Stable patients can now benefit from a comprehensive workup, including CT with and without angiography. Immediate interventions for the unstable patient should include DL, bronchoscopy, and/or esophagoscopy. The common carotid artery is the most common injury in zone 2. Patients who remain hemodynamically stable must receive routine physical examinations to monitor for changes in vital signs and symptoms.

DIAPHRAGMATIC INJURIES

Traumatic disruption of the diaphragm is an infrequent occurrence in victims of nonpenetrating trauma. It is detected in only approximately 3% of patients who survive MVAs and in 4% to 7% of immediate fatalities. Left-sided diaphragmatic hernias occur in 95% of these patients; the remaining patients having right-sided hernias (Fig. 6-13). A force of great magnitude is needed to disrupt the diaphragm; therefore, associated



FIGURE 6-13 Diaphragmatic rupture, with massive herniation of intraabdominal contents into the chest. This herniation results in compression of the lung and displacement of the mediastinum to the contralateral side, followed by marked respiratory distress, cyanosis, and hypotension. The characteristic radiographic findings are elevation of the left hemidiaphragm, presence of an air-fluid level above the diaphragm, and displacement of the heart and mediastinal structures.

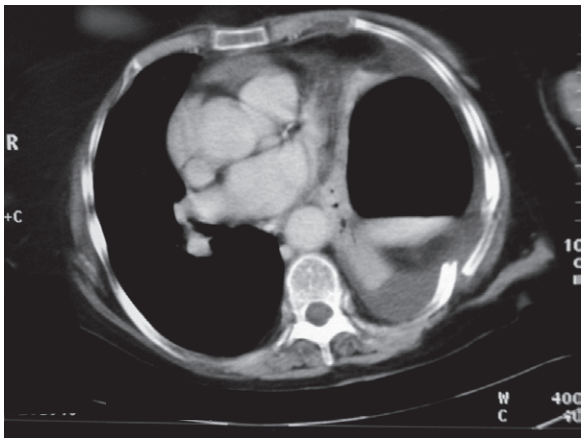


FIGURE 6-14 Diaphragmatic rupture.

intra-abdominal injuries are common, such as spleen, kidney, or liver lacerations and intestinal perforations. Those with small diaphragmatic tears are usually asymptomatic. Those with large diaphragmatic tears usually have symptoms as a consequence of massive herniation of the intra-abdominal contents into the chest (Fig. 6-14). This herniation results in compression of the lung and displacement of the mediastinum to the contralateral side, followed by marked respiratory distress, cyanosis, and hypotension. The characteristic radiographic findings are elevation of the left hemidiaphragm, presence of an air-fluid level above the diaphragm, and displacement of the heart and mediastinal structures. The diagnosis of traumatic disruption of the diaphragm should be suspected in any patient who has sustained blunt trauma to the chest and in whom the previously described clinical and radiographic findings are present. Note that the treatment of every form of traumatic disruption of the diaphragm is surgical. The mortality rate for traumatic disruption is dependent on the severity of associated injuries and degree of cardiorespiratory distress that results from herniated abdominal contents.

RUPTURE OF THE AORTA

An estimated 800 deaths annually are caused by aortic disruption.[37] Aortic injury is the second most common cause of death in blunt trauma next to brain injury. The cause of aortic injury is mostly MVAs and falls, with MVAs resulting in 80% of injuries. In 1990, only 9% of aortic ruptures survived at the scene. For drivers and front seat passengers, the steering wheel deformity on arrival to the emergency department is an independent predictor of serious thoracic injury . Classification of aortic injuries are in six groups: intimal hemorrhage, intimal hemorrhage with laceration, medial laceration, complete laceration, false aneurysm formation, and periaortic hemorrhage. Patients who arrive alive usually have an incomplete, noncircumferential lesion to the media or intima. The tunica adventitia and mediastinal pleura then prevent rupture. A patient with a suspected thoracic vascular injury should be evaluated immediately by a thoracic surgeon. Of those who have sustained a traumatic rupture of the aorta, 10% to 20% reach the hospital alive because the aortic blood is contained temporarily by the adventitial or mediastinal pleura, forming a pseudoaneurysm. Left untreated, approximately 25% of patients will die within the first 24 hours, with 5% of the remaining patients dying each day during the next 2 weeks. Approximately 6% of the original group will survive to develop chronic injuries, leading to a rate of late rupture of 2%/year. To prevent this catastrophic outcome, it is imperative that the diagnosis be established quickly and appropriate therapy be instituted.

Traumatic rupture of the thoracic aorta is caused by the following mechanisms:

1. Deceleration with or without chest compression, most commonly encountered in motor vehicle collisions or in falls from great heights
2. Compression or crushing injuries of the chest; injuries frequently sustained when a person is run over by a car or kicked in the chest by a large animal

The more frequent sites of rupture of the thoracic aorta are as follows:

1. Descending aorta (aortic isthmus) immediately distal to the origin of the left subclavian artery (55%)
2. Ascending aorta, proximal to the origin of the innominate artery (19%)
3. Innominate artery origin (4%)

Other sites of involvement include the left subclavian artery origin, midportion of the descending aorta, and distal descending aorta at the diaphragmatic hiatus (20%).

INJURIES OF THE DESCENDING THORACIC AORTA

The classic site of blunt aortic injury is the descending aorta at the isthmus, resulting in 36% to 54% of injuries. They can also occur at the ascending aorta proximal to the origin of the brachiocephalic, aortic arch, and distal descending or abdominal aorta.[12,37,41,42] The high index of suspicion that vascular trauma has occurred in the setting of sudden deceleration remains the most important factor in establishing the diagnosis of aortic injury. Despite the severe nature of the injury, one third of patients have no external evidence of thoracic trauma at the time of the initial physical examination. The clinical findings may be deceptively meager, but may include the following: acute onset of upper extremity hypertension, particularly suggestive if associated with excessive blood loss; presence of a harsh systolic murmur heard over the precordium or posterior interscapular area; and a difference in blood pressure between the upper and lower extremities. These findings, alone or in combination, occur in one third of patients with aortic rupture. Signs and symptoms of aortic rupture are dyspnea, chest pain, and back pain. On the physical examination, shock, steering wheel deformity, cardiac murmurs, hoarseness, palpable fractures of the sternum, or clavicles, and first rib should provoke suspicion of a thoracic vascular injury.

DIAGNOSIS

Although the chest x-ray may be normal in patients with traumatic rupture of the aorta, standard chest radiography continues to be an invaluable aid in diagnosing aortic rupture. Radiographic findings are suggestive, but not diagnostic, of significant intrathoracic vascular injury (Box 6-6). A supine chest radiograph can detect or rule out pneumothorax, hemothorax, and fractures. It has a negative predictive value of 95% to 98%. A chest radiograph can show sternal fractures, clavicle fractures, first rib fractures, obliteration of the aortic knob, depression of the left mainstem bronchus, loss of paravertebral pleural stripe, apical pleural cap, and lateral displacement of a trachea-widened mediastinum. A contrast CT is now an effective diagnostic screening tool for traumatic aortic injury; CT now approaches 100% specificity and sensitivity and is more cost-effective.[37]

Angiography is not recommended if mediastinal widening is present but the aortic contour is clearly defined and the nasogastric tube is not displaced. Angiography is recommended if mediastinal widening is present and the aortic contour is abnormal or obscured, or the nasogastric tube is displaced.[43]

Aortography remains the gold standard for the diagnosis of acute rupture of the aorta (Fig. 6-15). It has been found to be a highly sensitive and specific method of detecting aortic injuries. Additional advantages are that it can be performed safely and quickly in critically injured patients, it can be performed wherever the patient is being evaluated, and no nephrotoxic agents need be administered. Despite the severity of their injuries, transesophageal echocardiography can be carried out in victims of blunt chest trauma. It is important to remember that a negative transesophageal study does not exclude the presence of a ruptured aorta and should be followed by aortography in patients with an unexplained mediastinal hematoma. Aortography should be performed on any patient who has sustained a high-speed deceleration injury or blunt trauma, who has clinical findings suggesting aortic rupture, and who exhibits changes in serial chest radiographs. The following summarizes the indications for aortography in victims of blunt chest trauma:

1. History of deceleration injury
2. Any chest radiographic finding listed in Box 7-6
3. Massive hemothorax
4. Pulse deficits
5. Upper extremity hypertension
6. Systolic murmur
7. Unexplained hypotension

TREATMENT

Immediate repair of aortic injuries is recommended, but surgery still has a mortality rate of 30%. Preexisting cardiac disease increases mortality. Patients can be placed into two categories; hemodynamically stable patients can be afforded more preoperative time and workup and the mortality rate can approach 25%. In the unstable group, mortality increases to 90%. The likelihood of lethal secondary rupture of a false aneurysm mandates immediate surgical repair as soon as the diagnosis is established and the site of rupture is localized. Aortography should always precede thoracotomy and repair unless contraindicated by rapid deterioration of the patient's condition. In patients with aortic rupture, associated injuries such as rib fractures, flail chest, hemopneumothorax, extremity fractures, head injuries, and abdominal injuries occur more than 75% of the time. When combined injuries are present, the treatment of specific lesions must be prioritized. In patients with stable hemodynamics at the time of diagnosis, priority is given to repair of the aortic rupture. Abdominal injuries should be treated first in patients who have unstable hemodynamics but do not have hemothorax or an expanding mediastinal hematoma. Patients who are hemodynamically unstable and

BOX 6-6 Indications for Thoracotomy*

- Acute hemodynamic deterioration with cardiac arrest
- Cardiac tamponade
- Vascular injury at site thoracic outlet or great vessels
- Massive air leak from chest tube
- Tracheal or bronchial injury
- Esophageal injury
- Retained hemothorax
- Traumatic diaphragmatic hernia
- Traumatic cardiac valvular or aneurysmal lesions
- Traumatic pseudoaneurysms or aortic disruption
- Tracheoesophageal fistula

*Indications are based on physical findings, radiographic and echocardiographic imaging and, most importantly, the clinical course of the patient.



FIGURE 6-15 Aortograph used to diagnosis acute rupture of the aorta.

have an expanding mediastinal hematoma or hemothorax should undergo aortic repair, with subsequent abdominal evaluation. As noted earlier, of those who have sustained a traumatic rupture of the aorta, 10% to 20% reach the hospital alive because the adventitial or mediastinal pleura temporarily contains the aortic blood (Fig. 6-16). Patients should be managed with an antihypertensive regimen to decrease the risk of rupture. A systolic blood pressure of less than 100 mm Hg or mean arterial pressure (MAP) less than 80 mm Hg and heart rate (HR) less than 100 beats/min should be maintained. The use of beta blockers or vasodilators can help achieve these parameters. Repair with an interpositional graft is the treatment of choice.[44]

Innominate artery avulsion occasionally results from deceleration or crush injuries and, like aortic rupture, is frequently difficult to diagnose. Signs of distal ischemia are uncommon and diminution of radial or brachial pulses is present in only 50% of patients. Chest radiographic findings are comparable with those seen with descending aortic rupture, and aortography is necessary to establish the diagnosis. Treatment is immediate surgical repair because of the threat of exsanguination from secondary rupture of the pseudoaneurysm.

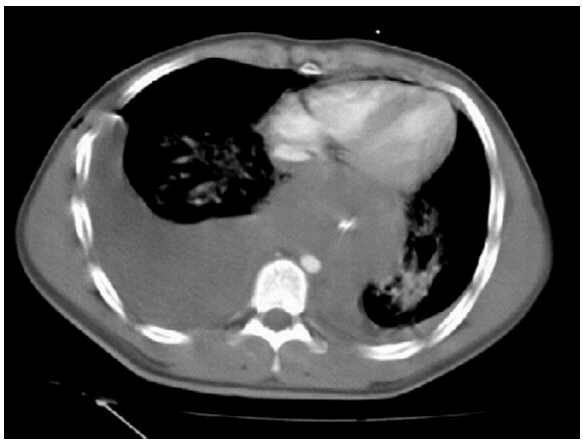


FIGURE 6-16 Example of an aortograph that shows a traumatic rupture of the aorta, where the aortic blood is contained temporarily by the adventitial or mediastinal pleura.

MYOCARDIAL CONTUSION

The exact incidence of myocardial contusion is difficult to determine because myocardial contusions are almost always well tolerated and because the clinical signs are

transient and difficult to recognize, may vary widely in severity, and may occur without external evidence of chest injury. It is estimated that myocardial contusion occurs in 30% of blunt trauma cases.[45] The most frequent cause of contusion is a steering wheel injury from sudden automobile deceleration. However, mechanisms of injury include direct pericardial impact, chest compression, increase in intrathoracic pressure, high-speed deceleration, or a combination of these. This injury has also been reported to occur from blows to the chest, falls from a great height, and upper abdominal trauma.

Commotio cordis is a phenomenon caused by sudden cardiac death resulting from a modest blow to the chest (e.g., softball) during the vulnerable phase of repolarization (just prior to the T wave) that leads to ventricular fibrillation and asystole. Only 13% of people survive.[45]

Myocardial contusion is often well tolerated; most patients will never suffer any symptoms and recover fully. Nevertheless, in some patients, life-threatening symptoms, such as ventricular arrhythmias and cardiac failure, will occur. Therefore, it is important to screen all patients with blunt thoracic trauma to identify those at risk for complications. There is a spectrum of myocardial injuries, ranging from myocardial stunning without myocyte damage to transmural necrosis and hemorrhage.[45] Symptoms can be a troponin rise to ventricular rupture. They may be synonymous with those of a myocardial infarction.

Myocardial contusion has a wide spectrum of symptoms. In patients with severe myocardial contusion, the diagnosis is not hard to determine. These patients will have hemodynamic instability. However, the symptoms might be caused by other factors in multitrauma patients. The diagnosis is more difficult in patients without clinical evidence of myocardial contusion. The patient may complain of chest pain or palpitations. These complaints are often attributed to chest wall contusion or fractures. Although most patients with myocardial contusion have chest wall lesions, the absence of the lesions does not exclude the presence of myocardial contusion.

In recent years, many studies have been done to find a diagnostic tool to identify patients with a myocardial contusion and those at risk to develop complications. Myocardial contusion cannot be diagnosed with a single test. Electrocardiography may help establish the diagnosis of myocardial contusion in patients with thoracic injuries, regardless of the presence or absence of symptoms. The incidence of electrocardiographic evidence of cardiac injury after blunt chest trauma varies from 18% to 38%. Although the electrocardiographic changes are frequently present when the patient arrives in the emergency department (or shortly thereafter), they may not appear until 24 to 72 hours later. Thus, serial electrocardiograms should always be obtained at 24-hour intervals. The most frequent electrocardiographic findings are ST-segment and T wave changes similar to those observed in myocardial ischemia and infarction. These electrocardiographic changes are usually reversible, but they may be observed for as long as 1 month following injury.

Cardiac dysrhythmias are another frequent finding stemming from myocardial contusion. A great variety of dysrhythmias and conduction disturbances have been reported. These include atrial fibrillation or flutter, supraventricular tachycardia, ventricular tachycardia, ventricular fibrillation, asystole, right and left bundle branch block, sinoatrial block, and complete heart block. Approximately 80% of dysrhythmias requiring treatment are demonstrable on the admission electrocardiogram.

Serum cardiac troponins—troponin I and troponin T—are highly specific to myocardial injury. They are not found in skeletal muscles and are released into the circulation only after loss of membrane integrity. Both troponin I and troponin T are important in diagnosing cardiac injury. Moreover, a normal concentration of cardiac troponin I or T is an indicator of the absence of myocardial injury.

To diagnose a myocardial contusion, the ideal moment of blood sampling has not been determined. However, if troponin I and T concentrations are within reference ranges on admission, a second measurement after 4 to 6 hours is necessary to exclude myocardial injury. Increased troponin I or T levels may persist for 4 to 6 days and can be used to evaluate patients days after the injury.

Echocardiography has been helpful in identifying structural abnormalities and pericardial effusions in patients who develop low cardiac output after blunt trauma. A screening strategy is used to identify patients at risk for cardiac complications in relation to the severity of associated injuries. The diagnosis of myocardial contusion remains difficult. Sybrandy et al[46] have suggested a screening strategy for detecting patients at risk for cardiac complications in relation to the severity of associated injuries (Fig. 6-17).

TREATMENT

Most patients with a cardiac contusion have a good prognosis, and those who die usually do so as a result of other injuries. The treatment for myocardial contusion is primarily supportive. Pharmacologic with inotropes or temporary transvenous cardiac pacing if third-degree atrioventricular (AV) block or high-degree AV block. The patient should be followed for up to 1 month. However, a decrease in cardiac output may develop in as many as 20% of patients with cardiac contusion. Therefore, and because of the potential electrical instability of the damaged area, general anesthetics should be avoided whenever possible in the period immediately following injury. If an operation is deemed necessary, the risk must be assumed. Procedures that are not urgent should be delayed until there is electrocardiographic evidence of complete healing or stabilization of the myocardial damage, a process that usually takes from 2 to 6 weeks.

Intraoperative monitoring of the central venous pressure or the pulmonary capillary wedge pressure with a Swan-Ganz catheter is of great value in these patients, because hypotension may be caused by low cardiac output rather than hypovolemia.

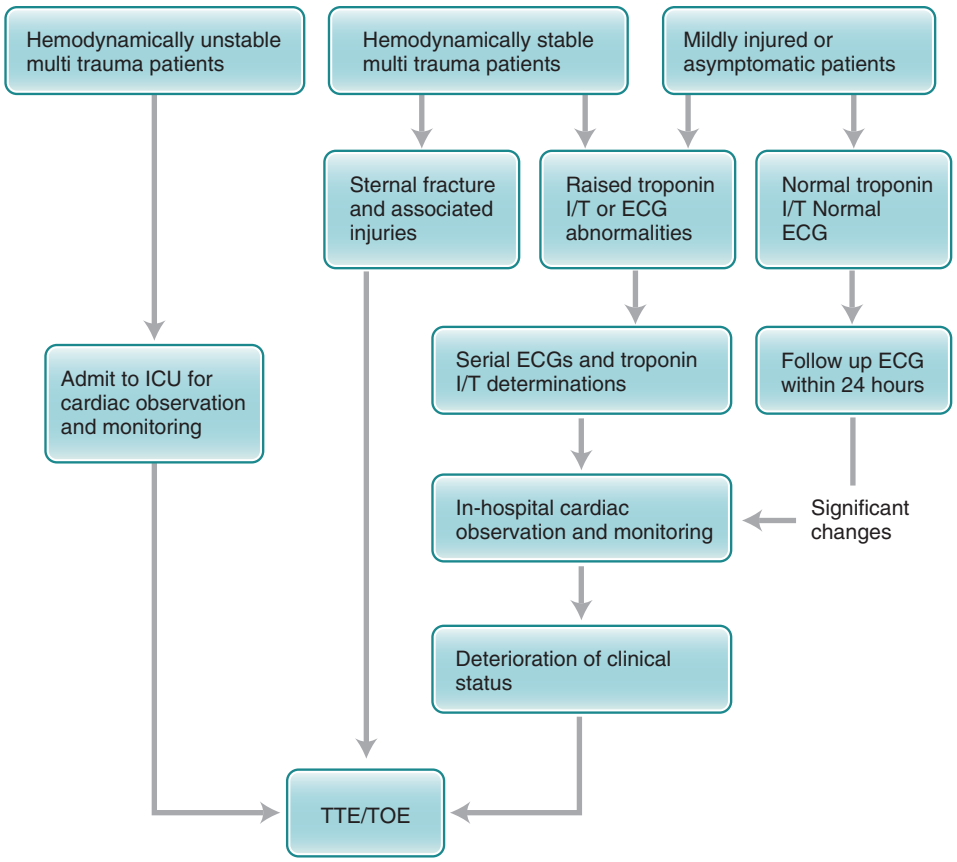


FIGURE 6-17 Screening strategy algorithm for detecting patients at risk for cardiac complications in relation to the severity of associated injuries. ICU, Intensive care unit; TOE, transesophageal echocardiography; TTE, transthoracic echocardiography. (Adapted from Sybrandy KC, Cramer MJ, Burgersdijk C: Diagnosing cardiac confusion: old wisdom and new insights. *Heart* 89:485, 2003.)

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CHAPTER

7

Recognition and Management of Shock

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OUTLINE

Cellular Changes
Systemic Response
Major Categories of Shock
Hypovolemic Shock
Cardiogenic Shock
Obstructive Shock
Distributive Shock
Treatment Principles

Shock occurs when the cardiovascular system fails to perfuse vital organs. There are numerous clinical events that cause shock, such as severe hemorrhage, trauma, burns, sepsis, anaphylaxis, myocardial infarction, and pulmonary embolism. Despite the cause, shock is defined and ultimately diagnosed by the clinical evidence of inadequate tissue oxygenation and cellular death. Once cell death occurs, inflammation, free radical formation and edema can exacerbate hypoperfusion, creating a viscous cycle of irreversible tissue damage that causes further cardiovascular collapse, despite aggressive treatment. Therefore, it is essential that shock be recognized and treated early to prevent end-organ failure and death of the patient.

Treatment of shock varies depending on the type of shock. There are four major categories of shock—hypovolemic shock (hemorrhagic versus nonhemorrhagic), cardiogenic shock, obstructive shock, and distributive shock (septic, anaphylactic and neurogenic).[1] Each category of shock may present with variable clinical signs and symptoms and requires different treatments. For example, aggressive volume replacement would be appropriate in a patient who is in hypovolemic shock, but may be detrimental in a patient with cardiogenic shock. Therefore, one must not only recognize shock but also be able to categorize it and implement the appropriate treatment based on the available clinical data. This chapter will give an overview of the cellular and systemic response to shock and then focus on its recognition, categorization, and current treatment philosophies.

CELLULAR CHANGES

Organ hypoperfusion creates a hypoxic environment that initiates cellular changes resulting in shock. At the cellular level, shock occurs when the delivery of oxygen is inadequate for cells to metabolize glucose for energy needed to run the cellular machinery. Cells depend on oxygen to carry out aerobic respiration via the mitochondria. Mitochondria are the power plants of cells that supply 36 adenosine triphosphate (ATP) molecules through the oxidation of each molecule of glucose. Without an adequate supply of oxygen, mitochondria switch to anaerobic respiration, yielding only two ATP molecules and lactate per glucose molecule. Lactate lowers the tissue pH and is released into the systemic system, becoming a useful marker for tissue hypoxia and acidosis. Not only is lactate toxic to cells, but a lack of ATP generation leads to free radical formation and damage to vital cell functions, most notably ATP-dependent ion channels that maintain normal membrane potentials. When ATP-dependent Na^+, K^+, and Ca^{2+} transport are disrupted, cellular membranes lose their electrical gradient, leading to intracellular bleb formation, cellular edema, and cell lysis. The tipping point for irreversible cell damage is not entirely understood but is thought to be a result of uncontrolled release of intracellular Ca^{2+} stores. Intracellular bleb formation and mitochondrial swelling are signs of irreversible cellular dysfunction that leads to cell lysis. When intracellular enzymes and cellular material are released into the surrounding tissue and circulation, further inflammation, edema, and tissue damage occur.[2,3]

Endothelium normally produces nitric oxide but when damaged by inflammation, nitric oxide synthase is overexpressed, producing toxic levels of nitric oxide and other oxygen-derived free radicals that are formed when tissue are reperfused with oxygen. This reperfusion injury creates free radicals and further activation of inflammatory mediators, notably interleukin-8 (IL-8), IL-1, and tumor necrosis factor (TNF). The combination of ATP depletion, tissue edema, pH changes, free radical formation, and inflammatory mediator formation leads to a complex vicious cycle of cell death and irreversible organ damage.[3]

SYSTEMIC RESPONSE

Different organ cells have extremely varying cellular oxygen demands. Two of the most metabolically active and oxygen-demanding tissues are the heart and brain. This is clearly evident in the systemic response to hypoperfusion. During shock, the cardiovascular system diverts blood flow from less vital structures to maintain

adequate perfusion to the heart and brain at the expense of tissues such as skin, skeletal muscle, gastrointestinal (GI) tract, and kidneys. This diversion is made possible by differences in local and distant regulatory mechanisms. For example, the heart and brain at rest already exhibit a high sympathetic vascular tone and thus tightly autoregulate perfusion through local metabolites. In contrast, splanchnic blood flow is normally in excess of metabolic demand and its vasculature is influenced much more by increases or decreases in sympathetic tone. During prolonged shock, sympathetic-mediated vasoconstriction in the splanchnic vasculature leads to ischemia and necrosis, but the brain and cardiac blood flow are preserved because of metabolic autoregulation.

Cardiac output (CO) is the amount of blood pumped from each ventricle each minute. It can be represented by the following equation:

$$\text{CO} = \text{stroke volume (SV)} \times \text{heart rate (HR)}$$

or

$$\text{CO} = \text{volume of blood pumped by the heartbeat} \times \text{beats/min}$$

It is continuously regulated to meet tissue perfusion demands. For example, during strenuous exercise, CO will increase from 5.8 to 15 liters/min to meet the requirements of exercising skeletal muscle. CO is dependent on three variables:

1. Preload
2. Afterload
3. Myocardial contractility

Preload is the amount of blood that returns to the heart prior to contraction, the end-diastolic volume (EDV). Increasing preload is often referred to as priming the pump. More blood returning to the ventricles prior to systolic contraction not only increases the amount of blood ejected per heartbeat, but also increases the force of cardiac contractility as indicated by the Frank-Starling law of the heart. The result is an increase in the stroke volume and cardiac output.

Preload is determined by the pressure difference between the central venous system (great veins of the thorax and abdomen) and peripheral venous system. Combined, the venous system holds 60% to 70% of the circulating blood volume; most of this is stored in the peripheral venous pool of organs such as the skin, skeletal muscle, and splenic system. Veins are compliant because of their thin walls, allowing them to collapse or distend with small changes in pressure. This compliance enables the venous system to act as a reservoir of circulating blood volume, most of which is in the peripheral veins and does not contribute to the central venous pressure. During hypovolemia, volume loss is reflected in a loss in this venous reservoir as blood moves from the periphery into the central circulation. Also, an increase in circulating catecholamines—norepinephrine and epinephrine—causes further venous constriction. This venous compliance allows a net shift of blood from the peripheral venous pool to the central venous system, thus maintaining adequate blood return to the ventricles.

Afterload is the pressure or resistance to the forward movement of blood being pumped from the heart. As the heart contracts, it must overcome the forward pressure to propel blood through the circulatory system. Myocardial contractility is the force of the pump. A more forceful contraction allows for a greater percentage of blood entering the heart to be ejected into the arterial system. The ratio of blood entering and leaving the heart is represented by the ejection fraction (EF); EF = SV/EDV, which is normally 55% to 80% (mean, 67%). An EF of less than 55% indicates depressed myocardial contractility.

When mean arterial pressure falls below 60 mm Hg, tissue perfusion is compromised and shock ensues. Hypotension and hypoxia are sensed by chemoreceptors and baroreceptors, resulting in reduced vagal tone and the release of catecholamines (norepinephrine and epinephrine) by the neuroendocrine system. This causes an increase in heart rate, cardiac contractility, vascular tone, and total peripheral resistance to maintain blood flow to vital organs. It is the constriction of arterioles that decreases blood flow to the peripheral organs and increases the total peripheral resistance. Blood flow to the brain and heart are less affected by distant autonomic regulation and rely more on local control mechanisms, such as oxygen tension and metabolic byproducts. This allows for preservation of blood flow, despite significant reductions in cardiac output. In contrast, splanchnic blood flow is normally in excess of metabolic demands and is controlled more by circulating catecholamines. In times of shock, these organs can experience severe arteriolar constriction and early ischemia.

Vasoconstriction is vital for maintaining adequate perfusion and diverting blood flow in times of shock but, without volume replacement, prolonged vasoconstriction will lead to ischemia. The body can increase volume through mobilizing extravascular fluid into the circulation and by decreasing renal excretion.

Renin is released because of increased sympathetic stimulation and a decrease in blood flow sensed by the juxtaglomerular apparatus in the kidneys. Renin stimulates the formation of angiotensin I, which is converted to angiotensin II in the lungs. Angiotensin II is a potent vasoconstrictor. It also stimulates the release of aldosterone by the adrenal cortex and vasopressin, also known as antidiuretic hormone (ADH), by the posterior pituitary. Aldosterone expands the intravascular volume by increasing Na^+ retention in the distal convoluted tubules and collecting ducts, and vasopressin retains water by increasing aquaporin channels in the collecting ducts. Overall, this causes a reduction in urinary output that can be measured to follow fluid status and resuscitation efforts. Generally, renal output of less than 0.5 mL/kg/hr in adults and 1 mL/kg/hr in children signifies renal hypoperfusion.

Not only does reducing the renal excretion of Na^+ and water increase the intravascular volume, but the body is able to regulate changes in transcapillary fluid movement to improve intravascular volume. This occurs with increases in capillary oncotic pressure and a decrease in hydrostatic pressure. When arterioles constrict, blood flow and hydrostatic pressure through capillary beds is

TABLE 7-1 Signs of Shock by Category

Signs	Cardiogenic	Hypovolemic	Obstructive	Neurogenic	Septic
Blood pressure	↓	↓	↓	↓	↓
Jugular vein	Distended	Nondistended	Distended	Nondistended	Nondistended
Urine output	↓	↓	↓	↓	↓
Peripheral temperature	↓	↓	↓	Normal to ↑	↑
Cardiac index	↓	↓	↓	Variable	↑
CVP	↑	↓	↑	↓	↓
PAWP	↑	↓	↑	↓	↓
SVR	↑	↓	↑	↓	↓

greatly reduced, causing a net shift of fluid into the vascular space. Also, larger molecules are unable to cross capillary fenestrations creating an oncotic pressure that drives extravascular fluid into capillaries. This oncotic pressure is magnified when catecholamines stimulate glycogenolysis and gluconeogenesis, causing an increase in the blood glucose concentration. These compensatory mechanisms are limited, and definitive treatment must be initiated quickly and will vary depending on the type and severity of shock.[4]

MAJOR CATEGORIES OF SHOCK

These are shown in Table 7-1.

HYPOVOLEMIC SHOCK

The most commonly observed form of shock, hypovolemic shock, results from a rapid loss of intravascular volume; this can be further subdivided into hemorrhagic and nonhemorrhagic types (Box 7-1). Trauma and/or GI bleeding are the most common sources of hemorrhagic shock, and exsanguination is responsible for 80% of deaths in the operating room and almost 50% of deaths in the first 24 hours after injury.[5] Retroperitoneal accumulation is always a diagnostic challenge and the presence or amount of blood loss is frequently underestimated. Nonhemorrhagic shock results from increased noncompensated fluid loss (e.g., dehydration, vomiting, diarrhea, polyuria, fluid redistribution), as seen in burns and anaphylaxis.

Hemorrhage decreases intravascular volume. The average blood volume in a 70-kg person is approximately 5 liters (0.07 × weight in kilograms). The physiologic response is directly proportional to the volume of blood that is lost and can be classified by the percentage exsanguinated. Class I hemorrhage is a blood volume loss up to 15%. In uncomplicated situations, there are minimal clinical signs. Patients with class I hemorrhage normally compensate by baroreceptor-mediated reflex vasoconstriction, with a concurrent increased level of catecholamines from baroreceptor stimulation and increased renin levels from the juxtaglomerular system. Intravascular volume is regenerated by osmotic shifts from the extravascular fluid and through fluid absorption from the GI tract. Vascular resistance increases secondary to the vasoconstriction.

BOX 7-1 Classification of Hypovolemic Shock

HEMORRHAGIC
Trauma
Gastrointestinal bleeding

NONHEMORRHAGIC
External fluid loss
- Diarrhea
- Vomiting
- Polyurea

Fluid redistribution
- Burns
- Anaphylaxis

Class II hemorrhage is a blood volume loss between 15% and 30%. Clinical symptoms include tachycardia, tachypnea, and a decrease in pulse pressure. These patients are normally stabilized and usually respond to expansion of the intravascular volume with crystalloids, but some patients may require blood transfusion.[1] Patients with class II blood loss can usually sustain normal pressures while supine but may be orthostatically dependent.[6]

Class III hemorrhage is a blood volume loss between 30% and 40%. These patients will have obvious signs of inadequate perfusion, including significant tachycardia and tachypnea, and are in need of blood products. In class III blood loss, patients are at great risk if resuscitation with restoration of intravascular volume is not performed.

Class IV hemorrhage is a blood volume loss of more than 40% and is an imminent life-threatening situation. Symptoms include a significant depression in systolic blood pressure and a very narrow pulse pressure. Urinary output is negligible and mental status is depressed.[1] These patients exhibit an impaired level of consciousness and will experience a precipitous death if intravascular volume is not quickly restored (Table 7-2).

Tachycardia by itself should never be considered a pathognomonic sign in hypovolemic patients, because it is not a reliable indicator of the presence or severity of shock.[6] Many hypovolemic patients will initially

TABLE 7-2 Pediatric Signs and Symptoms of Hemorrhagic Shock

System	Class I	Class II	Class III	Class IV
CV				
Heart rate	Normal or slow inc.	+	++	+++
Blood pressure	Normal	Normal or slow	—	— — —
Pulse	Normal	↓ peripheral	Thready peripheral	Thready central
Respiratory	Normal	+	++	+++
CNS	Normal/anxious	Confused	Lethargic	Coma
Skin	Warm	Cool	Cool, pallor	Cold, cyanosis
	Capillary refill	Delayed	Prolonged	
Renal	Normal	Oliguria, ↑ SG	Oliguria, ↑ BUN	Anuria

BUN, blood urea nitrogen; *CV,* cardiovascular; *CNS,* central nervous system; *SG,* specific gravity.
Modified from ACS: ATLS course, ed 4, Chicago, 1992, ACS

have symptoms of bradycardia. Also, older patients and patients on long-term beta blocker may not be able to increase their heart rate appropriately. Conversely, patients who are in pain and have minimal blood may be tachycardic.[7]

CARDIOGENIC SHOCK

Cardiogenic shock is an impairment of the cardiac pump to maintain cardiac output despite the presence of adequate intravascular volume and tone. It can be caused by intrinsic damage or a conduction disorder resulting in peripheral hypoperfusion, cyanosis, oliguria, and altered central nervous system function. On physical examination, patients in cardiogenic shock can appear ashen and cyanotic, with evidence of confusion or agitation from decreased cerebral perfusion. Extremities vasoconstrict and become cool and clammy to the touch. Pulses are usually rapid and faint and arrhythmias are common. The pulse pressure is narrowed and there are generally distant or muffled heart sounds on auscultation. Also, summation gallops may be auscultated because of third and fourth heart sounds. Systolic hypotension of less than 90 mm Hg or a decrease in systolic blood pressure from baseline by more than 30 mm Hg is usually noted. Other hemodynamic manifestations are elevated left ventricular filling pressures greater than 15 mm Hg and a reduction in cardiac index (cardiac output divided by the body surface area) to less than approximately 2.2 liters/min/m^2.

There are many causes of cardiogenic shock (Box 7-2). The classic presentation is heart failure caused by extensive left ventricular damage—that is, ischemia to 40% or more of the left ventricle during a massive infarction. Most patients with cardiogenic shock (≈75%) have left ventricular failure, with right heart failure occurring in only 3% of patients.[8] A differential diagnosis of cardiogenic shock should also consider the mechanical effects of papillary muscle rupture, aneurysm of the left ventricular wall, ventricular septal defect, and aortic stenosis, which account for the other 12% of cases. Cardiac arrhythmias, such as atrial fibrillation with a rapid ventricular response or ventricular tachycardia, also may contribute to hypotensive shock. Shock can also occur in patients under treatment for a presumptive diagnosis of myocardial infarction (MI) secondary to medications such as nitrates, or from hemorrhage caused by anticoagulant or thrombolytic drugs. The physiologic response can cause a worsening of cardiogenic shock because of the perceived reduction in volume and perfusion by the body, stimulating vasoconstriction and salt and water retention through the renin-angiotensin system. This response increases afterload and worsens fluid overload, causing pulmonary edema. Patients at greatest risk for cardiogenic shock are those with diabetes, older patients, history of prior MI, female gender, peripheral vascular disease, angina, and EF less than 35%.

BOX 7-2 Causes of Cardiogenic Shock

Extensive left ventricular damage
Papillary muscle rupture
Aneurysm of the left ventricular wall
Ventricular septal defect
Aortic stenosis
Cardiac arrhythmias

OBSTRUCTIVE SHOCK

Obstructive shock occurs as a result of an extracardiac blockage of the cardiopulmonary system. The most common cause of obstructive shock is massive pulmonary embolism. Pulmonary emboli generate circulatory shock by obstruction of the pulmonary vessels, causing a selective right ventricular ischemia, which leads to an increase in right cardiac afterload.[9] Ensuing hypoxemia, coronary hypoperfusion, and systemic acidosis have a catastrophic effect on cardiac function, which eventually results in biventricular dysfunction and cardiac failure.[10] Other less common causes of obstructive shock are tumors, pericardial tamponade, pericarditis, tension pneumothorax, and primary pulmonary hypertension.

DISTRIBUTIVE SHOCK

Distributive shock is caused by profound circulatory venodilation that results in severe abnormal fluid redistribution. The most common causes of distributive

shock are sepsis, anaphylaxis, and central neurogenic hypotension.

Septic Shock

Septic shock is hypotension caused by a generalized activation of the immune system by a suspected or culture-proven infection. It can be produced by infection with any microbe, although gram-negative aerobic bacteria are most often implicated. Lipopolysaccharide (LPS), a component of the gram-negative cell wall, has been found to be the primary causative agent of septic shock. LPS consists of a fatty acid lipid A core and a polysaccharide coat. Similar proteins have been isolated in gram-positive bacterial walls and exotoxins (i.e., *Staphylococcus*-induced toxic shock), and fungus. LPS binds in combination with circulatory blood proteins to CD14 molecules on leukocytes, endothelial cells, and other cell types. The mononuclear phagocytes respond to LPS by producing TNF, which in turn induces IL-1 synthesis. TNF and IL-1 both act on endothelial cells to produce other cytokines, such as IL-6 and IL-8. Increasing concentrations of LPS induce nitric oxide and platelet-activating factor that can produce profound vasodilation. IL-1 also increases the temperature set point in the hypothalamus, increasing core body temperature.

Hypovolemia, cardiovascular depression, and systemic inflammation must all be managed during the resuscitative efforts of a patient in sepsis. Increased venous capacitance and an absolute hypovolemia secondary to capillary leakage lead to a relative loss of fluid into the extravascular tissue. Peripheral vasodilation of vessels to the skin and skeletal muscle shunts blood away from the central circulatory system, causing a decrease in perfusion to the brain, heart, bowel, liver, and kidney.

Mortality rates from septic shock exceed 50% and have two different presentations. The first type of mortality is the short course. Short-course patients die within hours of the onset of sepsis because of overwhelming refractory vasodilation. The second type has a longer lingering course, which has symptoms of a clinical pattern of severe hypotension, lactic acidosis, and vasoconstriction.

Anaphylactic Shock

Anaphylactic shock is the result of vasodilation secondary to a massive histamine release from an immunoglobulin E (IgE)–mediated systemic response to an allergen. Cardiovascular collapse occurs as a result of vasodilation capillary leakage, leading to intravascular volume depletion.[11] Treatment goals are to slow or reverse this pathophysiologic process. Epinephrine and diphenhydramine hydrochloride should be administered during anaphylactic shock. All patients should receive supplemental oxygen, isotonic fluid infusion, and continuous cardiac monitoring. A large volume of crystalloid fluid may be necessary to reverse the vasodilatory hypotension that is associated with anaphylaxis.

Central Neurogenic Hypotension

Central neurogenic hypotension occurs transiently from vagus-induced venodilation or secondary to spinal cord injury, which results in a venodilatory spasm. In spinal cord lesions rostral to T1, cardiac vagal influences dominate and bradycardia is present. This venodilation causes a decrease in preload. Blood pressure is initially maintained by expanding intravascular volume. Poor perfusion may result in a larger ischemia in the damaged area. For this reason, α_1-adrenergic specific therapy should be initiated for peripheral vasoconstriction and correction of hypotension.

TREATMENT PRINCIPLES

Once shock is recognized, treatment to restore adequate perfusion should be instituted without delay (Fig. 7-1). In the trauma patient, lifesaving measures should first be directed according to Advanced Cardiovascular Life Support (ACLS) and Advanced Trauma Life Support (ATLS) protocols by assessing and management of the ABCDEs (see Chapter 5). In a hemorrhaging patient, management of obvious massive bleeding is put before airway management, especially in the prehospital or battlefield setting. Bleeding can usually be controlled by direct pressure, a tourniquet around extremities, or a pneumatic antishock garment (PASG) if bleeding is from pelvic and lower extremity fractures. If massive hemorrhaging in not stopped soon, patients will exsanguinate and die before an airway can be secured. Most often, trauma response is a team approach, with multiple areas of treatment being orchestrated together. Patients in shock should have an established airway with supplemental oxygen to maintain oxygen saturation above 95%. Breathing in shock can be rapid and shallow, a response that uses negative intrathoracic pressure to act as a central venous pump to increase venous return and preload.

Vascular access should be attained with two large-bore IVs (16-gauge or larger) to allow for rapid transfusion. Poiseuille's law (Fig. 7-2) demonstrates that flow rate is proportional to the radius of the capillary by the fourth power. Central lines are inadequate for rapid transfusion because of the flow rate being inversely proportional to the length. Infusion pumps or an inflated blood pressure cuff around the solution can speed infusion by increasing the pressure difference. Fluid warmers are used to prevent coagulopathy caused by hypothermia and increase viscosity. Vascular access can be challenging because of vein collapse. Therefore, other measures such as an intraosseous or venous cutdown may be employed.

Once vascular access is obtained, patients may receive a fluid challenge of 1 to 2 liters of an isotonic solution (lactated Ringer's or normal saline solution). The patient's response to this challenge is carefully monitored for signs of improvement. In trauma patients, hypovolemic shock caused by hemorrhage is the rule unless there is an obvious cause for cardiogenic shock, such as isolated trauma above the torso. If shock persists following primary fluid therapy, the exact cause (i.e., hypovolemic versus cardiogenic shock) must be ascertained.[1]

Differentiating shock and determining the patient's fluid status rapidly is sometimes difficult. Central hemodynamic monitoring can be performed with ultrasonog-

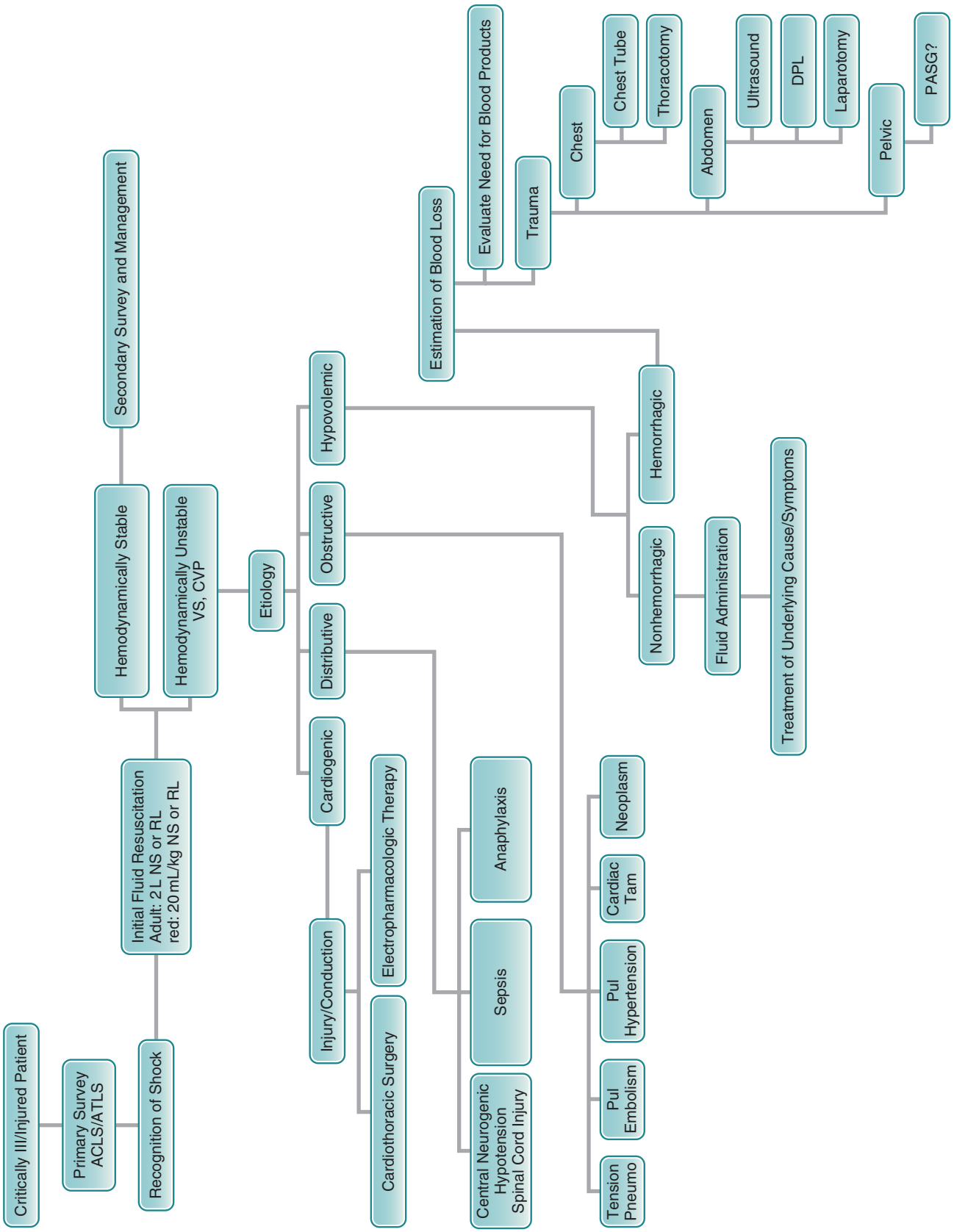


FIGURE 7-1 Diagnostic and management algorithm for shock.

raphy or catheters (Fig. 7-3) in the pulmonary artery to provide end points during fluid resuscitation. The pulmonary artery wedge pressure (PAWP) and central venous pressure (CVP) are interpreted as indices of the end-diastolic pressure in the left and right ventricles. When PAWP and CVP pressures are less than 6 mm Hg (normal, 4 to 12 and 6 to 12 mm Hg, respectively), most patients in shock will benefit from fluid infusions administered as repeated boluses equivalent to 1% to 2% of body weight. Care must be exercised, because distributive shock patients can manifest severely decreased arterial pressure despite exponential increases in their cardiac output.[12]

Recent technologic advances and training in ultrasonography allow for more rapid and less invasive assessment of cardiac function and volume status in critical settings compared with pulmonary arterial catheters. Table 7-3 compares the advantages of each. Transesophageal echocardiography (TEE) is more invasive (complication rate of 0.5%) than noninvasive transthoracic echocardiography (TTE). However, TEE is rarely indicated in an acute setting unless TTE is nondiagnostic. Both can yield valuable information, such as left ventricular filling, EF, CO, diastolic dysfunction, valvular abnormalities, and vena cava distensibility (inferior vena cava [SVC] TEE and inferior vena cava [IVC] TTE).[13]

In trauma, a FAST examination (***f***ocused ***a***ssessment with ***s***onography in ***t***rauma) is a rapid way to locate bleeding in the pericardial, pleural, or intraperitoneal space. Patients with cardiac tamponade exhibit Beck's triad of decreased arterial pressure, muffled heart sounds, and distended neck veins. Tamponade can be distinguished from tension pneumothorax, which also presents with distended neck veins, but tension pneumothorax presents with absent breath sounds and hyperresonance to percussion. These findings may be difficult to ascertained in a noisy trauma bay. If a tension pneumothorax is suspected and the patient is unstable, treatment should not be delayed to obtain a chest radiograph. Immediate chest tube placement or needle decompression with a large-bore angiocatheter below the second rib in the midclavicular line is a temporizing measure until a chest tube can be placed. In severe intrathoracic hemorrhage, thoracotomy should be performed emergently. In multisystem trauma, if abdominal hemorrhage is suspected, a diagnostic peritoneal lavage or abdominal ultrasound should be performed, depending on operator preferences and equipment available.

$$F\alpha \frac{\Delta P \times r^4}{\eta \times L}$$

F (flow) = Δ*P* (pressure) multiplied by *r* (radius)^4 divided by blood viscosity multiplied by *L* (vessel length).

FIGURE 7-2 Poiseuille's law demonstrates that flow rate is proportional to cannula radius by the fourth power.

In the setting of volume loss, expanding the intravascular volume is critical for maintaining cardiac output. However, what, when, and how much to give is a controversial topic. As mentioned, a trial of 1 to 2 liters of IV crystalloid may help assess the amount of blood loss based on the patient's response and may be the only volume expander rapidly available in the field. However, studies have shown that patients with class III or IV hemorrhage will need blood replacement; giving IV crystalloid transfusions may further increase inflammation and tissue edema and decrease coagulation, leading to worsening shock and increased mortality rates.[14] One alternative is hypertonic saline (HTS). HTS reduces the transfusion volume needed and can modulate or decrease the inflammatory response associated with crystalloid infusions. However, the ability of HTS to improve survival outcomes is inconclusive in trauma resuscitation.[15]

Unlike crystalloids, giving blood will increase the oxygen-carrying capacity and provide clotting factor. Unfortunately, component therapy is currently the best treatment available to reverse massive blood loss. Blood products are taken from donors, separated into various components, and then stored for up to 40 days for packed red blood cells (PRBCs), 30 days for fresh-frozen plasma

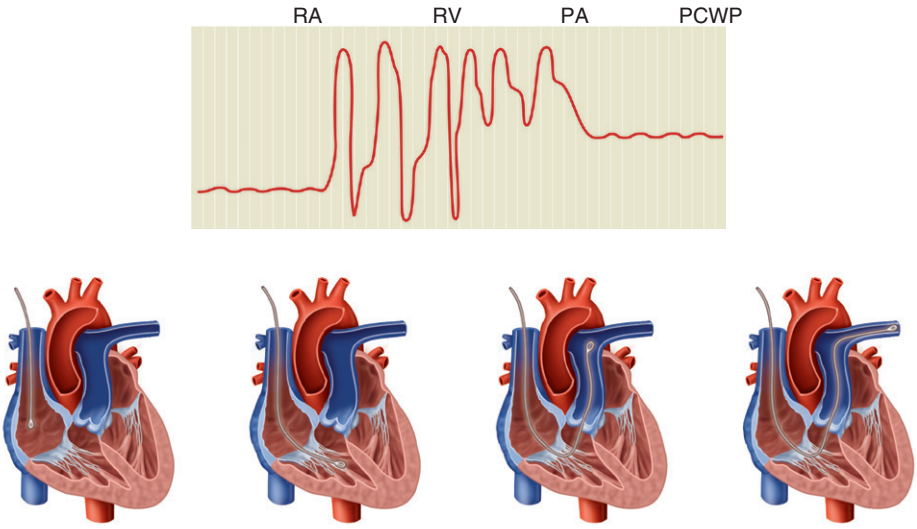


FIGURE 7-3 Central hemodynamic monitoring catheter placement for central venous pressure and pulmonary wedge pressure. (From Lederman RJ, Winshall JS: *Tarascon internal medicine and critical care*, ed 2, 2000, Tarascon Press.)

(FFP), and 5 days for platelets. All products have a shelf life and, over time, they slowly lose efficacy. Preservatives in stored blood and the length of time blood is stored may have direct effects on calcium and potassium levels, respectively. The affinity of hemoglobin for oxygen may increase if levels of 2,3-diphosphoglycerate decrease. In the situation of massive transfusion, adjuvant therapy to correct coagulopathy and electrolyte imbalance is often required (Table 7-4).[16] Depending on institutional transfusion protocols and blood bank availability, older blood products closer to expiration are generally given first. Multicenter randomized clinical trials are underway to determine whether shelf life affects blood component efficacy,[17] but cohort and observational studies have indicated a possible detrimental clinical effect associated with the transfusion of stored red blood cells.[18,19]

Recent data from multicenter trials and the wars in Iraq and Afghanistan have shown a benefit in replicating what is lost—whole blood. PRBCs alone lack clotting factors and, by giving more platelets and plasma upfront with PRBCs, one can limit further bleeding and improve survival. Also, blood replacement by major trauma centers is more effective if they have a massive transfusion protocol (MTP) in place. This includes limiting crystalloid infusion, reducing time for infusion, and establishing predetermined blood component replacement ratios (plasma-to-platelets-to-PRBCs). Fortunately, less than 5% of civilian trauma patients will require massive transfusion (10 or more units of PRBCs in the first 24 hours). However, this 5% account for 75% of the blood utilization in busy urban trauma centers.[5]

Although not yet adopted in the current ATLS protocol, research and trends in trauma resuscitation are providing evidence to support hypotensive resuscitation—target mean arterial pressure (MAP) of 50 mm Hg rather than 65 mm Hg—until active bleeding stops. A prospective, randomized controlled trial of 90 patients by Morrison et al has demonstrated that hypotensive resuscitation can decrease postoperative coagulopathy and lower the risk of early postoperative death while reducing the amount of blood product transfusions and overall IV fluid administration.[20,21] Other adjuvants that can limit transfusion amounts and coagulopathy associated with trauma are the use of autotransfusion devices (e.g., cell saver) and recombinant factor VIIa.[5]

The treatment of cardiogenic or obstructive shock is management of the underlying cause. In tension pneumothorax, immediate treatment by needle decompression or chest tube placement is essential. In myocardial contusion, proper pharmacologic support should be initiated to restore hemodynamic stability. Patients with cardiogenic shock secondary to arrhythmia are managed using ACLS protocols. Patients with pericardial tamponade should have a pericardiocentesis. If shock persists, thoracotomy should be initiated for definitive management. In cardiogenic shock (i.e., failure secondary to MI), right-sided heart catheterization is helpful for diagnosis and treatment followed by emergent placement of an intra-aortic balloon pump or left ventricular assist device (LVAD).[22] Revascularization with mergent percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass grafting (CABG) appears to provide early and longer term survival in eligible patients.[23]

Dopamine, dobutamine, and epinephrine are inotropic drugs that in clinical trials have proven to be beneficial for the hemodynamic support of patients in septic

TABLE 7-3 Advantages of Echocardiography and Pulmonary Arterial Catheters

Advantages of Echocardiography	Advantages of PAC
Cardiac chamber volumes	Pressures
LV ejection fraction	SVO_2
LV diastolic function	Continuous monitoring
Pericardial space assessment	PAOP
Valvular dysfunction	CO measures
Noninvasive CO measures	Not operator-dependent

CO, Cardiac output; *LV,* left ventricle; *PAC,* pulmonary artery catherter; *PAOP,* pulmonary artery occlusion pressure; *SVO_2,* mixed venous oxygen saturation. From Salem R, Valle F, Rusca M, et al: Hemodynamic monitoring by echocardiography in the ICU: the role of the new echo techniques. Curr Opin Crit Care 14:561, 2008.

TABLE 7-4 Blood Products

Product	Indications	Dose
Packed red blood cells (PRBCs)	Class III heme Class IV heme Hgb < 10	1 unit increases hematocrit by 3%
Platelets	Thrombocytopenia Platelets less than 50,000	1 platelet concentration increases platelet count by 5,000-10,000
Fresh-frozen plasma	Deficiency of factor concentration PT/PTT > 1.5 normal	10-15 mL/kg increases to 30% of normal plasma factor concentrations reversal of warfarin therapy for which 5-8 mL/kg
Cryoprecipitate	Hemophilia A, von Willebrand disease, factor XIII deficiency, microvascular bleeding in transfused patients with fibrinogen concentration < 80-100 mg/dL	1 unit /10 kg raises plasma fibrinogen concentration by ≈50 mg/dL

PT, Prothrombin time; *PTT,* partial thromboplastin time.

shock.[24] Dopamine effects are dose-dependent. At low dosages (1 to 10 μg/kg/min), dopamine is an agonist for β_1-adrenergic receptors, which increase myocardial contractility and renal blood flow. At dosages higher than 10 μg/kg/min, α_1-adrenergic receptors become increasingly occupied, resulting in vasoconstriction. Dobutamine is predominantly a β_1-adrenergic receptor agonist, with less stimulation of β_2-adrenergic receptors. Adverse myocardial effects of dobutamine include heart rates more than 130 beats/min, ischemic changes on electrocardiography, and tachyarrhythmias. Care must be exercised because dobutamine can also stimulate β_2-adrenergic receptors, which may further vasodilate patients with vascular tone already decreased by inflammatory mediators. Epinephrine provides a combination of α_1- and α_2-adrenergic effects, and also stimulates β_1- and β_2-adrenergic receptors, increasing systemic vascular resistance (SVR) and CO simultaneously.

In the management of septic shock, antibiotics play an essential role, although consideration to the large amount of LPS that is released during cell death must be accounted for. The release of LPS can cause a severe reduction in SVR, with a resulting precipitous hypotension. SVR is defined as resistance against which the left ventricle must work to eject its stroke volume and is normally 900 to 1300 dyne/sec/cm^5. Patients in septic shock often have a calculated SVR of less than 1000 dyne/sec/cm^5 associated with a decline in mean arterial pressure below 60 mm Hg and critical organ dysfunction.[25] Phenylephrine and norepinephrine are effective agents in the treatment of distributive shock by constricting vessels in the skin and skeletal muscle.

Phenylephrine is a nonselective α-adrenergic agent that causes vasoconstriction but does not have direct cardiac stimulatory effects. Phenylephrine is useful in the specific case of septic shock in patients at high risk for cardiac arrhythmias. Norepinephrine has combined alpha and beta effects, and extensive use of this drug in patients with septic shock has been well documented.[26] The refractory vasodilation of septic shock can be reversed by norepinephrine, which rescues the underperfused renal circulation and restores renal function in patients with septic shock.[27]

Because accumulation of lactic acid occurs during shock, metabolic and/or respiratory acidosis must be recognized and managed. Lactic acidosis is defined as a lactate concentration of 0.5 mmol/liter and a pH of 7.35. Bicarbonate therapy has been advocated for the treatment of metabolic acidosis, but there is lack of clinical evidence that it improves oxygenation, tissue perfusion, or overall survival, and its use in patients with lactic acidosis is not advocated.[28] Respiratory acidosis is managed by ventilation and is based on the presence of physical signs and depth of acidosis. P_{CO_2} is measured to calculate the relationship between carbon dioxide production and elimination. Respiratory rates are then adjusted to regulate P_{CO_2} levels.

SUMMARY

Regardless of the category, severity, or multiple causative factors, shock is the result of inadequate oxygen delivery to cells for oxidative metabolism. Through numerous compensatory mechanisms, the body attempts to preserve CO and maintain blood flow to vital organs such as the heart and brain. However, as shock progresses, a positive feedback of cell death, acidosis, inflammation, and coagulopathy will further worsen tissue perfusion and ultimately become irreversible and fatal. Treatment should be initiated first to stabilize the patient; then clinical examination, diagnostic tests, and continuous monitoring are necessary to help identify the underlying cause. Ultimately, the definitive treatment can be established.

ACKNOWLEDGMENT

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CHAPTER

8

Neurologic Evaluation and Management

Michael Gladwell | Ivan J. Sosa | Kevin Arce

OUTLINE

Initial Assessment
Resuscitation: Initial Trauma Management
History
Evaluation
Mental Status
Cranial Nerve Examination
Grading the Severity of Injury
Anatomic Signs
Functional Signs
Mayo Head Injury Classification System for Traumatic Brain Injury
Diagnostic Studies in Head Injury
Management of Head Injury
Vascular Injury
Superior Orbital Fissure Syndrome
Skull Fractures
Epidural Hematoma
Subdural Hematoma
Diffuse Axonal Injury
Cerebral Contusions
Cervical Spine Injury
Occipital Condyle Fractures
Atlanto-Occipital Dislocation
Atlas Fractures
Axis Fractures
Lower Cervical Spine Fractures

According to the Centers for Disease Control and Prevention (CDC), one unintentional injury kills more people between the ages of 1 and 44 years than any other disease or illness. More than 180,000 deaths occur from injury each year, with one person dying every 3 minutes. It also leads to more than $406 billion annually in medical costs and lost productivity. In the United States, approximately 1.7 million traumatic brain injures (TBIs) occur each year. Of these, 1,365,000 (80.7%) were emergency department (ED) visits, 275,000 (16.3%) were hospitalizations, and 52,000 (3.0%) were deaths.

A good neurologic examination, in combination with a thoughtful battery of tests, will invariably achieve the correct diagnosis. It is vital for the surgeon to become equally comfortable with the rapid assessment of the trauma patient and the potential for neurologic involvement. Once initial stabilization of the patient has been achieved, management of the neurologic injury is determined by specific causative conditions. Rapid recognition of potentially reversible causes (e.g., basilar artery occlusion [BAO], nonconvulsive status epilepticus, herniation), followed by the appropriate emergency consultations, can often prevent or reduce morbidity and mortality.[1-3]

This chapter is intended as a practical, rational, and efficient approach to the diagnosis and management of the maxillofacial trauma patient with neurologic involvement. Detailed discussions of neuroanatomy and the cranial nerves are available elsewhere and have been omitted from this chapter.

INITIAL ASSESSMENT

RESUSCITATION: INITIAL TRAUMA MANAGEMENT

The initial evaluation of a person who is injured critically from multiple traumas is a challenging task, for which every minute can make the difference between life and death. Over the past 50 years, assessment of trauma patients has evolved because of an improved understanding of the distribution of mortality and the mechanisms that contribute to morbidity and mortality in trauma. Mortality can be grouped into immediate, early, and late deaths. Immediate deaths are caused by a fatal disruption of the great vessels, heart, and lungs or a major disruption of body cavities. Early deaths may occur at any time, from minutes to hours after the injury. These patients frequently arrive at a hospital before death and succumb because of cardiovascular and/or pulmonary collapse. This leads to failed oxygenation of the vital organs, massive central nervous system injury, or both. The mechanisms of failed tissue oxygenation include inadequate ventilation, impaired oxygenation, circulatory collapse, and insufficient end-organ perfusion. Massive central nervous system trauma leads to inadequate ventilation and/or disruption of brainstem regulatory centers. Injuries that cause early trauma mortality occur in predictable patterns based on the mechanism of injury, the patient's age, gender, and body habitus, or environmental conditions. Late trauma mortality peaks from days to weeks after injury and is primarily caused by sepsis and multiple organ failure. Organized systems for trauma care are focused on the salvage of a patient

TABLE 8-1 Neurologic Examination

Parameter	Description or Function	Cranial Nerve Injury Associated Deficit
Mental status	Level of consciousness Glasgow Coma Scale Simplified motor scale AVPU ACDU	
Sensation	Touch, vibratory, proprioception, pain	
Cranial nerves	CN I: Olfactory—sense of smell	Anosmia
	CN II: Optic—visual fields and acuity	Visual field deficit; blindness
	CN III: Oculomotor—eyelid elevation, pupil reaction to light	Pupillary abnormalities, diplopia, ptosis
	CN IV: Trochlear—extraocular movement, superior oblique muscle	Diplopia secondary to superior oblique muscle paralysis
	CN V: Trigeminal—muscles of mastication and face sensation	Facial numbness, loss of corneal reflex, weakness of muscles of mastication
	CN VI: Abducens—extraocular movement (lateral rectus muscle)	Diplopia secondary to lateral rectus muscle paralysis (inability to abduct the eye)
	CN VII: Facial nerve—muscles of facial expression	Paralysis of muscles of facial expression
	CN VIII: Acoustic—hearing and balance	Vestibular dysfunction, hearing loss
	CN IX and X: Glossopharyngeal and vagus—gag reflex, swallowing, vocal cord mobility	Loss of gag reflex, dysphagia; vocal cord paralysis, tachycardia
	CN XI: Spinal accessory—movement of trapezius and sternocleidomastoid muscles	Paralysis of trapezius and sternocleidomastoid muscle, shoulder dysfunction
	CN XII: Hypoglossal—movement of tongue	Tongue deviation toward side of injury, tongue atrophy
Motor system	Muscle tone and strength	
Cerebellar function	Fine motor and gait	
Reflexes	Deep tendon reflexes, Babinski sign	

from early trauma mortality, whereas critical care is designed to avert late trauma mortality.

Surgeons' recognition of these patterns of injury have led to the development of the Advanced Trauma Life Support (ATLS) approach by the American College of Surgeons. ATLS is the standard of care for trauma patients; it is built around a standardized protocol for patient evaluation. This protocol ensures that the most immediate life-threatening conditions are actively identified and addressed in the order of their risk potential. The objectives of the initial evaluation of the trauma patient are as follows: (1) to stabilize the trauma patient; (2) to identify life-threatening injuries and to initiate adequate supportive therapy; and (3) to organize definitive therapy or transfer to a facility that provides definitive therapy efficiently and rapidly.[4-7]

The first and key part of the assessment of patients is the primary survey, in which life-threatening problems are identified and resuscitation efforts are also begun. A simple mnemonic, ABCDE, is used as a memory aid for the order in which problems should be addressed:

A, airway maintenance with cervical spine protection
B, breathing and ventilation
C, circulation with hemorrhage control
D, disability (neurologic evaluation)
E, exposure and environmental control

On completion of the primary survey, resuscitation efforts are well established, the vital signs are normalizing, and the secondary survey is begun. This may occur during transport or on arrival at the hospital. The secondary survey consists of a head to toe systematic assessment of the abdominal, pelvic and thoracic areas, complete inspection of the body surface to find all injuries, and a neurologic examination (Table 8-1). It also includes a complete history and physical examination, including the reassessment of all vital signs. Each region of the body must be fully examined. The purpose of the secondary survey is to identify all injuries so that they may be treated. A missed injury is one that is not found during the initial assessment (e.g., as a patient is brought into a hospital's ED), but rather manifests at a later point in time. Radiographs and further imaging indicated by examination are obtained. If at any time during the secondary survey the patient deteriorates, another primary survey is carried out because a potential life threat may be present. The person should be removed from the hard spine board and placed on a firm mattress as soon as reasonably feasible because the spine board can rapidly cause skin breakdown and pain whereas a firm mattress provides equivalent stability for potential spinal fractures.[6]

HISTORY

More than 3 million facial injuries occur in the United States each year. Maxillofacial injuries are commonly encountered in emergency and trauma settings, with most of them being secondary to assaults and motor vehicle accidents (MVAs). Information about the causes

of facial fractures depends on the country and location of the trauma center; therefore, reported statistics vary widely. More than 50% of patients with these injuries have multisystem trauma requiring coordinated management among ED physicians, trauma surgeons, maxillofacial trauma surgeons, and other medical and surgical specialists as required for the best outcome for each patient.[8,9]

Trauma to the maxillofacial anatomy mandates special attention. The maxillofacial region is a complex area that controls specialized functions, including seeing, hearing, smelling, breathing, eating, and talking. Also, the vital structures in the head and neck region are intimately associated. It is also of great importance to remember that the psychological impact of disfigurement can be devastating. The associated head injures and damage to the neurologic system are important to identify early in a patient who has sustained facial trauma. It is imperative that the maxillofacial surgeon be scrupulous during the initial history taking and physical examination of the patient to avoid any ill-intended oversight of an injury. Once the initial stabilization has been performed, a detailed history and complete physical examination are essential.[10,11] If the patient is unable to give an accurate history, witnesses to the injury must be questioned. Essentials of the history that must be ascertained through questioning include the following:

1. Was there a witnessed loss of consciousness following the injury? How much time was involved?
2. Was there a lucid interval immediately after the injury? And, did this precede a loss of consciousness?
3. Has there been a change or deterioration in the patient's mental status?
4. Did the patient have any seizures or syncopal events following the injury? Was there tonic deviation of the eyes toward one particular side?
5. Was there associated spinal column trauma? Did the patient complain of tenderness along the spine?

After the history is taken, a detailed general physical examination is essential. A rapid neurologic assessment can be made within minutes, thereby facilitating definitive diagnosis and treatment.

EVALUATION

MENTAL STATUS

Mental status alterations involve changes in alertness, cognition, and behavior. When referring to an individual's alertness, one is indicating a person's ability to interact with his or her environment. Cognition is how one processes and integrates information and behavior refers to how we react to the environment. An altered mental status could be caused by a traumatic injury, a manifestation of a primary neurologic condition, an acute systemic process, or a metabolic imbalance. During the clinical examination, it is important to distinguish among memory problems, disorders of thought, and an altered arousal or level of consciousness. The presence of memory problems or disorientation is indicative of a medical or neurologic illness, whereas thought disorders suggest a psychiatric illness.[12] A depressed level of consciousness represents failure of global brain function (Table 8-2).[13]

TABLE 8-2 Terms Describing Altered Mental Status

Term	Definition
Coma	State of total unresponsiveness
Stupor	State of severely impaired arousal with some unresponsiveness to vigorous stimuli
Obtundation	Decreased arousal state with some responsiveness to touch or voice
Lethargy (somnolence)	Decreased arousal state maintained spontaneously or with light stimulation
Confusion	State of impaired attention but adequate arousal
Delirium	Acute state of confusion with periods of agitation and sometimes hypervigilance, active irritability, and hallucinations; typically, alternating periods during which the level of arousal is depressed
Dementia	Insidious onset of progressive and irreversible decline in intellectual function; patients may experience also superimposed delirium, normal arousability, alertness

An immediate assessment of the patient's level of consciousness should be made in the trauma patient, because a decrease could represent an emergent condition. A patient with a normal level of consciousness is one who is awake, alert, and aware.[14] Quick assessment tools such as the Glasgow Coma Scale (GCS), AVPU, ACDU, and Simplified Motor Score (SMS; Table 8-3) can be used to rule out conditions that warrant immediate neurosurgical intervention but are not a substitute for a formal neuropsychiatric evaluation when it has been determined that it is indicated. It is also important to know the patient's baseline functional status, any history of altered behavior, current medications, medical comorbidities, prior psychiatric history, and any relevant circumstances that preceded the trauma event. The assistance of family members to obtain this pertinent history might be necessary in the trauma patient secondary to the presence of an artificial airway, sedation, or altered level of consciousness. An assessment of orientation, attention, memory, and executive function forms the foundation of the comprehensive cognitive mental status examination.[15]

The GCS score is the most commonly used tool for the assessment of altered levels of consciousness. It also guides therapeutic and diagnostic decision making and allows estimation of prognosis in TBIs.[16] It was developed in 1974 to improve communication among health care providers caring for patients with brain injuries. The current 15-point scale was published in 1976,[17] with the modification of the best motor response as having six levels and the division of flexion into two categories. Its three components assess the function of the cerebral cortex and the upper brainstem but do not

TABLE 8-3 Neurologic Scales for Assessment of Level of Consciousness

Scale	Score
SIMPLIFIED MOTOR SCALE	
Obeys commands	2
Localizes pain	1
Withdrawal to pain or less response	0
AVPU	
Alert	A
Responds to verbal stimuli	V
Responds to painful stimuli	P
Unresponsiveness to all stimuli	U
ACDU	
Alert	A
Confused	C
Drowsy	D
Unresponsive	U
GLASGOW COMA SCALE	
Eye Opening	
Spontaneous	4
To voice	3
To pain	2
None	1
Verbal Response	
Oriented, normal conversation	5
Confused conversation	4
Words but not coherent	3
Incomprehensible sounds	2
None	1
Motor Response	
Obeys commands	6
Localizes to pain	5
Withdraws to pain	4
Abnormal flexion (decorticate posture)	3
Abnormal extension (decerebrate posture)	2
None	1

assess cognition or differentiate delirium from dementia. Sedation and endotracheal intubation can affect its predictive value of the severity of the brain injury, need for neurosurgical intervention, and long-term outcome. The GCS was originally established for in-hospital use; however, it has been demonstrated that there is a correlation between field GCS scores and patient outcome and the need for neurosurgical intervention.[18] Reporting each GCS component in conjunction with the total score provides a better estimate of prognosis, because up to 38 combinations with 13 different sum scores are possible.[19-21] There is variation in scoring according to providers and institutions, which can lead to unnecessary referrals and neurosurgical interventions.[22] The neurologic assessment should be repeated multiple times, with each interval documented to obtain reliable clinical information about the patient's neurologic status. The best eye opening, best verbal response, and best motor response are reported using one of two schemes, EVM or EMV. The former is recommended, because it follows a logical clinical evaluation; the patient is asked to open the eyes and a verbal response is prompted, followed by a motor response.[22]

AVPU is an acronym used in ATLS. AVPU scores roughly correlate to a GCS score of 15, 13, 8, and 6 respectively.[23] As with the GCS, the AVPU system does not differentiate causes of altered mental status. The ACDU mnemonic was devised in Sweden. In a study comparing it to AVPU and the GCS scale in neurosurgical patients, ACDU scores roughly correlated to a GCS score of 15, 13, 10, and 6.[24] This study also found that the AVPU scale was not as effective for detecting early changes in level of consciousness and that the ACDU was easier to use, in particular in patients with small decreased level of consciousness. As such, it could be considered for use as a screening tool for ward patients.

The SMS was developed from the motor component of the GCS because it was thought that the individual parts of the GCS score were as predictive of outcome as the GCS score as a whole. The motor component alone of the GCS appears to be as sensitive as the GCS total score when predicting the severity of head injury in trauma patients and their risk of dying.[25,26] The SMS scale is as follows: obeys commands (2), localizes pain (1), and withdrawal to pain or less response (0). The three-point SMS demonstrates almost identical discriminatory value when compared with the six-point motor component of the GCS, emphasizing the importance of the 4 and 6 range in the GCS in predicting the need for intubation, neurosurgical intervention, and outcomes in TBI.[27,28] Furthermore, it has been found to have better interrater reliability than GCS, AVPU, and ACDU in patients presenting to the ED with an altered level of consciousness of traumatic or nontraumatic cause.[29]

CRANIAL NERVE EXAMINATION

Cranial nerve injury is an important component of neurotrauma, which may not be readily apparent in the emergency room to which the patient is brought after having sustained a maxillofacial injury. The incidence of cranial nerve injury in craniocerebral trauma varies between 5% and 23%.[30] The initial evaluation includes a detailed history and physical and neurologic evaluation, in which sensory and cortical function and hemodynamic status are assessed. It is also important to evaluate the ocular movements, pupils, facial symmetry, and laryngeal function. This will help determine the functional integrity of the cranial nerves. A detailed evaluation of each individual cranial nerve is a time-consuming and elaborate exercise, even in a conscious patient, and is not feasible in a comatose patient. Injury to the cranial nerves can occur by shearing forces, rapid acceleration or deceleration, injury to the skull base, and penetrating craniocerebral injuries.

Olfactory Nerve

Head injury is the most common cause of injury to the olfactory nerve[31,32] and the olfactory nerve is the most

common cranial nerve damaged in head injury.[33] Diagnosis of post-traumatic anosmia may not be of importance in immediate management. However, it may assume medicolegal importance for the patient in the future. Olfaction is impaired because of injury to the olfactory nerve filaments at the cribriform plate or because of injury to the olfactory bulb or olfactory tracts. The olfactory nerve filaments can get torn by fractures involving the cribriform plate of the ethmoid bone. Frontal or occipital blows can shear off the olfactory filaments through a shearing or stretching mechanism of the nerve fibers and any fractures in the area can lacerate the filaments. Olfaction can also be impaired by edema, hematoma, or ischemia.

Closed head injury can produce impairment of olfactory recognition despite preserved olfactory detection, secondary to injury to the orbitofrontal and temporal lobes.[34] Olfactory nerves and tracts can also be injured iatrogenically during surgical interventions involving the anterior skull base. Olfactory nerve damage is suspected in the presence of nasal bleed, periorbital ecchymosis, proptosis, and cerebrospinal fluid (CSF) rhinorrhea in the setting of craniocerebral injury. Clinically, patients often complain of an altered taste sensation.[35] In TBI with olfactory nerve dysfunction, almost 40% recover their olfaction. Most of these patients recover it within 3 months, and may continue up to 2 years.[36] In injuries in which there is severe disruption, spontaneous reconnection of the nerve fibers is not possible, making anosmia permanent.[37]

Optic Nerve and Chiasm

The optic nerve is approximately 4.5 to 5.0 cm in length, of which the intraorbital segment is the longest and the intraocular segment is the shortest. The intracanalicular portion (contained within the bony optic canal of the sphenoid bone) measures approximately 5 to 7 mm and is relatively immobile because of the fusion of the dural sheath of the nerve to the periosteum. The intracranial segment measures approximately 9 to 10 mm and joins with the opposite optic nerve to form the optic chiasm.

Optic nerve injury secondary to trauma has been described as a rare injury,[38] with only a few series reported in the literature.[39-41] Loss of vision is the chief complaint of a conscious patient with an optic nerve injury. A history of loss of consciousness is elicited in almost 90% of cases of optic nerve damage[40] and the presence of an associated epistaxis (Maurer's triad—head injury, epistaxis caused by traumatic pseudoaneurysm of the internal carotid artery, uniocular blindness) should alert the clinician to the possibility of an accompanying optic nerve injury.[42,43] Diagnosis of optic nerve injury is difficult in patients with severe head injury with altered sensorium. An afferent pupillary defect (Marcus-Gunn pupil) is seen in an isolated optic nerve injury; bright light shone on the normal side elicits a consensual reflex on the affected side, whereas light shone on the injured side elicits relative pupillary dilatation (Fig. 8-1). Optic nerve atrophy sets in approximately 4 to 6 weeks after an injury has taken place.

Operative versus nonoperative management of an optic nerve injury is controversial. Mahapatra and Bhatia were the first to carry out a protocol based on a prospective study and established the role of visual evoked potentials in the management of optic nerve injury.[44] They performed studies following different protocols, and showed spontaneous visual recovery in 51% to 57% cases with conservative management.

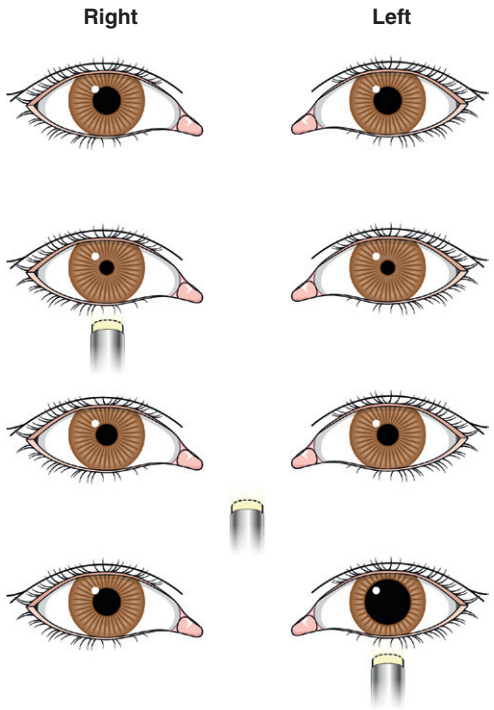


FIGURE 8-1 Left eye relative afferent pupillary defect (Marcus-Gunn pupil). When light stimulates the right eye (unaffected eye), both pupils constrict equally and simultaneously. The right pupil has intact direct reflex and the left pupil constricts because of an intact consensual reflex. When the light is shined to the left eye, pupils might dilate momentarily because neither eye is stimulated. When the light stimulates the left eye, the pupil will continue to be dilated because of an afferent defect and the right eye will not have a consensual reflex. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

The treatment used is as follows:

1. All patients are given large doses of dexamethasone (initial dose, 0.75 mg/kg, followed by 0.33 mg/kg every 6 hours for the next 24 hours)[45] Steroids are beneficial by reducing cellular edema, a membrane-stabilizing action, and reducing circulatory spasm. Studies have shown various recovery rates, from 30% to 100%, after nonoperative treatment.[46,47]
2. No patient is operated within 3 weeks. The visual evoked potential (VEP) test is given every week.
3. In cases of visual recovery or in patients with no perception of light, no surgery is carried out.
4. In patients with partial recovery that becomes static, optic nerve decompression can be carried out.
5. In patients in whom identifiable bony fragments and hematomas are present, optic nerve decompression is carried out immediately.[48]

Chiasmal injury is rare, seen in only 0.3% of all cases of head injury.[49] It is often associated with a severe acceleration-deceleration strain; therefore, it is likely that the low incidence of chiasmal injury is related to the low

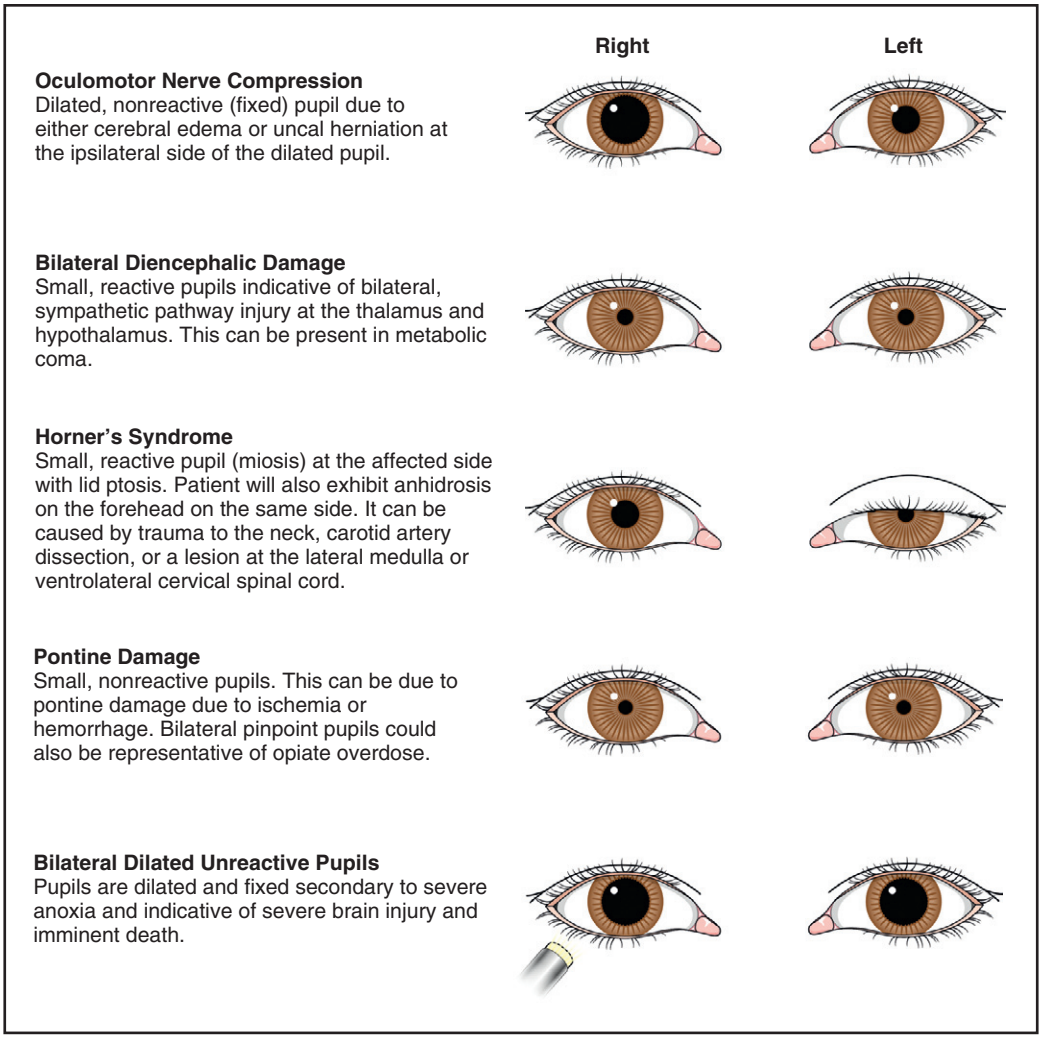


FIGURE 8-2 Abnormal pupillary responses during ocular examination. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

overall survival from this severe type of head injury. Severe frontal impact can cause fracture in the sella and clinoid regions. There may be multiple facial fractures, causing separation of the optic foramina and splitting of the chiasm.[50] Skull base fracture can also extend to the sella and clinoids, causing direct injury to the chiasm. During clinical examination, bitemporal field defects are noted and, in the case of associated optic nerve injury, there will be a visual loss in the involved eye. Funduscopic examination also reveals pallor of the optic disc on the nasal side. Other cranial nerves may be involved in a chiasmal injury and the management of this type of injury is restricted to relieving the compressive bony fragment or a hematoma.

Oculomotor Nerve

Head trauma accounts for 8% to 16% of all oculomotor palsies,[51,52] which is seen in 2.9% of all head injuries, including patients with multiple cranial nerve involvement.[53] Of all the cranial nerves, oculomotor palsy in a patient with head injury imparts a sense of urgency in imaging and management because of the possibility of an expanding intracranial hematoma (Fig. 8-2). Midbrain hematoma in the tectal region can present with oculomotor palsy, which often results from compression of the nerve at the tentorial hiatus by the uncus during transtentorial herniation. Avulsion or stretching of the nerve at the mesencephalopontine junction can also result in an isolated oculomotor palsy.[54] Frequently, oculomotor palsy occurs in conjunction with other ocular motor nerves contained in the cavernous sinus in the case of skull base fracture. Injury at the superior orbital fissure, orbit, or maxillofacial injury can result in injury to the superior or inferior divisions of the nerve. The oculomotor nerve can also be injured in its course in the brainstem by a shearing injury.

In the setting of a head injury, unilateral mydriasis (Hutchinson's pupil) assumes ominous importance as being representative of an ipsilateral, supratentorial, expanding hematoma. Invariably, this is accompanied by concurrent alteration in sensorium. Mydriasis may also be accompanied by ptosis and extraocular muscle weakness. An avulsion or stretch oculomotor nerve injury is suggested by the rapid recovery of consciousness in an otherwise complete motor palsy present in the clinical examination.[55] This injury may be relatively mild.[56] In a

patient with proptosis, time should be allowed for the swelling to regress before ocular movements can be assessed appropriately. Detection of ocular pulsations and bruit over the eyeball makes the diagnosis of a carotico-cavernous fistula obvious.

Ocular motility should be assessed after improvement in the level of consciousness and resolution of periorbital swelling. Palsy caused by a compressive lesion is likely to resolve on removal of the lesion, whereas injury to the fascicles is likely to give rise to an aberrant regeneration, in which axons terminate in inappropriate structures. Such a regeneration can result in elevation of the optotic eyelid on adduction and a pupil that is poorly reactive to light but constricts with adduction.[57] Management of unresolved oculomotor palsy is difficult; extraocular muscle surgery can achieve binocular vision in the primary position and reading positions.

Trochlear Nerve

The incidence of trochlear nerve injury is 2.14% in head injuries.[53] It is often accompanied by injury to other ocular motor nerves. When the trochlear nerve is injured in isolation, this tends to occur at its subarachnoid course. A sudden deceleration impact or blow to the head may cause the brain to move back and the brainstem to impact against the tentorium, resulting in a trochlear nerve injury. The presence of bilateral trochlear nerve injury is always caused by trauma.[58,59]

Diagnosis of trochlear nerve injury is generally made in a conscious and cooperative patient. The patient complains of diplopia with a perceived slant in the environment, which is compensated by adopting a characteristic head tilt (Bielschowsky's head tilt), with the head being tilted away from the affected eye. Clinical examination reveals hypertropia (visual axis higher in the affected eye) that worsens on lateral gaze. Bilateral trochlear palsy is diagnosed by the presence of alternating hyperdeviation (upward deviation of one eye) in various positions of upward gaze. In the presence of concomitant oculomotor nerve palsy, trochlear nerve palsy can be suspected in the absence of intorsion of the eye. Spontaneous recovery occurs in 65% of patients with a unilateral trochlear nerve palsy.[60] The use of an eye patch or prisms pasted onto spectacles can be useful for achieving binocular single vision. In case of incomplete recovery after 12 months, corrective ocular muscle surgery can be carried out.

Abducens Nerve

Head injury accounts for almost 3% to 15% of abducens palsies.[52] Its long intradural course, with passage over the petrous ridge, its relative fixed course under the petroclinoid ligament, and its location in the cavernous sinus makes it vulnerable to stretch or tear.[61] Hyperextension trauma to the cervical spine can also lead to an abducens nerve palsy, which is typically accompanied by lower cranial nerve palsies.[62]

A conscious patient typically complains of diplopia on distant fixation; examination of ocular movements reveals convergence of the visual axes and lateral rectus palsy. In an unconscious patient, the eyeball can be seen adducted, with no abduction on oculocephalic response. Lateral rectus palsy may recover completely or incompletely. Diplopia may be corrected by prisms or by botulinum therapy for antagonism of the medial rectus to achieve binocular vision. Ocular muscle surgery is offered in cases of persistence of lateral rectus palsy after 6 to 12 months.

Trigeminal Nerve

Branches of the trigeminal nerve are often injured during severe maxillofacial and skull base injury, because these nerves exit the various foramina from the skull. The supraorbital and infraorbital nerves are injured in trauma to the forehead, orbit, or maxilla. The inferior dental branch and the mandibular division (V3) of the trigeminal nerve can be injured in fractures of the mandible. Skull base injury with injury to the cavernous sinus region can result in ocular motor dysfunction, with sensory impairment over the ophthalmic division (V1). Closed or penetrating injuries can cause injury to the trigeminal ganglion.[63] Skull fractures involving the middle fossa can extend into the foramen ovale and foramen rotundum and damage the exiting nerves.

Sensation along the cutaneous distribution of the involved nerve is affected (Fig. 8-3). Hyperpathia can occur along the distribution of the affected nerve and patients should be evaluated for corneal anesthesia, because the eye requires sensation to be protected against exposure keratitis and corneal ulceration. Patients who develop a hyperalgesia can be treated with carbamazepine or gabapentin. Alternatively, in intractable cases, the involved root can be sectioned or radiofrequency ablation of the ganglion can be done to relieve pain.

Petrous Fractures

The temporal bone itself is composed of five parts—the squamous, petrous, mastoid, and tympanic portions, as well as the styloid process (Fig. 8-4). The squamous portion is smooth and convex; the temporalis muscle attaches to this region. The zygomatic arch projects forward from the inferior part of the squamous portion, giving rise to the articular tubercle just anterior to the glenoid fossa. The fossa is bounded posteriorly by the tympanic portion of the temporal bone and the bony external auditory canal. The pyramid-shaped petrous

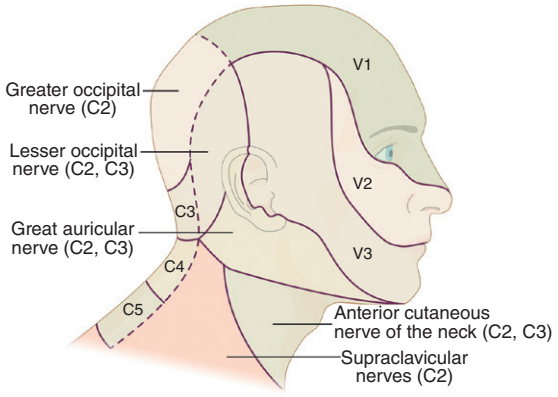


FIGURE 8-3 Cutaneous sensory distribution of the head and neck. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

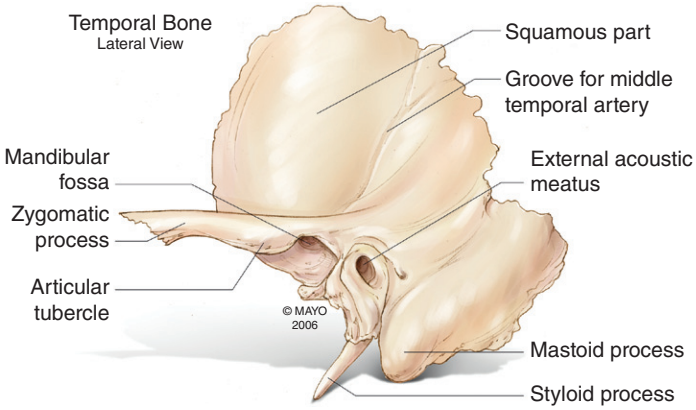


FIGURE 8-4 Lateral view of the temporal bone. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

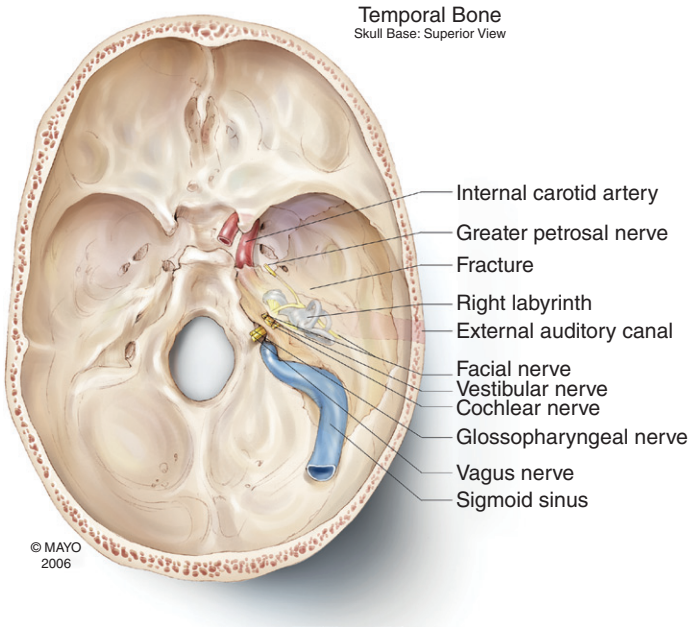


FIGURE 8-5 Superior view of the temporal bone. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

portion of the temporal bone is wedged between the sphenoid and occipital bones at the base of the skull. There are many important structures that pass through it, including the carotid canal, internal jugular vein, and facial nerve (Fig. 8-5). An injury to the facial and vestibulocochlear nerves is common after head injury and a temporal bone fracture is a frequent accompaniment to this injury. As such, it would be pertinent to review briefly this type of fracture here.[64]

There are generally two types of temporal bone fracture patterns described and their reported incidence is variable. Studies citing the radiographic incidence of each fracture type span a time period during which advances in imaging technology have led to much more detailed radiographic images, and thus to differences in the frequency of each radiographic diagnosis. Some fractures are not purely of a single type, but rather are a combination of longitudinal and transverse. As a general rule, classic longitudinal fractures account for about 80% of cases, with the remaining 20% being transverse fractures.

Longitudinal fractures are most frequently caused by a lateral blow to the skull in the parietal region. Transverse fractures are more commonly caused by an intense blow to the occipital region or by a direct frontal injury. The latter are more frequently serious or fatal. A fracture with significant comminution or fracture lines running in both the longitudinal and transverse directions is termed a *mixed fracture*. Because studies have suggested that the traditional classification system may not predict the presence of sequelae, such as facial nerve injury or CSF leak, alternatives to the traditional classification scheme have been developed. One alternative scheme that has demonstrated better correlation classifies fractures based on whether they are otic capsule–sparing or otic capsule–violating. Others have suggested that fracture classifications should include more specifics regarding fracture location because of the difficulty in describing many fractures as longitudinal or transverse, or should describe fractures in a manner that correlates with the optimal surgical approach to repair the facial nerve.[64]

Facial Nerve

Trauma is the second most common cause of facial paralysis after Bell's palsy.[65] During a deceleration head injury, the facial nerve is injured at the geniculate ganglion, where it is tethered by the greater superficial petrosal nerve. The shearing force results in an intraneural contusion, edema, and hemorrhage. Transection can also occur in severe head injury cases. The presence of a fracture of the otic capsule indicates severe trauma, and the facial nerve damage is generally complete in such cases.[66] Facial nerve palsy can be seen in 45% to 50% of patients with a gunshot wound to the face.[67] A missile fragment enters from the lateral or inferior aspect of the temporal bone and, as it lodges in the bone, it dissipates its energy, damaging the facial nerve. The facial nerve is commonly injured at its vertical segment as it exits the cranium at the stylomastoid foramen.[68] Immediate paralysis carries a worse prognosis and is caused by transection of the nerve or another form of severe neural trauma. Delayed paralysis, longer than 24 hours, is caused by nerve edema and swelling of the nerve within its sheath or epineurium; this carries a better prognosis. Delayed paralysis can also be caused by external compression by an expanding hematoma or edema of the loose fibrous tissue and periosteum between the nerve and bony canal.[69]

Generally, facial asymmetry is clinically obvious in a conscious patient. In an unconscious patient, there could be incomplete eye closure, with associated Bell's phenomenon. Bleeding from the ear, CSF otorrhea, the Battle sign, and hearing impairment are other features that should alert the physician to the possibility of a facial nerve injury. Detailed evaluation of the secretomotor and taste sensations can be done when the patient is conscious and cooperative. The site of injury can be ascertained by Schirmer's test, submandibular salivary flow, stapedial reflex, and electrogustometry testing. However, in contemporary practice, high-resolution computed tomography (CT) and electrical testing studies can delineate the site of the injury.

Management of traumatic facial palsy continues to be controversial, with no consensus about the indications and timing of surgical intervention. Turner has reported on the natural history of immediate and delayed traumatic facial paralysis, with satisfactory return of facial function in 82% of cases of delayed facial palsy. Patients with immediate paralysis had good recovery in 53% of cases. An incomplete recovery may be functionally acceptable in some patients. Patients should be reassured and watchful waiting initiated.

In patients with a facial nerve paralysis, care is taken to avoid exposure keratitis and dryness of the cornea at the affected site. Earlier recommendations of waiting for 3 weeks have now become obsolete for patients requiring surgical intervention, with findings of experimental studies advocating early surgery when appropriate. Decompression of the facial nerve improves recovery if performed within 48 hours; slitting of the epineurium does not seem to confer any benefit.[70] Presence or absence of anacusis determines the approach to be taken for decompression of the facial nerve. In anacusis, the translabyrinthine approach is used because it allows excellent access to the entire intratemporal segment of the nerve. In the presence of hearing, a combined transmastoid middle fossa approach is used, which permits access to the perigeniculate and vertical segments of the nerve.[70] Results of facial nerve grafting appear to favor early repair over delayed grafting. Anatomically intact facial nerve has a higher potential for recovery. However, there may be unpleasant sequelae of recovery in the form of dysacusis, ageusia, epiphora, and gustatory lacrimation. Late sequelae can be in the form of facial contractures, tics, spasm, and synkinesis. Recovery from facial palsy is graded according to the House-Brackmann grading system.

Vestibulocochlear Nerve

Following head injury, hearing loss can occur because of damage to the vestibulocochlear nerve. Transverse fractures of the petrous bone can damage the anterior portion of the vestibule and cochlea. These organs can also suffer a concussion. An injury can occur with damage to the central auditory pathways in the brainstem. Pressure waves generated by a blow to the head can damage the hair cells in the cochlea and cause high-frequency hearing loss and tinnitus.[71] In addition to fracture of the bony otic capsule, the nerve can also be damaged at the internal acoustic meatus.

In a patient with an altered sensorium, ear bleeding, CSF otorrhea, and the Battle sign should arouse the suspicion of an eighth nerve injury. In a conscious patient, a thorough medical history should include information about the presence of vertigo, nausea, tinnitus, and impaired hearing. Otoscopic examination might reveal hemotympanum or the presence of CSF in the middle ear. Facial palsy often is associated with this injury. Conductive deafness generally has a useful recovery and is amenable to surgical intervention. However, sensorineural deafness has a poor recovery, especially if the hearing loss has been complete. Tinnitus is usually self-limiting and requires no more treatment other than reassurance. However, labyrinthine symptoms of vertigo, nausea, and dizziness may persist, which may require treatment with labyrinthine sedatives.

Glossopharyngeal, Vagus, and Accessory Nerves

The three lower cranial nerves are injured together because of their close proximity with one another in the jugular foramen. These nerves, after exiting the brainstem, enter the jugular foramen and a fracture of this region can lead to injury of the three nerves. This type of injury is uncommon because of the infrequent injury pattern of the posterior skull base in comparison to that of the anterior and middle skull base. More often, the nerves are injured in their extracranial course because of stab wounds, gunshot injuries, or stretch from high falls. Occipital condyle fracture can also lead to injury to the ninth and tenth nerves.[72]

Injury to the ninth, tenth, and eleventh nerves is underestimated, because it may not be readily apparent in the setting of head injury. A patient might experience dysphonia, dysphagia, depressed gag reflex, and/or ipsilateral palatal palsy with paralysis of the trapezius and sternocleidomastoid muscles (Vernet syndrome). Indirect or direct laryngoscopy would help confirm ipsilateral vocal cord palsy. This is accompanied by loss of sensation over the posterior third of the tongue, soft palate, uvula, and larynx. A lesion outside the skull is likely to affect the cervical sympathetic, and the clinical picture includes Horner's syndrome.

Prognosis is generally favorable in cases unaccompanied by skull base fracture. These patients may require nasogastric feeding if swallowing is impaired and causing aspiration. Once swallowing is reestablished, oral feeding can be resumed. If the risk of aspiration persists, these patients will require alternative long-term enteral feeding to maintain hydration and nutritional intake. Detailed evaluation by a speech or swallowing pathologist will be required for assistance in long-term care and management.

Hypoglossal Nerve

The hypoglossal nerve is one of the least injured nerves in traumatic head injuries. Fractures of the occipital condyle can damage the hypoglossal nerve as it passes medial to the condyle. Hypoglossal palsy can occur late, after a minor head trauma and condylar injury.[73,74] More often, the hypoglossal nerve is injured in the course of surgery on the neck while operating on the submandibular gland and on C2-C3 cervical disc procedures.

Patients with a hypoglossal nerve injury can experience speech alteration and also have difficulty in moving the food bolus in the mouth. Clinical examination will reveal that tongue movement is absent on the side of injury, with deviation of the protruded tongue to the injured side. Recovery is expected in mild injury cases.

Motor System

The motor system can be divided into the following: (1) the peripheral apparatus, which consists of the anterior horn cell and its peripheral axon, the neuromuscular junction, and muscle; (2) the more complex central apparatus, which includes the descending tracts involved in control; and (3) the systems involved in

initiating and regulating movement, the basal ganglia and cerebellum.

Dysfunction in individual components of the motor system results in fairly specific abnormalities that can be evaluated at the bedside. Although multiple components may be involved, particularly with diseases of the central nervous system, isolated involvement of the various components commonly occurs. Examination for motor dysfunction includes assessment of strength, muscle tone, muscle bulk, coordination, abnormal movements, and various reflexes. Many of these are best detected through simple but careful observation. However, a few maneuvers aid in the detection of abnormality.

The power of the individual muscle groups is measured on a scale from 0 to 5:

- Grade 0—no motor activity
- Grade 1—palpable muscle contraction; no joint motion
- Grade 2—complete range of motion with gravity eliminated
- Grade 3—complete range of motion against gravity; no resistance
- Grade 4—complete range of motion against gravity with some resistance
- Grade 5—complete range of motion against gravity with full resistance

The respective muscle groups are referenced in terms of fractions of five (e.g., four fifths or strength as four out of five.

When it is possible to assess the patient's gait, important information is provided regarding the extent of injury. Gait is tested by having the patient walk normally and in tandem. In the latter, the patient is asked to walk with one foot immediately in front of the other (i.e., heel to toe). A tendency to sway or fall to one side indicates ataxia, suggesting an ipsilateral cerebellar dysfunction. Atonia and asthenia can occur in other lesions of the nervous system and are not specific to the cerebellum; their testing is described elsewhere.

Loss of ability to perform finger to nose, heel to shin, and rapid alternating movements points to cerebellar disease. Hemispheric lesions usually lateralize ipsilaterally, whereas midline (vermian) lesions tend to manifest with bilateral dysfunction.

Sensory System

Light touch, pain, heat, cold, and vibration provide an evaluation of the peripheral nerves and their central pathways to the thalamus. Light touch is tested by touching the skin with a wisp touch, using a piece of cotton or tissue. Pain is tested using a sharp instrument such as a pin. Temperature can be tested by touching the patient's skin with test tubes, one with warm water and the other with cold water. A comparison between both sides provides a reference point with the unaffected side. Vibration is tested with a tuning fork, preferably at a frequency of 128 Hz. Again, compare this on both sides and assess those findings with the same body part of the examiner.

The cortical sensory system includes the somatosensory cortex and its central connections. This system enables the detection of the position and movement of the extremities in space, size and shape of objects, tactile sensations of written patterns on the skin, and tactile localization and tactile discrimination on the same side or both sides of the body. Position sensation is tested with the patient's eyes closed. The examiner moves various joints, being sure to hold the body part so that the patient does not recognize movement simply from the direction in which the patient may feel the pressure from the examiner's hand. Stereognosis is tested by placing some familiar object in the patient's hand while his or her eyes are closed and asking the patient to identify the object. Inability to recognize the size or shape is referred to as astereognosis. Agraphesthesia is the inability to recognize letters or numbers written on the patient's skin. These abilities are impaired in lesions of the right parietal region.

Check the deep tendon reflexes using impulses from a reflex hammer to stretch the muscle and tendon. The limbs should be in a relaxed and symmetrical position, because these factors can influence reflex amplitude. As in muscle strength testing, it is important to compare each reflex immediately with its contralateral counterpart so that any asymmetries can be detected. If you cannot elicit a reflex, you can sometimes do so by certain reinforcement procedures. For example, have the patient gently contract the muscle being tested by raising the limb very slightly, or have him or her concentrate on forcefully contracting a different muscle group just at the moment when the reflex is tested. When reflexes are very brisk, clonus is sometimes seen. This is a repetitive vibratory contraction of the muscle that occurs in response to muscle and tendon stretch. Deep tendon reflexes are often rated according to the following scale:

- 0, absent reflex
- 1+, trace, or seen only with reinforcement
- 2+, normal
- 3+, brisk
- 4+, nonsustained clonus (repetitive vibratory movements)
- 5+, sustained clonus

Deep tendon reflexes are normal if they are 1+, 2+, or 3+ unless they are asymmetrical or there is a dramatic difference between the arms and legs. Reflexes rated as 0, 4+, or 5+ are usually considered abnormal.

In addition to clonus, other signs of hyperreflexia include spreading of reflexes to other muscles not directly being tested and crossed adduction of the opposite leg when the medial aspect of the knee is tapped.

GRADING THE SEVERITY OF INJURY

Neurologic injury can be characterized anatomically or functionally.

ANATOMIC SIGNS

Plum and Posner have described coma anatomically, using respirations, pupils, eye movements, and motor examination to localize lesions and to further describe the rostrocaudal progression of brainstem herniation.[75] Tests for oculocephalic (doll's eyes) and oculovestibular (cold caloric) responses can yield valuable information.

Both tests assess the integrity of the brainstem. With an intact brainstem, the eyes should remain fixed straight ahead while the head is forcefully turned to one side or the other (doll's eyes). Injection of cold water into one ear causes conjugate movement of both eyes ipsilaterally (toward the irrigated ear) in a patient with an intact brainstem (cold caloric).

Cheyne-Stokes respirations are characterized by periods of hyperventilation separated by periods of apnea. In apneustic breathing, the victim has gasping and irregular respirations, which are separated by long periods of apnea. Decorticate posturing implies the destruction of cortical input to the red nucleus of the midbrain (rubrospinal tract); it is characterized by arms adducted and flexed, with the wrists and fingers flexed on the chest. The legs may be internally rotated and stiffly extended, with plantar flexion of the foot. Decerebrate posturing reflects the absence of cortical input further caudally, resulting in the arms adducted and extended, with the wrists pronated and the fingers flexed. The legs may be internally rotated and stiffly extended with plantar flexion of the feet.

FUNCTIONAL SIGNS

Jennett and Teasdale[76] have described GCS in an attempt to characterize the severity of injury and possible outcome.[76-80] The scale uses three parameters—eye opening, best motor response, and best verbal response. The postresuscitation GCS is generally considered more reliable in providing an initial assessment of injury severity. Although not consistently defined, this score generally refers to the best GCS obtained within the first 6 to 8 hours of injury following nonsurgical resuscitation. In severe head injury, the scale usually reads between 3 and 8, in moderate head injury, it is between 9 and 13, and in mild head injury, it is between 14 and 15.

Coma is defined as the inability to obey commands, utter words, and open the eyes. No single score within the range of 3 to 15 points forms a cutoff point for coma. However, 90% of all patients with a score of 8 or less, and none of those with a score of 9 or more, are found to be in coma according to the preceding definition. Therefore, a GCS score of 8 or less has become the generally accepted definition of coma.

MAYO HEAD INJURY CLASSIFICATION SYSTEM FOR TRAUMATIC BRAIN INJURY

Category A

If one or more of the following criteria apply, classify the injury as moderate to severe (definite) TBI:

1. Death caused by this TBI
2. Loss of consciousness of 30 minutes or longer
3. Post-traumatic anterograde amnesia of 24 hours or longer
4. Worst GCS full score in the first 24 hours less than 13, unless invalidated on review (e.g., attributable to intoxication, sedation, systemic shock)
5. One or more of the following present: intracerebral hematoma, subdural hematoma, epidural hematoma, cerebral contusion, hemorrhagic contusion, penetrating TBI (dura penetrated), subarachnoid hemorrhage, brainstem injury

Category B

If none of the criteria in category A apply, classify as mild (probable) TBI if one or more of the following criteria apply:

1. Loss of consciousness of momentary to less than 30 minutes
2. Post-traumatic anterograde amnesia of momentary to less than 24 hours
3. Depressed, basilar or linear skull fracture (dura intact)

Category C

If none of the criteria in category A or B apply, classify as symptomatic (possible) TBI if one or more of the following symptoms are present: blurred vision; confusion (mental state changes); dazed; dizziness; focal neurologic symptoms; headache; nausea.[81]

DIAGNOSTIC STUDIES IN HEAD INJURY

CT is the imaging modality of choice for acute head-injured patients. It is used during the initial screening of head trauma patients because of its ease, availability, and diagnostic value.[82-84] The radiographic evaluation of the trauma patient with suspected intracranial injuries allows the identification of life-threatening injuries that require immediate intervention; the findings can also be correlated with those of the neurologic examination to help guide treatment and prognosis.[85] The use of CT scanning is of increasing value in the severely injured patient, in whom the need for intubation, ventilation, and sedation limits the clinical examination and assessment of the injury severity.

In a retrospective review of trauma patients admitted at a large trauma center, almost 50% of patients required head CT as part of their initial evaluation.[86] Of these, 12.4% of patients were also diagnosed with a facial fracture, demonstrating the value of CT in the overall survey of the polytrauma patient. When considering whether a facial CT should also be ordered at the time of obtaining a head CT, the presence of a lip laceration, intraoral laceration, periorbital contusion, subconjunctival hemorrhage, and nasal laceration were found to have a high correlation with the presence of facial fractures.[87]

In patients with severe injuries, CT scans are typically used to assess for internal injuries involving the head and neck, chest, and abdomen. Determining which stable, alert trauma patient requires spinal imaging can be challenging. The history, mechanism of injury, physical examination, and clinical decision rules aid in determining which patients need spinal imaging and which type is to be used. The clinical decision rules for spinal and head imaging help limit the amount of unnecessary CT scans, provide for better use of resources, and limit the amount of radiation exposure while not missing life-threatening injuries. The clinical decision rules available include the Canadian Computed Tomography Head Rule (CCHR), the New Orleans Criteria (NOC), and the NEXUS Low-Risk Criteria (NLC)[88-90] (Box 8-1).

BOX 8-1 Clinical Decision Rules for Obtaining Cervical Spine or Head Radiographs in Trauma Patients

NEW ORLEANS CRITERIA

CT is required for patients with a minor head injury with any one of the following findings (these criteria apply only to patients who also have a GCS score of 15):

- Headache
- Vomiting
- Age ≥ 60 yr
- Drug or alcohol intoxication
- Persistent anterograde amnesia (deficits in short-term memory)
- Visible trauma above the clavicle
- Seizure

NATIONAL EMERGENCY X-RADIOGRAPHY UTILIZATION STUDY (NEXUS): LOW-RISK CRITERIA

Cervical spine radiography is indicated for trauma patients unless they satisfy all five criteria:

- No posterior midline cervical spine tenderness
- No evidence of intoxication
- Normal level of alertness
- No focal neurologic deficits
- No painful distracting injuries

CANADIAN C-SPINE RULE

Radiography is indicated for patients in whom a C-spine injury is a concern when any of the following is present:

- Age ≥ 65 yr
- Dangerous mechanism of injury (fall from 3 ft or five stairs, axial load to the head, MVA at high speed, ejection from a vehicle)
- Paresthesias in the extremities

In patients with none of the high-risk characteristics listed, assessment for any low-risk factor that allows for safe assessment of neck range of motion is then done:

- Simple rear end MVA
- Sitting position in ED
- Ambulatory at any time
- Delayed onset of neck pain
- Absence of midline cervical spine tenderness

Patients who do not exhibit any of the low-risk factors are not suitable for range-of-motion-testing; therefore, radiographs must be obtained. When range of motion can be tested, radiographs are indicated in patients who are not able to rotate their neck actively 45 degrees left and right.

CANADIAN COMPUTED TOMOGRAPHY HEAD RULE

High Risk for Neurosurgical Intervention

- Glasgow Coma Scale score < 15 at 2 hr after injury
- Suspected open or depressed skull fracture
- Any sign of basal skull fracture (hemotympanum, raccoon eyes, otorrhea or rhinorrhea, Battle sign)
- Two or more episodes of vomiting
- Age ≥ 65 yr

Medium Risk for Brain Injury Detection by CT

- Amnesia before impact ≥ 30 min
- Dangerous mechanism (struck by a motor vehicle, ejected from a motor vehicle, or fall from an elevation of 3 feet or more or five stairs

The rule is not applicable if the patient did not experience a trauma, has a GCS score lower than 13, is <16 yr of age, and is anticoagulated or has an open skull fracture.

In the NLC decision instrument, no spine radiographs are necessary as long as the patient satisfies all of the following five criteria: (1) no midline cervical tenderness; (2) no focal neurologic deficit; (3) normal alertness; (4) no intoxication; and (5) no painful distracting injury.[88] In the NOC, a trauma patient should undergo CT scanning if any one of the following findings is present: headache, vomiting, age older than 60 years, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicles, and seizure.[90] The CCHR is designed for patients older than 16 years with a GCS score between 13 and 15 and loss of consciousness but no neurologic deficit, seizure, or anticoagulant therapy. Patients with minor head injuries are categorized into two levels of risk, medium and high. A patient with any one of the five high-risk factors or one with two medium-risk factors should have CT because of the high risk of requiring neurosurgical intervention or having a clinically important lesion diagnosed on CT.[89]

Traumatic intracranial injuries can be classified into penetrating and nonpenetrating. In the penetrating type, a foreign object or a fracture fragment penetrates the intracranial compartment. Nonpenetrating trauma occurs by contact with an object or an acceleration-deceleration or rotational motion injury. In penetrating injuries, the use of CT scans helps determine the need for surgical intervention and the presence of a foreign object and can help predict complications that can arise from the surgical intervention. Neuroimaging findings with prognostic implications include the following: (1) entry and exit sites; (2) missile track; (3) presence of intracranial fragments; (4) presence of intracranial air; (5) transventricular injury; (6) midline shift; (7) mass lesion; and (8) basal cistern effacement. Intracranial injury from missiles depends on the size and velocity of the foreign object. The injury can be caused by direct laceration, shock wave transmission, and cavitation.[91]

The prediction of outcome in patients with severe head injuries allows for proper therapeutic interventions to be instituted, allocation of required resources, and planning for adequate rehabilitation. Important independent prognostic factors in regard to outcomes of TBI are age, GCS motor score, pupil response, presence of traumatic subarachnoid hemorrhage (SAH), and CT findings.[92] The Marshall CT classification groups patients into six categories according to CT findings and helps estimate the risk of neurologic deterioration that would warrant early surgical intervention (Table 8-4). It uses the status of the mesencephalic cisterns, degree of midline shift, and presence or absence of local lesions to categorize patients. There is a strong correlation between the Marshall CT classification and mortality and frequency of elevated intracranial pressure (ICP),[91,93] with early

TABLE 8-4 Marshall Computed Tomography Classification of Traumatic Brain Injury

Category	Definition
Diffuse injury	
I	No visible intracranial pathology evident on CT scan
II	Cisterns present with midline shift of 0-5 mm and/or lesion densities present; no high- or mixed-density lesion > 25 mL; may include bone fragments
III (swelling)	Cisterns compressed or absent with midline shift 0-5 mm; no high- or mixed-density lesion > 25 mL
IV (shift)	Midline shift > 5 mm; no high- or mixed-density lesion > 25 mL
Mass lesion	
Evacuated (V)	Any lesion surgically evacuated
Nonevacuated (VI)	High- or mixed-density lesion > 25 mL; not surgically evacuated

death being correlated with the presence of midline shift greater than 5 mm on initial CT scan and a high-density or mixed lesion larger than 25 mL in volume.[91] The Marshall CT classification is easy to use but it has its limitations, because it depends on the arbitrary assessment of the volume of mass lesions, does not take traumatic SAH into consideration, and two of the categories (V and VI) are based on a retrospective review of whether or not a surgical intervention was performed.[91]

MANAGEMENT OF HEAD INJURY

The pathophysiology of traumatic brain injury is complex. A TBI could be the result of direct or indirect traumatic forces. Rotational, acceleration, or deceleration inertial forces during a fall or MVA can lead to superficial or deep brain lesions. After a patient survives the initial direct trauma, morbidity and mortality are mostly dependent on the severity of the secondary biomechanical processes. A cascade of molecular and functional changes takes place after an injury, with some of these changes being beneficial but many being harmful. Within the rigid skull, the relationship between intracranial volume and ICP becomes very important. Brain tissue edema, hemorrhage, and hematomas lead to an increase in cerebral volume, which in turn causes an increase in ICP.

During primary brain injury, direct mechanical forces lead to cell damage and axonal dysfunction. This direct damage is manifested as focal or diffuse lesions. Focal brain injuries include cerebral contusions, subarachnoid hemorrhage, and subdural and epidural hematomas. Their impact on morbidity and mortality is based on their location, size, and progression. Diffuse brain injuries include concussion and diffuse axonal injury (DAI) secondary to the shearing of axons.

Secondary brain injury occurs as a result of biochemical substances released because of the initial injury, which lead to further neural tissue damage and cause progressive neurologic deterioration and functional compromise. Brain trauma can also lead to dysfunction of the blood-brain barrier (BBB), which in turn contributes to the progression of cerebral edema. This cerebral edema is classified as vasogenic or cytotoxic. In vasogenic edema, there is increased permeability of the BBB because of osmotically active molecules, such as plasma proteins, that lead to their accumulation in the interstitial fluid. Cytotoxic edema is caused by cellular accumulation of osmotically active molecules secondary to changes in cell metabolism and malfunction of membrane-associated pumps and ion transporters. Cellular edema then leads to an increase in tissue pressure and elevation of the ICP. TBI also causes an increase in proinflammatory cytokine production in the brain parenchyma, which leads to acute and delayed neuronal death and interference with growth factors.

Hypotension and hypoxia are major causes of secondary brain injury.[94,95] The brain requires a continuous supply of oxygen and glucose to remain functional and, unlike other organs, does not have high energy reserves. It makes up only 2% of the body weight but receives 15% to 20% of the total cardiac output. Cerebral autoregulation is the mechanism that maintains cerebral blood flow (CBF) across a wide range of cerebral perfusion pressures (CPPs), which is the difference between mean arterial pressure (MAP) and ICP.[96] Normal values for CPP range from 70 to 100 mm Hg; however, CBF remains relatively constant; even when the CPP is between 40 and 140 mm Hg[97] because of cerebral autoregulation. This autoregulation is achieved by the constant modulation of cerebral vascular resistance, in which the cerebral arteries respond to a fall in CPP by dilating and maintaining relatively constant perfusion to the brain. Although the optimal level for CPP after brain injury is debated, a CPP below 50 mm Hg is associated with brain tissue ischemia and failure of cerebral autoregulation. When this happens, CBF and oxygen delivery cannot sustain normal aerobic metabolism and compensatory brain mechanisms reach their physiologic limits. Therefore, a low CPP, which can be caused by low MAP, high ICP, or a combination of these changes, carries a significant risk for cerebral ischemia. Increases in ICP may be caused by mass lesions, cerebral edema, increased CSF production, and obstructive hydrocephalus. Arterial hypotension may be caused by acute blood loss, fever, or decreased cardiac output. The elevation of ICP is associated with poor outcomes in TBI.[98,99] ICP is normally maintained in adult humans at approximately 7 to 15 mm Hg,[100] with sustained increases in ICP above 20 mm Hg harmful to the brain because of lower CPP and tissue ischemia.

VASCULAR INJURY

Blunt cerebrovascular injuries (BCVIs) are caused by blunt trauma to the head, face, or neck. They result from high-speed deceleration to the thoracoabdominal areas, which leads to sudden hyperextension or hyperflexion of the cervical spine. Crissey and Bernstein have described the following mechanisms of injury for BCVI: (1) direct blow to the neck; (2) hyperextension with contralateral rotation of the head; (3) laceration of the artery by adjacent fractures of the sphenoid or petrous bones; (4) direct intraoral trauma.[101] The most common

mechanism is hyperextension caused by stretching of the carotid artery as it crosses over the lateral articular processes of C1 to C3.[102] The different mechanisms that lead to injury cause an intimal tear, with exposure of subendothelial collagen that leads to platelet aggregation and thrombus formation. Stenosis or occlusion may occur, as well as the development of a pseudoaneurysm from the passage of blood through the area of the intimal tear.[103] Patients may present with a latent asymptomatic period, usually of 10 to 72 hours before the onset of a stroke.[102] Practice management guidelines and algorithms have been developed for the screening and treatment of patients with BCVI (Box 8-2).

The two major types of vascular injuries after head trauma are traumatic carotid-cavernous fistulas (TCCFs) and arterial dissection. TCCFs are rare and are associated with skull base fractures.[104,105] Liang et al, in a retrospective review of 312 patients with skull base fracture, found an overall incidence of TCCF of 3.8%. The incidence was higher in middle cranial fossa fractures (8.3%) when compared with anterior (2.4%) and posterior (1.7%) cranial fossa fractures. TCCFs develop because of the abnormal communication between the carotid artery and the veins of the cavernous sinus, creating a high-pressure, high-flow fistula. The most frequent signs and symptoms include proptosis, bruit, chemosis, vision impairment, ophthalmoplegia, and facial pain. Loss of vision can occur in up to 25% of patients.[105,106] The abducens nerve is affected commonly because of its location within the cavernous sinus. The differential diagnosis for TCCF should include superior orbital fissure syndrome, orbital apex syndrome, retrobulbar hematoma, and cavernous sinus thrombosis. Angiography provides the definitive diagnosis of TCCF, although magnetic resonance (MR) angiography and CT angiography can also be used for the diagnosis. Transvascular detachable balloon embolization has become part of the treatment algorithm for TCCF.[107]

Dissection of the internal carotid artery (ICA) occurs commonly along the cervical and petrous segments of the artery. The presence of a carotid canal fracture (35%) is associated with an ICA dissection. Of patients with a traumatic ICA injury, 10% exhibit immediate symptoms, 55% experience symptoms within the first 24 hours, and 35% experience no symptoms until 24 hours after injury.[108] Patients can present with a headache, Horner's syndrome, unilateral facial weakness, or paralysis.[85,108] ICA injuries are typically diagnosed after the development of an unexpected neurologic deficit. Approximately 80% of patients will progress to cerebral infarction within 1 week after the onset of initial symptoms[108] The diagnosis of an ICA dissection can be made with CT angiography, magnetic resonance imaging (MRI), MR angiography, or catheter angiography. Diagnostically, an irregular stenosis or occlusion of the artery can be seen, as well as the presence of an intimal flap or a pseudoaneurysm.

An arteriography grading scale with therapeutic and prognostic implications has been proposed for the management of ICA injury. In this scale, the injuries are categorized into nonhemodynamically significant intimal injuries (grade I), potentially hemodynamically significant dissections and hematomas (grade II), pseudoaneurysms (grade III), occlusions (grade IV), and vessel transections (grade V).[109] On MRI, the most common finding is that of a crescent-shaped hyperintense signal indicative of an intramural hematoma and suggestive of an ICA dissection.[85] The treatment of traumatic carotid injuries is influenced by the type of injury, mechanism of injury, and neurologic findings. Anticoagulation, direct repair, and endovascular repair can be performed depending on the type of lesion present and neurologic findings.[110,111]

BOX 8-2 Screening Criteria and Risk Factors for Blunt Cerebrovascular Injury (BCVI)

WESTERN TRAUMA ASSOCIATION ALGORITHM

Patients with the following findings should undergo diagnostic evaluation for screening and diagnosis of BCVI:

- Any neurologic abnormality that is unexplained by a diagnosed injury
- Blunt trauma patient with epistaxis from suspected arterial source

Asymptomatic patients with significant blunt head trauma and the following risk factors for BCVI should also be considered for screening:

- GCS < 8
- Petrous bone fracture
- Diffuse axonal injury
- C-spine fracture, particularly those of C1-C3 and fracture through the foramen transversarium
- Cervical spine fracture with subluxation or rotational component
- LeFort II or III facial fracture

EASTERN ASSOCIATION FOR THE SURGERY OF TRAUMA GUIDELINES

Trauma patients with these signs and symptoms should undergo diagnostic evaluation for screening and diagnosis of BCVI:

- Arterial hemorrhage from neck, mouth, nose and ears
- Large or expanding cervical hematoma
- Cervical bruit in a patient < 50 yr
- Focal or lateralizing neurologic deficit; includes hemiparesis, transient ischemic attack, Horner's syndrome, oculosympathetic paresis, signs of vertebrobasilar insufficiency
- Cerebral infarction evident on CT or MRI
- Any neurologic deficit incongruous with CT or MRI findings

Patients with the following risk factors are at high risk of BCVI:

- Injury mechanism compatible with severe cervical hyperextension with rotation or hyperflexion
- LeFort II or III midface fracture
- Basilar skull fracture involving the carotid canal
- Closed head injury consistent with diffuse axonal injury; GCS score < 6
- Cervical vertebral body or transverse foramen fracture, subluxation, or ligamentous injury at any level; any fracture at level of C1-C3
- Near-hanging resulting in cerebral anoxia
- Seat belt or other clothesline-type injury with significant cervical pain, swelling, or altered mental status

SUPERIOR ORBITAL FISSURE SYNDROME

Superior orbital fissure syndrome (SOFS) was first reported by Hirschfield in 1858.[112] Traumatic SOFS is uncommon, with a reported incidence from 0.3% to 0.8% in craniomaxillofacial fractures. It can result from direct compression of displaced bone segments on the contents of the superior orbital fissure or from an indirect mechanism, such as edema or hematoma secondary to the traumatic event.[113] The superior orbital fissure is pear-shaped and is approximately 22 mm in length and 2 to 3 mm in width at its narrower part and 7 to 8 mm at its broadest portion.[113] Its width has been estimated in cadaveric studies to be 3.21 ± 1.09 mm.[114]

The clinical findings in SOFS are secondary to injury to the neurovascular structures that pass through the superior orbital fissure. These include the lacrimal nerve, frontal nerve, nasociliary nerve, oculomotor nerve, abducens nerve, trochlear nerve and superior ophthalmic vein. The clinical symptoms consist of ophthalmoplegia, ptosis, numbness along the forehead and upper eyelid, and a fixed, dilated pupil. The symptoms can be present in various degrees depending on the severity and timing of the examination in relation to the injury. In a series of 33 patients with SOFS, the most commonly associated fractures were those of the zygomatic and orbital bones. SOFS can also be caused by a carotid-cavernous fistula; when it is the causative mechanism, a better functional recovery after treatment is achieved when compared with the fracture group. Recovery of CN II, IV, and VI appears to reach a plateau approximately 6 months after injury.[115] Of the cranial nerves, the trochlear nerve appears to be the least severely injured and also has the highest level of functional recovery. This can be correlated to its anatomic position within the superior orbital fissure, where it passes above the tendinous ring.

The management of traumatic SOFS is still controversial and no definitive guidelines exist. Some studies have shown partial or complete recovery of motor and sensory function following no intervention,[116,117] leading some authors to recommend a conservative approach with steroid therapy alone.[118] Others favor surgical reduction of concomitant facial fractures to improve functional outcomes.[113,119]

SKULL FRACTURES

Cranial fractures are associated with a high incidence of intracranial lesions, neurologic compromise, and poor outcomes.[120,121] They can be classified by location in the skull vault or those involving the skull base. Vault fractures can be linear, stellate, or comminuted. They are also categorized according to the degree of bone displacement as depressed or nondepressed fractures. The most common type of vault fracture is the linear type; this is associated with the lowest incidence of intracranial injury. Despite this, the presence of a cranial fracture and loss of consciousness indicate a high risk of developing an intracranial hematoma.[122] An epidural hematoma could be present in temporal and occipital bone fractures, with injury to the middle meningeal artery or dural venous sinuses, respectively. Closed linear fractures are nonoperative lesions unless they are associated with an intracranial pathology.

Depressed fractures can be present in up to 6% of head injuries and are associated with significant morbidity and mortality because of parenchymal injury.[123] Compound or open fractures account for up to 90% of depressed fractures.[124] They are clinical indicators of significant injuries, given their 1.4% to 19% mortality rate. Depressed cranial fractures have an infection rate of 1.9% to 10.6%[124] and a 15% incidence of late epilepsy.[125] Treatment of depressed cranial fractures is aimed toward improving cosmesis, decreasing the incidence of late-onset post-traumatic epilepsy, and improving neurologic deficits. Surgical treatment of compound depressed fractures is performed as soon as possible after the injury. The wound is débrided and the displaced bone segments are elevated in an attempt to decrease the risk of infection and late epilepsy. Operative delay of longer than 48 hours for compound depressed fractures increases the infection risk to 36.5%.[94,126]

The management of the bone fragments in compound fractures has traditionally been removal after elevation and delayed cranioplasty for reconstruction of the defect. Evidence suggests that in a selected group of patients, such as those with absence of gross wound infection, the immediate replacement of the bone fragments appears not to carry an increased risk of infection.[124] Patients with compound depressed fractures can be treated nonoperatively if there are no clinical or radiographic signs of dural involvement, significant intracranial hematoma, depression greater than 1 cm, frontal sinus involvement, wound infection, gross deformity, and presence of pneumocephalus or gross wound contamination.[123,124] Closed or simple depressed fractures are treated surgically if the extent of the depression is greater than the full thickness of the adjacent intact bone or if there is an intracranial hematoma with mass effect that requires evacuation.

The skull base is comprised of five bones—the orbital plate of the frontal bone, cribriform plate of the ethmoid bone, sphenoid bone, occipital bone, and squamous and petrous portions of the temporal bone. Skull base fractures may involve the anterior, middle, or posterior fossa. In cases of high-velocity trauma, the frontal and anterior skull base regions are frequently involved. The upper cranial nerves (CN I, II, III, IV, and VI), paranasal sinuses and cavernous sinus could be injured as a result of this fracture pattern. In patients with frontobasal fractures, CSF rhinorrhea is seen in 4% to 48% of patients and pneumocephalus is seen in 10% to 50% of patients. There are several classifications of anterior skull base fractures that use the location of the injury, the force vector, or clinical findings as part of the scheme. In the classification by Manson et al,[127] frontobasal fractures are divided into three types: type I, isolated, linear, skull base fractures; type II, vertical-linear vault (frontal bone) fractures with basilar involvement; and type III, comminution of lateral and frontal vaults and orbital roof. In the Sakas et al classification, type I are cribriform plate fractures, type II are frontoethmoidal fractures, type III are lateral frontal fractures, and type IV are mixed fractures, with any combination of types I to III. In the Raveh et al classification,[128] an injury to the frontobasal region is classified into two broad categories—type 1, which consists of frontonasoethmoidal and medial orbital frame

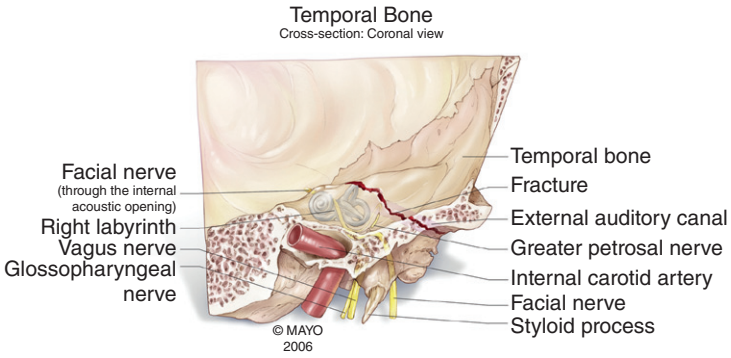


FIGURE 8-6 Longitudinal fracture of the temporal bone. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

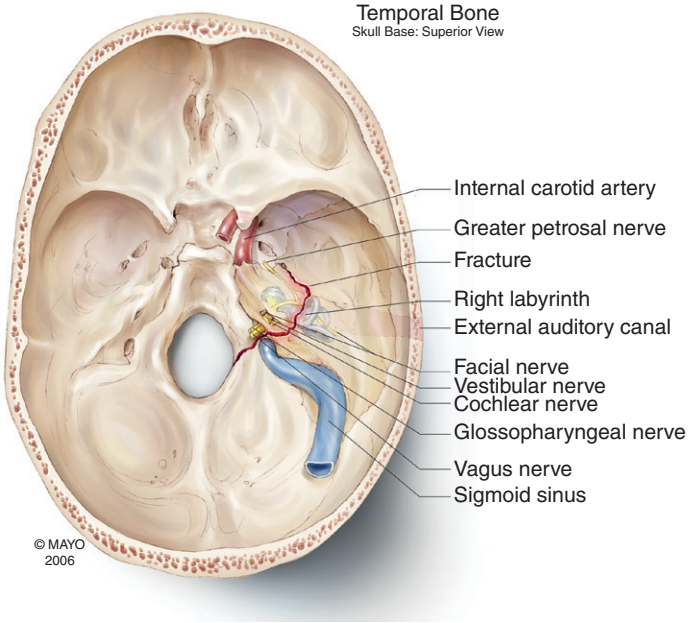


FIGURE 8-7 Transverse fracture of the temporal bone. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

fractures without skull base involvement and type 2, which consists of combined skull base, frontonasoethmoidal, and medial orbital frame fractures, with frequent optic nerve compression. Type 2 fractures in this classification are associated with significant cosmetic deformity, neurologic injury, dural tears, and intracranial pathology because of the displacement of the posterior frontal sinus wall, telescoping of the nasal pyramid, and intracranial displacement of the orbital roof and sphenoidal-parasellar regions.

Temporal bone fractures are classified according to their relationship to the long axis of the petrous pyramid. As noted, 80% of temporal bone fractures are longitudinal and occur after a direct lateral blow to the temporoparietal region (Fig. 8-6). These fractures begin in the weaker squamous portion of the temporal bone and course toward the carotid and jugular foramen. Clinical findings can include a tympanic membrane tear and conductive hearing loss secondary to middle ear ossicle disruption. Patients can also present with bleeding from the external auditory canal; approximately 25% will have a facial nerve injury at the geniculate ganglion or facial canal that leads to paresis of the muscles of facial expression on the affected side. Transverse temporal bone fractures occur after severe trauma to the occipital region (Fig. 8-7). These fractures begin in the jugular foramen and course across the petrous pyramid, through the foramen spinosum and foramen lacerum, and into the foramen magnum. Approximately 50% of patients will have facial paralysis from CN VII involvement at the internal auditory meatus or at the medial wall of the tympanic membrane. Transverse fractures involving the otic capsule and internal auditory canal can lead to severe sensorineural hearing loss. Vestibular symptoms secondary to temporal bone injury can result for various reasons, including direct otic capsule injury, labyrinth injury, brainstem or nuclei injury, and presence of a perilymphatic fistula.

Approximately 20% of skull base fractures will develop a CSF leak manifested as rhinorrhea or otorrhea; these are the result of a tear of the dura and arachnoid at the skull base. Of these, approximately 80% occur within 48 hours of the injury[129,130] and pneumocephalus is present in 30% of patients with a CSF leak.[131] In CSF leaks with a delayed onset, it is thought that lysis of a blood clot blocking the fistulous tract, an increase in ICP, or secondary trauma to the area contributes to the delayed presentation. Delayed otorrhea is a rare finding. Early post-traumatic CSF fistulas resolve spontaneously within 1 week in 70% of patients and, in 80%, they heal by 6 months. However, there is a 10% recurrence of rhinorrhea after initial spontaneous cessation. Otorrhea usually resolves spontaneously.[132,133]

A CSF fistula is not always clinically apparent. Patients can experience headaches, decreased hearing, and a salty taste. To facilitate the diagnosis of a CSF leak, a few drops of the fluid can be placed on a tissue. CSF has a more rapid diffusion than blood, leading to a larger, clearer CSF ring surrounding a sanguineous central ring. This is termed the *double-ring* or *halo sign*. Laboratory tests for CSF detection include the presence of glucose oxidase, CSF glucose levels greater than 30 mg/dL, protein content less than 2 g/L, and detection of beta-2 transferrin.

Bacterial meningitis is the primary cause of morbidity and mortality in patients with a CSF fistula. The incidence has been estimated to be from 2% to 50%.[134] The most common organism is *Pneumococcus*, followed by *Streptococcus* and *H. influenzae*.[135] In anterior cranial base fractures, the proximity to the midline (cribriform plate), fractures larger than 1cm, and prolonged rhinorrhea (lasting 8 days) are associated with an increased risk of meningitis[134] The value of antibiotic prophylaxis in patients with CSF leakage is debatable. Current evidence does not support prophylactic antibiotic use in patients with skull base fracture whether or not there is evidence of a CSF leak.[136]

In patients with a CSF leak, the head is elevated 30 degrees, fluid intake is restricted, and the patient is cautioned against blowing her or his nose because of the risk of intracranial space contamination. If a leak persists beyond 72 hours, a lumbar drain may be effective in reducing ICP and sealing the leak. Fewer than 5% of patients with traumatic CSF leaks actually require repair. Immediate surgical intervention is indicated for open wounds that communicate with the dura or by the presence of an intracranial abscess or hematoma. Delayed surgical repair is indicated for persistent or increased CSF leakage over 1 or 2 weeks, persistent or enlarging pneumocephalus, and the presence of meningitis. Endoscopic extracranial techniques have effectively replaced the intracranial approach for the surgical repair of CSF fistulae.[137] Accurate identification of the site of CSF leakage is necessary if a surgical repair is required. The most reliable methods for achieving this are high-resolution CT and MR tomography.

EPIDURAL HEMATOMA

Acute epidural hematomas (EDHs) are hemorrhagic collections in between the inner table of the skull and dura. Their incidence among traumatic brain injury is between 2.7% and 4%. The mortality rate of EDH is approximately 10%[138] and among patients in a coma, up to 9% had an EDH that required a craniotomy. The peak incidence of EDH is in the second decade of life, with it being rare in patients older than 60 years. Historically, bleeding from the middle meningeal artery was considered the main source of EDH, although it can also result from injury to the middle meningeal vein, diploic veins, and venous sinuses. EDHs are most frequently located in the temporoparietal and temporal regions[138] and are caused by a low-velocity lateral blow to the head. There appears to be a slight right-sided predominance and from 2% to 5% of patients can have bilateral EDH.[138,139]

The clinical presentation in EDH is typically of a comatose patient on admission or prior to surgery. A lucid interval, during which the patient is initially unconscious, wakes up, and proceeds to deteriorate neurologically thereafter occurs in up to 47% of patients with an EDH.[138] This could lead to a patient being discharged from the hospital with an undiagnosed EDH and who could have subsequent serious neurologic consequences from the hematoma expansion and brain edema. The presence of an altered level of consciousness is caused by cortical compression, which could proceed to transtentorial herniation, in which the patient is in a coma, with fixed dilated pupils and decerebrate posture. If not treated promptly at this stage, death will follow. Clinical presentation could also be that of seizures and hemiparesis caused by the contralateral compression of the cerebral peduncles. Associated intracranial lesions such as contusions, intracerebral hemorrhage, subdural hematomas, and diffuse brain swelling can also be found in up to 50% of patients with a surgically evacuated EDH.[138,140]

CT is the imaging modality of choice for the diagnosis of an acute EDH. It not only allows for identification of the lesion but also for determining the presence of a midline shift, traumatic subarachnoid hemorrhage, obliteration of the basal cisterns, and calculation of the thickness and volume of the hematoma. The typical CT appearance is that of a biconvex, hyperdense, extra-axial fluid collection (Fig. 8-8). An EDH typically does not cross suture lines unless there is a dural tear present. Management of EDH is dependent on the neurologic and CT findings. Surgical decompression through a craniotomy is indicated when the hematoma is larger than 30 cm^3 regardless of the GCS score. Observation in a neurosurgical unit with serial CT and serial neurologic evaluations can be done in the presence of an EDH smaller than 30 cm^3, less than 15 mm in thickness, midline shift less than 5 mm, and GCS score greater than 8, with no focal findings.[138] The patient's age, presence of pupillary abnormalities, associated intracranial lesions, time interval between neurologic deterioration and surgery, and ICP have all been identified as prognostic factors in the management of EDH.

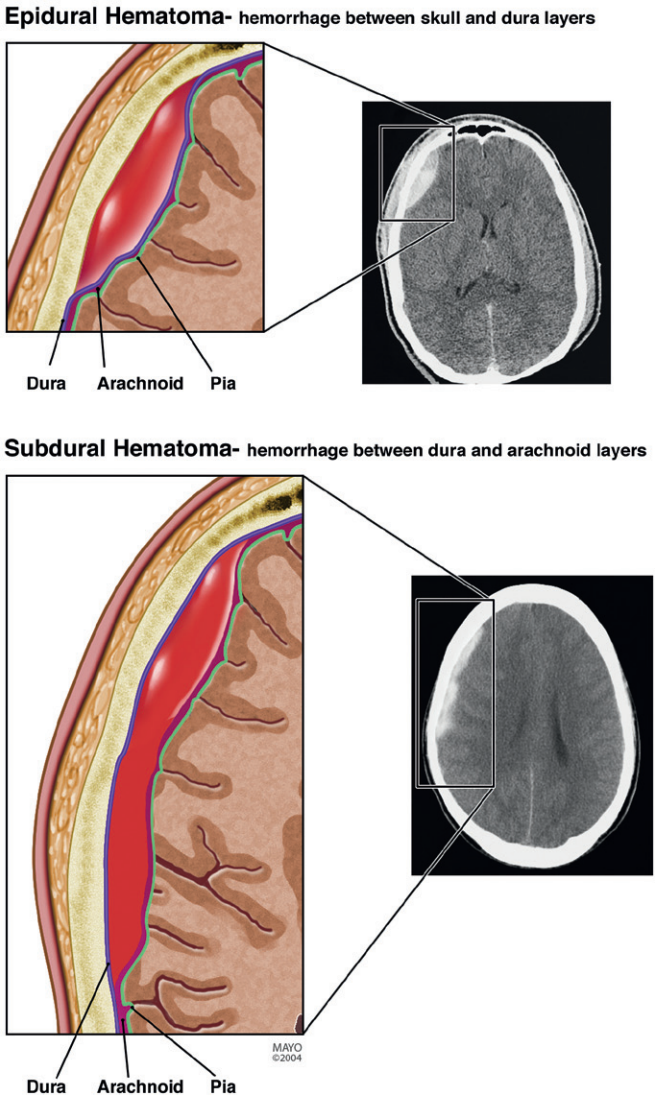


FIGURE 8-8 Hematomas—epidural *(top)* and subdural *(bottom).* (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

SUBDURAL HEMATOMA

Acute subdural hematomas (SDHs) have a 30% incidence in severely-injured patients. Their estimated mortality rate is 40% to 60%[141] in patients requiring surgery and is frequently associated with skull fractures. An acute SDH can have three different causes—a direct laceration of a cortical artery or vein with a penetrating brain injury, a large contusion and, the most common mechanism, tearing of the bridging veins of the brain to the dural sinuses. The mechanism of injury leading to an SDH differs among age groups. In young adults (18 to 40 years), most SDHs are caused by MVAs; in patients older than 65 years, falls are the predominant mechanism. In this patient population, brain atrophy leaves the bridging veins exposed to a higher risk of injury secondary to the increase in extra-axial CSF space. Acute SDHs are caused by the strain rate sensitivity of the bridging veins being exceeded, even in the setting of seemingly minor trauma and not necessarily the impact of an object contacting the head.[142] This is the phenomenon observed in shaken baby syndrome, in which the violent shaking of the head exceeds the bridging vein tolerance and leads to the development of an SDH.

There is no consensus in the literature with regard to the terms *acute*, *subacute*, and *chronic* when referring to SDH. An acute SDH is one that is typically 1 to 2 days old, whereas subacute is one that has been present for 3 to 14 days after the initial injury. The term *chronic SDH* can be used for an SDH present for 15 or more days. Up to 80% of patients[141] with an acute SDH present with a GCS score of 8 or less. Most cases of SDH are also associated with contusions and intracerebral hematomas, which can lead to a greater degree of mass effect on the brain than that which would typically be seen with an SDH alone. SDHs usually occur along the cerebral convexities, falx cerebri, and tentorium cerebelli. On CT scans, they tend to have a crescent-shaped appearance (see Fig. 8-8) because of the dural attachments and can be supratentorial or infratentorial. SDHs can also cross suture lines and be large enough to surround a cerebral hemisphere. When interpreting CT scans, intra-axial and extra-axial hemorrhages have a typical appearance and pattern of evolution. Acute to subacute hemorrhage is hyperattenuating and becomes isointense and eventually hypointense to the brain over days to weeks. Subacute, isodense, and chronic subdural hematomas may be difficult to recognize.[143] MRI is more sensitive than CT for the detection of small SDHs and also has a characteristic temporal progression that is influenced by the hemoglobin level, field strength, and oxygen tension.[143]

Surgical evacuation of an SDH is indicated when the clot thickness is more than10 mm or there is a midline shift more than 5 mm, regardless of the GCS score.[124] In patients requiring surgery, there is a poor outcome in patients older than 60 years who have a severe traumatic brain injury.[141] Evacuation of an SDH hematoma can be performed by twist drill trephination or craniostomy, burr hole trephination, craniotomy, or subtemporal decompressive craniectomy. Patients in a coma (GCS < 9), an SDH with a thickness less than 10 mm, and a midline shift less than 5 mm can be treated nonoperatively with ICP monitoring as long as they are neurologically stable, have no pupillary abnormalities, and no intracranial hypertension (ICP > 20 mm Hg).[124]

DIFFUSE AXONAL INJURY

DAI is relatively common in patients who sustain a severe closed head injury. It is the predominant mechanism of injury in 40% to 50% of patients admitted for a TBI and is the most common cause of persistent vegetative state and severe disability in TBI.[144] DAI is caused by shearing forces during rapid head accelerations that affect the white matter. The predominant sites affected are the parasagittal white matter of the cerebral cortex, corpus callosum, and pontine-mesencephalic junction adjacent to the superior cerebellar peduncles. It is assumed that DAI has occurred when there is any loss of consciousness in a trauma situation.[144,145] Depending on the severity of the injury, patients have an immediate loss of consciousness or confusion at the time of the injury that can persist as a coma or cognitive dysfunction.

DAI is caused by axonal degeneration, which represents a progression that evolves from axonal transport disruption to axonal swelling and, finally, to wallerian degeneration.[146] DAI lesions can be hemorrhagic or nonhemorrhagic. DAI is graded by its anatomic location into grade I, injuries isolated to the gray-white matter junction, grade II, lesions present in the corpus callosum, and grade III, lesions isolated to the brainstem.[147] CT is relatively insensitive for the detection of DAI, particularly if the lesions are nonhemorrhagic. Gentry et al have found that only approximately 19% of DAI lesions have an associated hemorrhage.[148] Hemorrhagic lesions appear as punctate hyperattenuating foci at the corpus callosum, gray matter junction of the cerebrum, and pontine-mesencephalic junction adjacent to the superior cerebellar peduncles on CT scans. However, only 10% of patients demonstrate these classic CT findings.[143,148] MRI allows for better identification of DAI, especially in nonhemorrhagic lesions. These lesions are hypointense on T1-weighted images and hyperintense on T2-weighted images. Blood-sensitive MR sequences (e.g., GRE T2 sequences) can detect more lesions than CT.[147] DAI can also be detected using diffusion-weighted imaging (DWI), susceptibility-weighted imaging (SWI), and diffusion-tensor imaging (DTI); tractography also has shown promise in the evaluation of DAI.[143]

A broad spectrum of cognitive deficits can be present as a result of DAI. Patients can experience difficulties with encoding and retrieving new information and processing of information; those who can speak may have well-preserved retrograde memories but are unable to recall new information. Potential therapeutic targets aimed at preventing the progression of injured axons, such as cyclosporine A and erythropoietin, have been identified and could be used in the future for the management of patients with TBI.[149]

CEREBRAL CONTUSIONS

Cerebral contusions account for approximately 40% of traumatic brain injuries.[85] These lesions are caused by injuries to capillaries, which leads to a small amount of petechial hemorrhage and edema. The most common location for contusions are the anterior and inferior

surfaces of the frontal lobes and the temporal and occipital poles.[143] These lesions result from translational movements along the floor of the anterior and middle cranial fossa. Cortical contusions can result from coup or contrecoup injuries. In a coup injury, the brain strikes the skull at the site of the direct impact; a contrecoup injury occurs opposite to the site of the external force.

Approximately 50% of contusions are hemorrhagic on CT. They typically have the appearance of round or oval hyperdense foci, with associated edema and mild mass effect.[85] Nonhemorrhagic contusions are difficult to detect initially on CT. After a few days, a hypodense lesion might be apparent as a result of delayed hemorrhage, which represents a severe injury. Hemorrhagic contusions are hypointense on T2-weighted MRI during the first few days and become hyperintense on T1-weighted and T2-weighted images. Nonhemorrhagic contusions are hypointense on T1-weighted images and hyperintense on T2-weighted images.[85,150]

Patients with progressive neurologic deterioration, intracranial hypertension refractory to medical management, or radiographic evidence of mass effect should be treated surgically. Also, patients with a frontal or temporal contusion larger than 20 cm^3 in volume, GCS score of 6 to 8, and midline shift of 5 mm or more should also be treated operatively. Surgical treatment options include craniotomy with evacuation of mass lesion, bifrontal decompressive craniectomy, subtemporal decompression, and temporal lobectomy. Nonoperative management in the intensive care unit (ICU) with serial imaging and monitoring can be done for patients who are neurologically stable, with controlled ICP, and no significant signs of mass effect.[151]

CERVICAL SPINE INJURY

Spinal cord injury (SCI) carries a high morbidity and mortality rate in addition to its emotional, financial, social, and medical impacts on the patient and family members. Most SCIs are caused by MVAs (42.1%), followed by falls (26.7%), violence (15.1%), and sports (7.6%).[152] Survival after an acute SCI is associated with age, level of injury, and neurologic grade.[153] Most patients with an SCI have a complete injury according to the American Spinal Cord Association (ASCA) grading system. It has been estimated that more than half (57.5%) of patients with an SCI are employed at the time of their injury and, at postinjury year 1, only 22% remain employed.[154] The average initial hospitalization and rehabilitation charges for a SCI have been reported to be $282,245. This is in addition to the cost of home and vehicle modifications and attendant care for assistance with activities of daily living that patients with an acute SCI may require.[155] These direct and indirect costs can lead to a medical debt burden that produces financial insolvency and ultimately bankruptcy filing.[156]

In acute SCI, the cervical spine is involved 55% of the time in comparison to 15% for each of the other regions of the spine.[157] Cervical spine injuries, including fractures and ligamentous injuries, are primarily caused by trauma to the head and neck regions. Ruling out a cervical spine injury remains an essential component of the initial evaluation of the trauma patient, given that a missed injury could have devastating consequences. This could be difficult from a clinical perspective because of swelling, anatomic distortion, or the presence of distracting injuries. Knowledge of the prevalence of cervical spine injury in relationship to facial bone fractures, head injury, and other risk factors could provide early SCI recognition and prompt management for the prevention of adverse events. The repair of a facial fracture in the presence of an occult cervical spine injury could lead to permanent neurologic injury and long-term disability. A retrospective review of the National Trauma Bank from 2002 to 2006 found that in 1,309,311 of reported patients with an injury, the incidence of facial fracture was 13.5% in patients with a C-spine injury, 21.7% in patients with a head injury, and 24.0% of patients with combined C-spine and head trauma. The possible severity of a maxillofacial and neck injury in a trauma patient is reflected by the fact that in this study, head injuries were found in 40.2% of patients with a C-spine injury and in 71.5% of patients with combined C-spine injury and facial fracture.[158]

The cervical spine is divided into an upper cervical spine and lower or subaxial spine. This reflects the anatomic and functional differences between the upper two and lower five cervical vertebrae. C1 and C2 have the most rotational mobility, with limited frontal and sagittal plane mobility, whereas the lower cervical spine allows for flexion-extension and inclination–rotation. The widest portion of the spinal canal is from C1 to C3, with a midsagittal diameter that ranges from 16 to 30 mm. There is also a safety zone between the anterior wall of the spinal canal at the level of the atlas and axis. At this level, one third of the canal is occupied by the spinal cord, one third by the odontoid process and one third is free space (safe zone), referred to as Steel's rule of thirds.[159] The diameter of the spinal canal narrows from 14 to 23 mm at C4 to C7.

Most cervical spine fractures occur at the upper or lower ends of the cervical spine.[160-162] Upper cervical spine injuries are those that involve the occipital condyles, atlanto-occipital articulation, C1, C2, and the atlantoaxial joint. C1 to C3 injuries have a high mortality rate and also show the greatest recovery after an initial complete injury in survivors when compared with thoracic and thoracolumbar injuries.[157] In older adults, falls lead to most spine injuries, with the presence of degenerative changes and osteopenia complicating treatment and outcomes.[152,163] Ligamentous injuries and fractures of the upper cervical spine are more common in children younger than 11 years, whereas adolescents more frequently sustain fractures to the lower cervical spine.[164,165]

Management of the potentially traumatized spine focuses on three principles: (1) restoration and maintenance of spinal alignment; (2) protection of the spinal cord; and (3) establishment of spinal stability.[166] Trauma patients may require an emergency airway prior to a full assessment for cervical spine injuries. Patients with a GCS less than 9 have a high risk of a spinal injury[167] and are also most likely to require an emergent airway. Manual in-line immobilization (MILI) is used to prevent head movement during intubation or any other intervention that might be required in the maxillofacial region.[166]

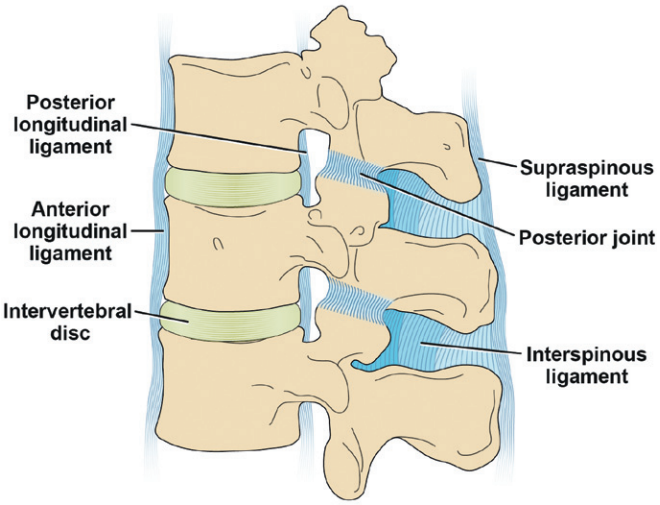


FIGURE 8-9 The three-column theory of Denis divides the spinal column into anterior, middle, and posterior columns. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

MILI is typically provided by an assistant positioned at the head or side of the bed. The patient's head and neck are maintained in neutral position. If the assistant is at the head of the bed, the mastoid process is grasped with the fingertips and the occiput is cradled in the palms of the hands. If at the side, the mastoid process is cradled and the occiput is grasped with the fingertips. When MILI is in place, the anterior portion of the cervical collar can be removed, facilitating examination and access for an airway intervention.

Mechanical stability and neurologic findings are important in determining the appropriate treatment strategy for SCIs. The three-column theory of Denis is used to predict the stability of the spine after an injury.[168] It divides the spinal column into anterior, middle, and posterior columns (Fig. 8-9). The anterior column consists of the anterior part of the vertebral body, anterior longitudinal ligament, and anterior annulus fibrosus. The middle column is formed by the posterior vertebral body, posterior longitudinal ligament, and posterior annulus fibrosus. The posterior column is composed of the posterior bony elements, which include the pedicles, lamina, facets and spinous processes. The ligamentum flavum, interspinous ligaments, supraspinous ligaments, and facet joint capsule are also part of the posterior column. A spinal injury is considered mechanically stable when only one column is involved and unstable when two columns are disrupted. An SCI could be the result of primary and secondary mechanisms, with the primary mechanism being the initial impact and compression, as seen with fracture dislocation and burst fractures.[157] The secondary mechanism of injury can be triggered by local vascular, electrolyte, or biochemical changes, apoptosis, and edema.[157]

OCCIPITAL CONDYLE FRACTURES

Occipital condyle fractures (OCFs) are difficult to diagnose on plain films. The use of computed tomography in trauma patients has led to an increase in its diagnosis, with reported incidence between 4% to 19%.[169] The clinical presentation is variable because of the proximity of the condyles to neurovascular structures. The brainstem, CN IX, X, XI, and XII, and venous and arterial vessels are at risk. The clinical presentation could be that of lower cranial nerve palsies (Collet-Sicard syndrome) in one third of patients. Two thirds of patients present with these neurologic findings acutely; in those in whom it presents in a delayed fashion, progressive compression from displaced bone segments or scar formation accounts for the delayed presentation.[169,170]

The Anderson and Montesano classification[171] is generally used for the categorization of OCFs. Type I fractures occur from excessive axial loading and can produce comminution of the occipital condyle, with no fragment displacement. Type II fractures occur as an extension of a linear skull base fracture, with the condyle maintaining its attachment to the cranial base. A condylar avulsion, which can result from excessive loading during rotation or lateral bending, leads to a type III fracture, which is considered to be unstable and could require internal fixation. Types I and II fractures are stable and isolated. Type I fractures do not need immobilization. Type II fractures with intact ligaments are treated with hard collar immobilization; in those with ligamentous disruption, a halo vest or internal fixation might be required.

ATLANTO-OCCIPITAL DISLOCATION

Atlanto-occipital dislocation is a rare injury. It is more commonly found in children because of their large cranium in relation to the facial bones.[152] It carries a high mortality rate; those who survive will have neurologic impairment, including lower cranial neuropathies, unilateral or bilateral weakness, and quadriplegia. They are highly unstable injuries; treatment involves an occipital C1-C2 fusion to prevent further neurologic compromise.[172]

ATLAS FRACTURES

Atlas fractures account for 25% of craniocervical injuries and 3% to 13% of cervical spine injuries.[173] Axis fractures are present in 40% to 44% of atlas fractures. In 1920, British neurosurgeon Sir Geoffrey Jefferson presented a series of patients with atlas fractures. The following classification is based on that description and was later modified by Gehweiler et al.[174] Type I fractures involve the posterior arch alone and, in type II, the anterior arch is involved. Type III fractures are bilateral posterior arch fractures associated with a unilateral or bilateral anterior arch fracture (Fig. 8-10). Type IV fractures involve the lateral mass and type V fractures are transverse fractures of the anterior arch.

The classic Jefferson fracture is a type III burst fracture caused by severe axial compression. Patients with an atlas fracture could present with neck pain, muscle spasms, limited neck motion, difficulty swallowing secondary to retropharyngeal swelling, and CN IX, X, XI, and XII involvement. However, in the classic Jefferson fracture, neurologic deficits are not common because of the expansion of the spinal canal as a result of the injury.

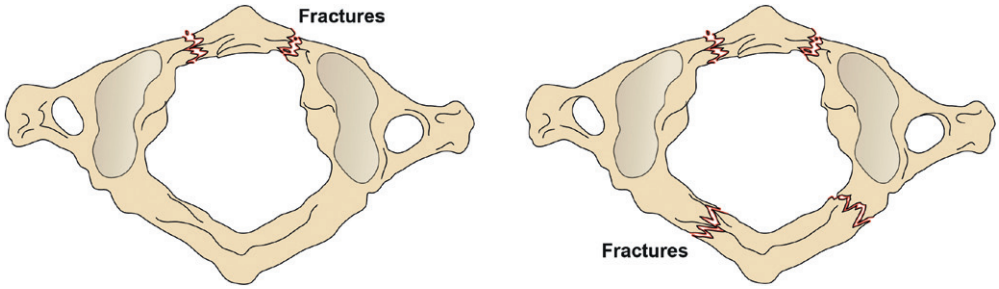


FIGURE 8-10 Atlas fracture types. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

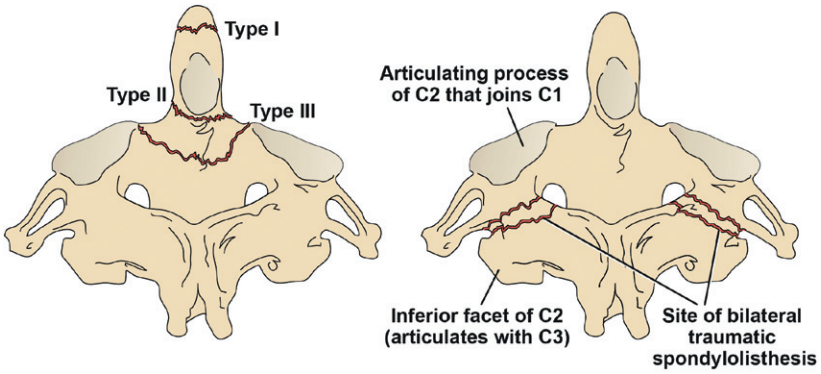


FIGURE 8-11 Dens fracture types and bilateral fractures of the pars interarticularis (hangman's fracture). (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

Atlas fractures are considered to be stable or unstable, depending on the integrity of the transverse ligament, which is crucial in atlantoaxial stability. Atlas fractures are treated with halo brace immobilization or surgical fixation, depending on whether they are present in isolation or in conjunction with other cervical spine injuries and on the integrity of the transverse ligament.

AXIS FRACTURES

Axis fractures account for approximately 20% of cervical spine fractures and have an estimated 8.5% rate of neurologic deficit.[175] There are three types of axis fractures—odontoid fractures involving the dens, bilateral traumatic spondylolisthesis of the pars interarticularis (termed a *hangman's fracture*), and nonodontoid, non–hangman's fractures.

The prevalence of dens fractures ranges from 10% to 15%.[172,176,177] The classification of odontoid (dens) fractures is based on that of Anderson and D'Alonzo.[178] Type I fractures are rare and involve an oblique fracture of the tip of the dens. Type II odontoid fractures are the most common type of axis fracture and also the most difficult to treat. They occur at the junction between the odontoid process and the vertebral body. They have the highest rate of nonunion, from 5% to 63%.[172] Type III fractures extend into the vertebral body and have a high fusion rate with the use of rigid bracing (Fig. 8-11). Odontoid fractures are a common pediatric cervical spine injury, with rare neurologic problems.[172,176,179] Overall, the treatment strategy of odontoid fractures is based on the fracture type, degree of displacement, age of the patient, transverse ligament integrity, and angle of the fracture line. The treatment options include the use of cervical collar or traction, halo device immobilization, and anterior and posterior surgical approaches with wiring fixation, Halifax clamps, and screw fixation. Older patients treated with halo immobilization have a high rate of nonunion and complications.

Bilateral fractures of the pars interarticularis of the axis was first described in 1866 by Haughton in criminals executed by hanging[180] (see Fig. 8-11). Since then, these fractures have been known as a hangman's fracture, fracture of the neural arch, fracture of the ring of the axis, and traumatic spondylolisthesis.[172] The incidence of neurologic findings with this injury is low, given the spacious canal space at this level.[172,181,182] Clinical presentation is typically that of neck pain. Most patients with a hangman's fracture are treated with halo immobilization for approximately 12 weeks.[172,181]

LOWER CERVICAL SPINE FRACTURES

There are several methods to classify subaxial cervical spine injuries; they are typically categorized depending on the mechanism of injury. The subaxial cervical spine injury classification system uses the injury pattern, integrity of the discoligamentous complex, and neurologic status of the patient to categorize the injury and guide treatment strategies.[183] The treatment of subaxial cervical spine trauma is based on a number of variables, including fracture pattern, mechanism of injury, spinal alignment, neurologic injury, and expected long-term stability.

The clay shoveler fracture (Fig. 8-12) is an oblique avulsive fracture of the lower cervical or thoracic spinous process. It is a hyperflexion injury whose name came from laborers who sustained this injury when shoveling soil, rubble, or snow over their head. Currently, it is not a common fracture and is more likely to occur in the trauma setting. The clay shoveler fracture is a stable fracture, although it may be painful. In most patients, immobilization of the neck by means of a collar and restriction

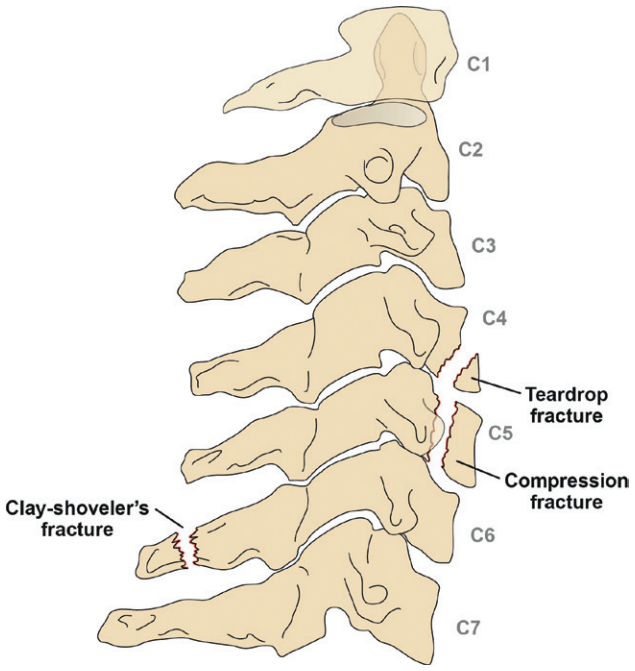


FIGURE 8-12 Clay shoveler fracture, compression fracture, and flexion teardrop fracture. (By permission of Mayo Foundation for Medical Education and Research. All rights reserved.)

of physical activity for 4 to 6 weeks frequently result in pain relief.

The flexion teardrop is a severe injury most commonly occurring at the C5 level.[152] There is complete disruption of all the soft tissues at the level of the injury and the impacted vertebral body fractures at the anterior and inferior aspects (see Fig. 8-12). There is disruption of the posterior ligament and the posteriorly displaced vertebral body impinges on the spinal cord. Patients present with an acute anterior cord syndrome, manifested as quadriplegia and loss of pain and temperature sensation.

Bilateral interfacetal dislocation (BID) is an extreme form of hyperflexion injury that occurs following a severe flexion force to the head and neck. This leads to significant anterior displacement of the spine and ligamentous disruption at the level of the injury. These are unstable injuries with major disruption of all supporting ligaments and, at the subluxed level, the inferior facets dislocate superiorly and anteriorly to the superior articulating facets of the lower vertebrae. BIDs are more common in the lower cervical spine.

Extension injuries occur because posterior displacement of the head, usually from trauma to the face or sudden deceleration. These injuries tend to affect the lower cervical spine. Extension teardrop fractures generally involve the C2 body. These fractures only affect the anterior column, and therefore are stable in flexion and unstable in extension. A hyperextension fracture dislocation more commonly occurs in older patients.

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CHAPTER

9

Abdominal Trauma: Evaluation and Management

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OUTLINE

Epidemiology
Mechanisms of Injury
Examination
Trauma Scores and Indices
Adjuncts To Physical Examination
Diagnostic Workup
Laboratory Studies
Imaging Studies
Diagnostic Peritoneal Lavage
Rigid Sigmoidoscopy
Diagnostic Laparoscopy
Laparotomy
Evaluation of Penetrating injuries
Blunt Abdominal Injury
Hollow Organ Injury
Abdominal Vascular Injury
Solid Organ Injury
Pelvic Injuries
Pediatric Abdominal Trauma
Occult Abdominal Trauma
Abdominal Trauma in Pregnancy
SUMMARY

Abdominal trauma assessment and management is a significant part of trauma management. Primary survey and circulation both involve an abdominal examination for determining hemodynamic stability. Thus, maxillofacial trauma surgeons must be cognizant of concomitant abdominal injuries and be proficient in the diagnosis, prioritization of acute management, and application of abdominal trauma management principles.

Many algorithms and treatment protocols have been devised over the years to assess and treat individuals with multisystem traumatic injuries. The most widely accepted and used worldwide is the Advanced Trauma Life Support (ATLS) system developed by the American College of Surgeons Committee on Trauma. This begins with an initial assessment or primary survey of the trauma patient comprised of the familiar ABCDE—***a***irway, ***b***reathing, ***c***irculation, ***d***isability, and ***e***xposure.

Abdominal examination is an adjunct to primary survey in hemodynamically unstable patients forming part of the assessment of circulation, specifically to confirm or exclude the abdomen as the source of concealed bleeding. In a stable patient with a history of chest or abdominal injury or symptoms, the abdominal examination is part of a secondary and tertiary survey.[1] Facial or closed head injury examination is secondary in the evaluation unless significant airway instability impedes proper access and control of ventilation. Any wound from the nipple to perineum anteriorly, to the vertebral column posteriorly, and bilateral flanks are the boundaries of abdominal review and the common areas of trauma. Mechanisms of injuries may be penetrating or blunt. The most common cause of mortality in abdominal trauma is secondary to delayed resuscitation or excessive hemorrhage with inadequate volume resuscitation. Intra-abdominal organ injury and rupture or perforation precipitates gastrointestinal (GI) content spillage into the peritoneal cavity, frequently leading to peritonitis and delayed mortality from severe sepsis.

The abdomen can be deceptively benign on initial assessment, with a precipitous change in clinical status. The most significant pitfall is delayed recognition of occult abdominal injury.[2]

EPIDEMIOLOGY

Injury is the fifth leading cause of death in the United States and is the leading cause of death for those in their first 3 decades of life. Abdominal trauma is commonly encountered in these cases. The United States, South Africa, and some South American countries have a high incidence of penetrating injuries from stab and gunshot wounds. In the United Kingdom, Australia, New Zealand, and most European countries, blunt trauma predominates as the cause for hospital admissions, although the incidence of penetrating trauma is rising.

MECHANISMS OF INJURY

A concentrated area of force on the abdominal area has a higher risk of injury to underlying organs (Fig. 9-1). The impact can be in the form of a blunt object such as a steering wheel or car door. The vector and velocity of the trauma become important to the clinician. To understand the mechanism of injury better, the treating physician should seek to obtain information from the injury report or from the paramedics. Often, the transporting medical personnel have obtained the accounts from witness information as well as assessment from the injury scene. This information forms a vital component of the clinical history and the likelihood of underlying injury.

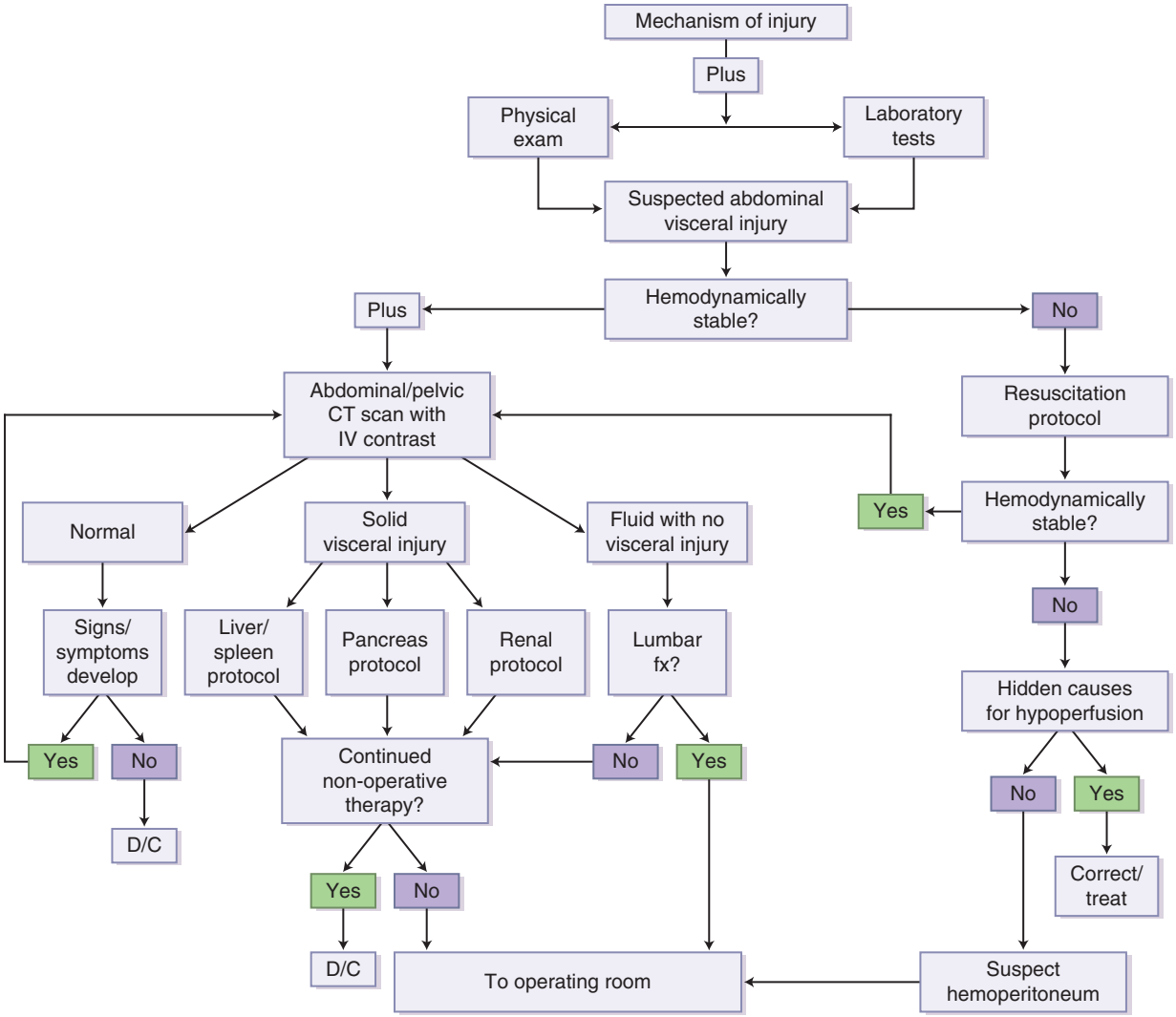


FIGURE 9-1 Mechanism of injury algorithm.

EXAMINATION

The abdomen is divided into three parts for consideration of injury—retroperitoneum and peritoneal and pelvic cavities. The pelvic cavity is comprised of the rectum, bladder, iliac, and uterine structures.

Abdominal examination in trauma must be a continuum of evaluative and diagnostic steps rather than a brief physical review of the patient. Any patient with chest and abdomen complaints is assumed to have intra-abdominal injury until proven otherwise.[3]

Undress and expose the patient from chest to perineum. Inspect and note the size, shape, and injury type: abrasions, lacerations, point of entry and exit of penetrating wounds, and evisceration of structures and organs. Gentle palpation of the abdomen may suffice in most cases, including an examination for pelvic stability. Urethral, perineal, and rectal examinations are mandatory in abdominal review. Any induration of the abdominal wall or underlying organs is a notable finding and needs further diagnostic workup. Auscultation findings may be nonspecific unless followed by further examination.

A rectal examination is invaluable, because the presence of frank blood frequently indicates anorectal disruption; prostrate position and sphincter tone on voluntary squeeze by the patient yields critical neurologic information. Similarly, inspection of the urethral meatus may indicate underlying pelvic, bladder, and urethral trauma.

TRAUMA SCORES AND INDICES

Trauma grading systems are comparative indices for quantification and description. These can be extrapolated for diagnostic, therapeutic, and prognostic measures. Commonly used indices are the Abbreviated Injury Scale (AIS) and the Penetrating Abdominal Trauma Index (PATI).[4,5] Previous editions of AIS (severity scale from 1 to 6) were for blunt trauma; the most current modification extends to include penetrating trauma. PATI has individual organ scores summative to a score of 25. Its major drawbacks are inadequate quantification for multiple complex injuries involving a specific anatomic area.

ADJUNCTS TO PHYSICAL EXAMINATION

Most adjunctive examinations are not performed in a stable, alert, cooperative, and asymptomatic patient who has suffered low-force transfer to the abdominal wall or internal organs. Any complaints of shoulder or abdominal pain, nausea, unrestrained patients, impairment by alcohol or drugs, visible contusive signs, and open injuries mandate abdominal examination adjuncts.[3]

Adjunctive Diagnostic Studies

Gastric Tubes. Gastric air is common in trauma, requiring decompression with nasogastric or orogastric tubes. Gastric tube insertion is a diagnostic and therapeutic procedure if no contraindication exists. Craniofacial injuries with unstable upper midfacial fractures are particular situations in which nasogastric tube insertion is contraindicated.

Urinary Catheters. Urinary catheters are useful for decompression of the bladder, urinalysis for macroscopic and microscopic hematuria, and monitoring adequacy of fluid resuscitation. Scrotal hematoma, perineal ecchymosis, pelvic fractures, blood at the meatus, high-riding prostrate with unstable pelvis, hematuria (>50 red blood cells [RBCs] per high-power field [HPF]) and inability to void are relative contraindications to urethral catheterization. In these cases, retrograde urethrography (RUG) is often used to diagnose urethral injury.[6]

DIAGNOSTIC WORKUP

LABORATORY STUDIES

Along with clinical examination, concurrent hematology and biochemical laboratory tests are required. These include blood typing and cross-matching for unstable patients as well as operative candidates, with a direct communication to the blood bank. Hematocrit is a worthy evaluation for ongoing management. A leukocyte count will not be specific but a significant increase is noted in solid organ injuries caused by catecholamine-induced demargination. The leukocyte count may not be an absolute essential for acute trauma management. Serum amylase and lipase levels are not specific but elevated levels or serial escalation may suggest pancreatic injury, as well as peripancreatic trauma. Correlation with imaging is mandatory. Base deficit is a frequently underused biochemical test for abdominal trauma resuscitation. Liver function tests, lactate levels, coagulation studies, creatinine kinase, and toxicology screens are also useful. Urinalysis with gross hematuria confirming renal damage, microscopic hematuria of less than 50 RBCs/HPF, or a visual inspection of blood-tinged urine is a good clinical indicator of urogenital injury.

IMAGING STUDIES

Plain Radiography

Anteroposterior (AP) chest and pelvic radiographs are standard initial assessments of patients with multisystem blunt trauma. If the patient is unstable, no radiographs are needed in the emergency room.[1,3] A radiographic marker on entry wounds of a penetrating injury may aid in determining the trajectory and path of missiles.

Focused Assessment Sonography in Trauma: Ultrasonography

Focused assessment sonography in trauma (FAST)[7] is a well-established trauma assessment by ultrasound of the cardiac, bilateral renal, and pelvic area that has been validated in several prospective randomized studies. FAST is operator-dependent but has great sensitivity, specificity, and accuracy comparable to diagnostic peritoneal lavage (DPL) and computed tomography (CT) for assessing intra-abdominal fluid and involves no radiation exposure or contrast administration. The advantages of FAST are immediate bedside intra-abdominal visualization, its noninvasive portable nature, avoidance of transport to radiology, and ability of repeat examinations to monitor interval changes. It is performed to document fluid in the pericardial sac, hepatorenal space (Morrison's pouch), splenorenal fossa, and pelvis (pouch of Douglas). Negative FAST results do not preclude the need for further evaluations with further CT imaging.[8] Equivocal studies should prompt immediate evaluation with contrast-enhanced CT for determination of solid organ injury. Disadvantages of FAST are poor sensitivity in pediatric patients, interference because of bowel gas and increased adiposity, uncooperative patients, and ascites (Box 9-1).

BOX 9-1 Focused Assessment Sonography in Trauma Procedure

1. A nasogastric tube is inserted before the examination, if needed, but the urinary catheter is withheld so that the distended bladder provides an acoustic window for visualization of blood in the pelvis.
2. Using a 3.5-MHz transducer, FAST surveys for blood in the pericardial sac and three dependent abdominal regions, including Morison's pouch, splenorenal recess, and pelvis.
3. Ultrasound transmission gel is applied to the pericardial area, right and left upper quadrants, and pelvis. (The pericardial area is visualized first, so that blood in the heart can be used as a standard to set the gain appropriately for the detection of hemoperitoneum and hemopericardium.)
4. The transducer is oriented for sagittal sections and positioned in the subxiphoid region to identify the heart and examine for blood in the pericardial sac.
5. The transducer is placed in the right midaxillary line between the 11th and 12th ribs to identify the sagittal section of the liver, kidney, and diaphragm.
6. With the transducer positioned in the left posterior axillary line between the ninth and tenth ribs, the spleen and kidney are visualized.
7. The transducer is directed for a transverse section and placed 4 cm superior to the symphysis pubis. It is swept inferiorly to obtain a coronal view of the full bladder and both sides of the pelvis.

From Rozycki GS, Root HD: The diagnosis of intra-abdominal visceral injury. *J Trauma* 68:1019–1023, 2010.

Contrast Studies

Simple contrast studies can be performed in the acute trauma setting to determine structural defects. Any contrast material can cause artifacts with CT imaging, which must be accounted for if CT is also indicated for other injuries. Upper gastrointestinal series can be performed in patients by swallowing or instilling water-soluble contrast and taking interval plain x-ray films or carrying out fluoroscopic studies. Esophageal, gastric, duodenal perforation, hematoma, and delayed transit of contrast into the distal small bowel can indicate injury. Extravasation patterns may indicate spasm and hematoma and define the anatomic location (e.g., intraperitoneal or extraperitoneal, proximal or distal defect). Air contrast can be used if insufflation is safe and performed by instilling air via nasogastric tube and noting any free subdiaphragmatic peritoneal or retroperitoneal air from proximal duodenal perforation.

Using endoscopic retrograde cholangiopancreatographic techniques, distal pancreatic duct injuries can be visualized by instilling contrast. This is indicated for possible pancreatic injuries in which the pancreatic head and duodenum are intact.

Cystography and RUG are invaluable bedside diagnostic procedures for pelvic and suspected urethral injuries—for example, in the setting of hematuria, blood at the meatus, or a differential prostrate examination. Instilling contrast material with controlled pressure, and interval before and postvoid plain x-rays can reveal disruptions in the bladder and urethra.[5] An intravenous pyelogram (IVP) confirms renal parenchymal, pelvic, calyceal, and ureteric integrity in the presence of hematuria and truncal trauma. Cystography reveals fine bladder detail better than IVP. If CT is indicated, IVP is redundant. IV contrast of 50 to 100 mL with plain film prior to laparotomy in penetrating injuries with hematuria yields detailed evaluation in the acute setting.

Computed Tomography

The first published study was by Federle et al in 1982 for blunt abdominal trauma. CT scanning, with its speed and resolution in a stable patient, has been a reference test for abdominal trauma. Intra-abdominal fluid visualization, solid parenchymal injuries, sites of active bleeding, retroperitoneal, and vertebral column injuries are well visualized by abdominothoracic CT. For the polytrauma patient, a single visit to the scanner can be combined with C-spine, head, chest, abdomen, and pelvis. The necessity of oral contrast is never a consideration but IV contrast enhances organ visualization. Contraindications include hemodynamically unstable patients in whom rapid deterioration may occur while they are in the scanner. These patients progress to immediate surgical exploration. Radiation and IV contrast have their inherent morbidity. Other significant limitations of CT include poor sensitivity for the visualization of superficial, mesenteric, diaphragmatic, and pancreatic injuries.[1]

DIAGNOSTIC PERITONEAL LAVAGE

DPL aspiration instead of a full lavage is also used in some institutions.[8] If a hemodynamically unstable patient has no gross blood aspirate, further diagnostic consideration needs to be applied to determine other possible causes. First described by Rozycki and Root, DPL is an invasive but highly sensitive test for the identification of intraperitoneal hemorrhage.[9] DPL involves a controlled infusion of 1 liter of warm normal saline (10 to 15 mL/kg for children) through a needle inserted intraperitoneally by a Seldinger technique. A gross aspirate of blood-stained fluid with a count of more than 100,000 RBCs/mm^3 is a positive examination for blunt abdominal injury. There is debate in penetrating abdominal injuries regarding the number of microscopic RBCs seen in the aspirate that indicate positivity. Patients with pelvic injuries, ascites, or retroperitoneal hemorrhage and hemodynamically unstable patients are all situations in which DPL has limited usefulness. CT has replaced DPL in many institutions. Retroperitoneal hemorrhage cannot be assessed by this intraperitoneal sampling technique—hence, the superiority of CT (Box 9-2).

BOX 9-2 Steps in the Performance of FAST

1. The urinary bladder and stomach are decompressed.
2. A vertical incision is made approximately 2 cm above or below the umbilicus, down through the fascia, until the peritoneum is identified.
3. The peritoneum is grasped with two hemostats, pulled upward gently, and a purse-string suture of 3-0 chromic catgut is placed.
4. The peritoneum is incised and an adult peritoneal dialysis catheter (without the stylet) is passed downward into the pelvis.
5. The purse-string suture is tightened and tied, and the fascia is closed around the catheter with permanent or semipermanent sutures.
6. Aspiration of > 10mL of blood, GI contents, vegetable fibers, or bile through the lavage catheter is considered a positive finding.
7. If < 10 mL of blood is aspirated, a lavage is performed by instilling 1 liter of warm crystalloid solution (10 mL/kg in children).
8. Recovery of the fluid is facilitated by siphon drainage after placing the bag on the floor.
9. The catheter is removed and the effluent (minimum, 300 mL) is sent for analysis. A positive test is indicated by >100,000 RBCs/mm^3, ≥500 white blood cells/mm^3, or a Gram stain with bacteria.

RIGID SIGMOIDOSCOPY

Penetrating or ballistic injuries to the gluteal areas crossing the midline can cause undetected extraperitoneal or rectal injuries. These may also be undetected during rectal examination; thus, careful rigid sigmoidoscopy is helpful in evaluating the extraperitoneal rectum with careful air insufflation. The limitations of this technique are poor visualization because of blood and fecal contents.

TABLE 9-1 Incidence of Penetrating Injuries

	PENETRATING INJURY	
Blunt Injury	**Stab Wound**	**Missile Wound**
Spleen, 40%-55%		
Liver, 35%-45%	Liver, 40%	Small bowel, 50%
Small bowel, 5%-10%	Small bowel, 30%	Colon, 40%
Retroperitoneal, 15%	Diaphragm, 20%	Liver, 30%
	Colon, 15%	Abdominal vascular, 25%

DIAGNOSTIC LAPAROSCOPY

Diagnostic laparoscopy is an essential surgical tool for patients with blunt and penetrating trauma. Experienced surgeons and meticulous surgical laparoscopic technique can obtain a less invasive view of the intraperitoneal contents, diaphragm, hepatic surfaces, and bowel condition.[10] It could resolve ambiguity of any peritoneal process and diaphragm injury with direct visualization and prevent further unnecessary imaging or exploratory intervention. Laparoscopy has its limitations—the hemoperitoneum can obscure the view and there could be small perforations of the bowel, retroperitoneal injuries, and a state of shock because venous return to the heart is impaired secondary to abdominal insufflation.

LAPAROTOMY

Laparotomy is a definitive surgical diagnostic and therapeutic maneuver in acute abdominal trauma. Intra-abdominal bleeding, unexplained refractory hypotension in an acute setting, an unexplained fall in the hematocrit value, progressive peritonitis, and confirmed organ injury are indications for laparotomy. Any suspicion of intra-abdominal bleeding in blunt and penetrating injuries by FAST, DPL, or CT is an absolute indication for laparotomy, because failure to explore can have life-threatening consequences.[4] Evidence of peritoneal cavity perforation by a missile in gunshot and penetrating wounds also warrants immediate surgical exploration.

Indications for Laparotomy

These include the following[3,9]:

1. Blunt abdominal trauma with hypotension and positive FAST or clinical evidence of intraperitoneal bleeding
2. Blunt trauma causing subdiaphragmatic or retroperitoneal air
3. Blunt abdominal trauma with positive DPL
4. Penetrating abdominal wound with hypotension
5. Penetrating injury causing bleeding from the stomach, rectum, or genitourinary (GU) tract
6. Gunshot wounds traversing the peritoneal cavity or visceral or vascular retroperitoneum
7. Evisceration
8. Unabating peritonitis following trauma
9. Progressive abdominal distress over 6 to 12 hours
10. GI or GU structure rupture in the intraperitoneal or extraperitoneal space or disruption by blunt or penetrating injury (Table 9-1)

EVALUATION OF PENETRATING INJURIES

Transient cavitation causes more severe organ injury from high-velocity missiles compared with tissue laceration from stabbing and low-velocity injuries. Of gunshot injuries, 90% cause intraperitoneal injury compared with 30% from stab wounds. Current ATLS practice can lead to underestimation of the significant damage of low-velocity, blunt, and high-velocity energy transfer to the viscera. Serial examination and local wound exploration are options prior to an extensive workup. DPL or CT can be used to evaluate anterior abdominal stab wounds. Contrast CT with a double- or triple-contrast medium is used for flank and back injuries.[4] The mainstay of treatment is exploratory laparotomy after astute rapid diagnosis in gunshot wounds; stabbing injuries can be conservatively managed in stable patients in whom imaging has excluded underlying visceral trauma and occult hemorrhage. Less than one third of stab wounds involving the abdomen penetrate the fascia; thus, an experienced surgeon may choose local exploration.[11] Any fascial penetration or left-sided thoracoabdominal gunshot wounds warrant laparotomy. Serial examination, chest x-ray, thoracoscopy, laparoscopy, and CT can be used for the evaluation of right-sided thoracoabdominal wounds to identify diaphragmatic hernias.

BLUNT ABDOMINAL INJURY

Trauma can be a combination of blunt and penetrating wounds in blast injuries. The management approach is the same. Blunt trauma results from crushing or shearing forces. The compressive forces of the fall or from the impact of automobile parts can cause rupture and secondary hemorrhage. Shearing forces of the lap belt or shoulder harness can often cause crush injuries because of improper fit. If, on examination a seat belt sign is noted, blunt injury is a consideration. With ligamental attachment of solid organs and their nonattached free parts subject to differential velocity impact, bleeding and organ disruption are common. Air bags can also cause abdominal injury, along with facial injuries. The spleen is the most commonly injured solid organ. Solid viscous injury is also a predictor of major hollow viscous injury in blunt abdominal trauma.[12]

HOLLOW ORGAN INJURY

Hollow abdominal organ injury is comparatively less common than solid abdominal organ injury. Common hollow organ injuries include a compressive injury against the duodenum and upper intestinal structures, such as the jejunal and ileal components, which may be compressed against the vertebral column. Colon perforation may also occur and, if missed, will lead to significant morbidity and mortality.[4] Stomach injury is less common compared with other parts of the intestine and esophagus. Proximal GI injuries are more conservatively managed. However, the injury with the highest morbidity

and potential for misdiagnosis is a duodenal injury. Missed intestinal injuries can cause externalization of the intraluminal microflora, which would manifest as peritonitis. Uninfected urinary bladder perforations can be managed conservatively. Urinary bladder injuries are commonly caused by pelvic ramus compressions and blunt injury against a distended bladder. The presenting symptoms of a bladder injury are lower abdominal pain, hematuria, azotemia from urine reabsorption, and inability to void. A common CT finding is pelvic fluid and further imaging with cystography will confirm this. A flame configuration is noted because of extraperitoneal extravasation. After contrast infiltrates the paravesical tissues, bladder base and neck injury need to be evaluated further. Intraperitoneal injuries need to be further evaluated by a urologist and usually are serious. Any finding of bony fragments in the bladder or pelvic area is to be considered serious if noted on imaging.[6] Urinary extravasation can be intraperitoneal or extraperitoneal, and drainage by a urinary catheter may allow healing of the ruptured bladder. Percutaneous drainage of extravasated urine can be performed. Surgical repair is considered an elective procedure, if necessary. Urethral injuries are diagnosed with RUG and confirmed by contrast extravasation.

ABDOMINAL VASCULAR INJURY

Blunt or penetrating abdominal trauma can cause hemorrhage from the viscera, mesentery artery, or major abdominal vessels. Exploration should only be considered with impending bowel ischemia and expanding hematomas (Fig. 9-2) Pancreaticoduodenal hematoma from blunt injury is explored, whereas stable hematomas in retroperitoneal, pelvic, perirenal, and retrohepatic sites are monitored. In penetrating injuries other than stable retrohepatic hematomas, all other sites are explored. Angiographic studies are indicated with color flow Doppler in vascular injuries of concern.[3]

Abdominal vascular injury, according to Feliciano et al, refers to injury to major intraperitoneal or retroperitoneal vessels.[4] These are classified as follows:

Zone 1: Midline to retroperitoneum; supramesocolic region; suprarenal abdominal aorta, celiac axis, proximal superior mesenteric artery, proximal renal artery, superior mesenteric vein

Zone 2: Upper lateral retroperitoneum renal artery and vein

Zone 3: Pelvic retroperitoneum iliac artery and vein

These injuries progress because of a delay in intervention and surgical access. Survival rates in penetrating abdominal aortic injuries are dismal, especially for suprarenal injury. Blunt injury to the suprarenal aorta is rare.[13]

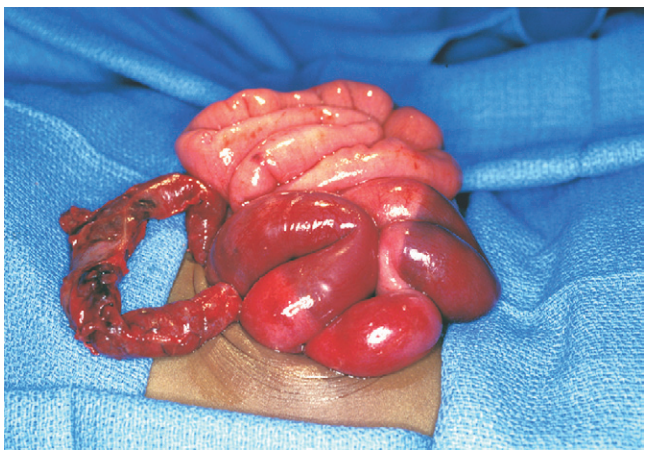


FIGURE 9-2 Bowel ischemia with vascular and mesenteric inury is noted with blunt abdominal injury

SOLID ORGAN INJURY

Liver, kidney, pancreatic, and splenic injuries can cause shock and hemodynamic instability and may necessitate urgent laparotomy. Conservative management has a role in stable patients, but they require close observation, repeated evaluation, and a low threshold for surgical exploration if deterioration is noted.

Spleen Injuries

The adult spleen is less pliable than the pediatric spleen. The presence of Kehr's sign, a rare presentation, should increase suspicion for splenic injury. Additional intra-abdominal injury in patients with splenic injury can be seen on CT scan, particularly in the bowel, pancreas, and left hemidiaphragm. Even though most splenic injuries are nonoperative, significant numbers require surgical or angiographic intervention and splenectomy. Morbidity and mortality from continued occult hemorrhage from splenic injury remain high.[14] Coagulopathy must be corrected prior to exploratory surgery and abdominal closure. A patient's status postsplenectomy or with nonfunctional splenic remnants requires the administration of prophylactic immunization against capsular organisms such as *Streptococcus pneumoniae*, *Haemophilus influenzae*, and meningococcus. Although splenic remnants survive and autotransplantation is no longer indicated, disrupted splenic vascularity results in loss of the spleen's immune function. Antimicrobial prophylaxis and immunization status in splenectomy patients should be noted because any sepsis or unexplained illness may result in overwhelming postsplenectomy infection (OPSI), which has significant mortality. Further information regarding the spleen is discussed later, in the pediatric section (Tables 9-2 and 9-3).

Older age, injury severity at presentation, and admitting hospital trauma designation are among many considerations in regard to nonoperative outcomes of blunt splenic injuries.[15] Rest and noncontact activity, with clinical and hematocrit monitoring, are undertaken in conservative management. These extrapolations are made for renal and nonpediatric injuries.[16] For example, a grade II splenic laceration is managed conservatively, with 3 days of strict bed rest and 3 weeks of noncontact activity.

Duodenal Injuries

Rupture and wall hematomas are seen with drivers not using seat belts and direct falls onto and blows to the abdomen. Frontal impact by automobile and bicycle handlebars impaling the abdomen are common presenting histories. Bloody gastric aspirate, retroperitoneal air, and contrast leak on upper GI series are frequent pre-

TABLE 9-2 American Association for the Surgery of Trauma Organ Injury Scale: Spleen

Grade	Injury Type	Injury Description
I	Hematoma Laceration	<10% area subcapsular <1 cm depth
II	Hematoma Laceration	10%-50% area subcapsular; <5 cm intraparenchymal, 1-3 cm depth, not involving trabecular vessel
III	Hematoma Laceration	>50% area or ruptured subcapsular; ruptured intraparenchymal; ≥5 cm intraparenchymal >3 cm depth or involving trabecular vessels
IV	Laceration	Segmental or hilar vessels with >25% devascularization
V	Laceration Vascular	Shattered spleen Hilar injury with devascularization

Adapted from American Association for the Surgery of Trauma: Spleen injury scale, 1994 revision (http://www.aast.org/Library/TraumaTools/InjuryScoringScales.aspx#spleen).

TABLE 9-3 American Association for the Surgery of Trauma Organ Injury Scale: Liver

Grade	Injury type	Injury description
I	Hematoma Laceration	<10% area subcapsular <1 cm depth
II	Hematoma Laceration	10%-50% area subcapsular; <10 cm intraparenchymal 1-3 cm depth, <10 cm length
III	Hematoma Laceration	>50% area or ruptured subcapsular; ruptured intraparenchymal; 10 cm intraparenchymal >3cm depth
IV	Laceration	Lobar disruption 25%-75% or 1-3 Couinaud's segments
V	Laceration Vascular	Lobar disruption >75% or >3 Couinaud's segments Juxtahepatic venous injury.

Adapted from American Association for the Surgery of Trauma: Liver injury scale, 1994 revision (http://www.aast.org/Library/TraumaTools/InjuryScoringScales.aspx#spleen).

sentations on imaging. CT with double contrast is indicated for detail.

Pancreatic Injuries

Frontal direct epigastric blows to the abdomen compress the pancreas against the vertebral column. One elevated pancreatic enzyme level seen immediately postinjury is not a criterion for diagnosis unless followed in series. Even double-contrast CT may not identify immediate significant pancreatic injury in 40% of patients. Repeat scans and subtle changes in peripancreatic areas on magnetic resonance imaging (MRI), magnetic resonance cholangiopancreatography (MRCP), and endoscopic retrograde cholangiopancreatography (ERCP) are helpful adjuncts to diagnosis. Surgical exploration is mandated if suspicion of injury persists. Various concerns exist in surgical management of pancreatic trauma because undiagnosed and unresolved pancreatic injuries carry a high fatality rate.

Urethral Injuries

Urethral injury and disruption is seen with anterior pelvic fractures. Saddle and isolated injuries cause anterior urethral disruption below the urogenital diaphragm. Posterior injury or injury above the urogenital diaphragm occurs in multisystem injuries and pelvic fractures.[3]

Diaphragm Injuries

The left posterolateral diaphragm is the most common site of diaphragm injury. An initial x-ray can be normal, with subsequent development of a diaphragmatic hernia. As for all abdominal trauma, reassessment cannot be overemphasized. Radiographic presentation varies from a normal blurred hemothorax to gastric loops in chest fields.

PELVIC INJURIES

Pelvic injuries can result from falls, crushing injuries, and ligamentous disruptions. Pelvic injuries always suggest significant impact.[1] Typically, pedestrians or motorists are involved. The impact forces are often significant and may cause bony, solid organ, or vascular injuries of the peritoneal structures. Pelvic fractures may be described as open or closed in anteroposterior or transverse directions. In the acute setting, pelvic injuries may lead to hypotension, shock, displacement of pelvic structures with disruption of the urethra, and arteriovenous structures.[17] Edema, ecchymosis, and laceration at the flanks may be other signs of pelvic injury. Rectal examination can detect a high-riding prostrate, blood, and sphincter tone. A pelvic x-ray is part of an initial trauma workup adjunct.

In shock, prior to insertion of a urinary catheter for urine output monitoring, any urethral disruption must be excluded. Urgent urologic consultation is necessary if there is renovascular injury or disruption of perineal structures. As noted earlier, ultrasound augments pelvic x-ray in the initial evaluation. A carefully performed RUG can diagnose any urethral injury. In most series, 85% to 95% of patients have gross hematuria with pelvic fractures and approximately 10% of all patients with a pelvic fracture have bladder injuries. Microscopic hematuria with more than 30 RBCs/HPF can be seen with most injuries and contusions. Further investigation is recommended for any positive finding.[18]

In pelvic ring disruptions, external pelvic stabilization by binders is an effective and simple technique for pelvic circumferential compression. A pelvic belt encircling the hips and greater trochanters reduces bleeding until external fixation devices can be applied in the operating room. Clamp devices are used in emergency conditions to apply transverse compression on the sacroiliac joints

by implanting percutaneous pins at the coccyx and sacroiliac joint. Angiographic embolization and surgical exploration are alternative considerations for hemorrhage control.

Damage control surgery by packing the pelvis, together with parietal closure, can be performed while waiting for arterial embolization, which requires monitored transfer, arterial access, and specialists.[17] Embolization is sequential, with review of any macrovascular lesions, missing arteries, wall irregularities, and contrast extravasation. In descending order of frequency, the superior gluteal, lateral sacral, iliolumbar, obturator, vesical, and inferior gluteal arteries are the most frequently involved. Nonselective embolization is also carried out in cases of hemodynamic instability and failure to identify specific arterial lesions.



FIGURE 9-3 Seat belt mark necessitates further careful evaluation and low threshold for surgical intervention.

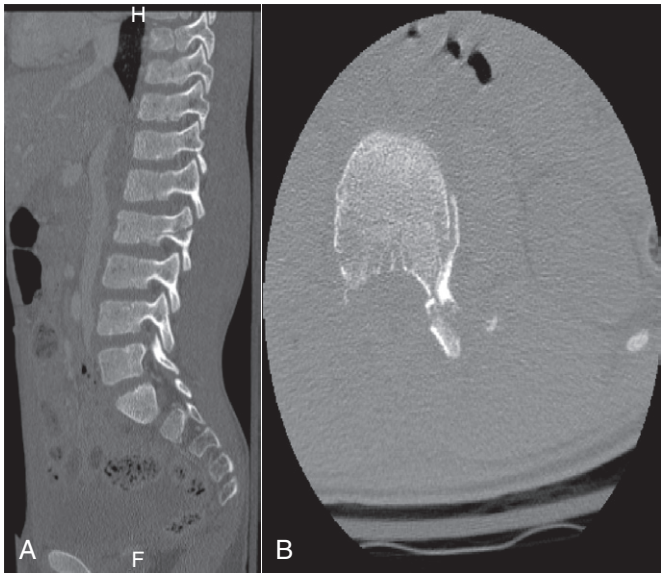


FIGURE 9-4 **A, B,** Chance fractures are noted with a lap seat belt and improperly worn shoulder restraint. Flexion of the thoracolumbar spine after a head-on collision can lead to fracture of the spine and laceration and contusion of the pancreas, duodenum, and mesentry.

PEDIATRIC ABDOMINAL TRAUMA

The abdominal examination following trauma is the same for children as for adults; however, a gentler technique is required. Decompression by orogastric tubes and bladder contents could aid in the examination because children tend to swallow air while crying. Findings of a seat belt sign and/or hemodynamic instability are important signs of the magnitude of injury (Fig. 9-3). The splenic capsule is thicker and the parenchymal consistency is firmer in children, unlike the adult spleen (Figs. 9-4 to 9-6). Thus, children are often managed without surgery. The anatomy and physiology in the pediatric population differs from that of the adult. The diaphragm is lower, so the pattern of liver and bladder injury is different. Also, frequent lower spine injury is noted.[19]

The presence of free fluid on a CT scan in children should not always be an indication for laparotomy because the incidence of intra-abdominal injury is only 3%.[3] FAST is increasingly used, but the amount of intraperitoneal blood is not a proportional guide to operative intervention. The DPL technique and interpretation

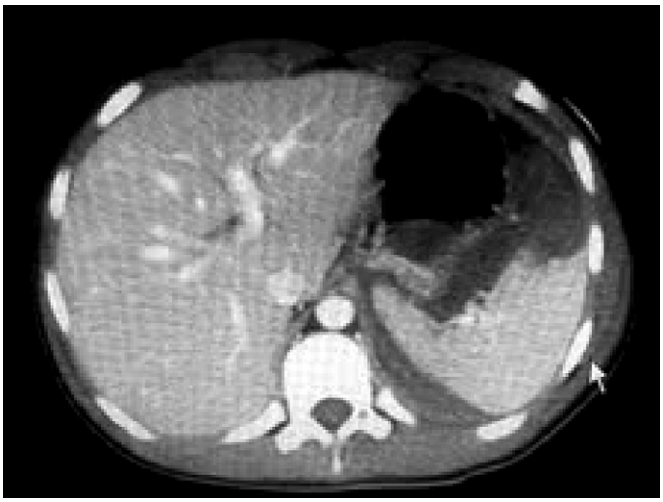


FIGURE 9-5 Splenic laceration with capsule disruption *(arrow).*

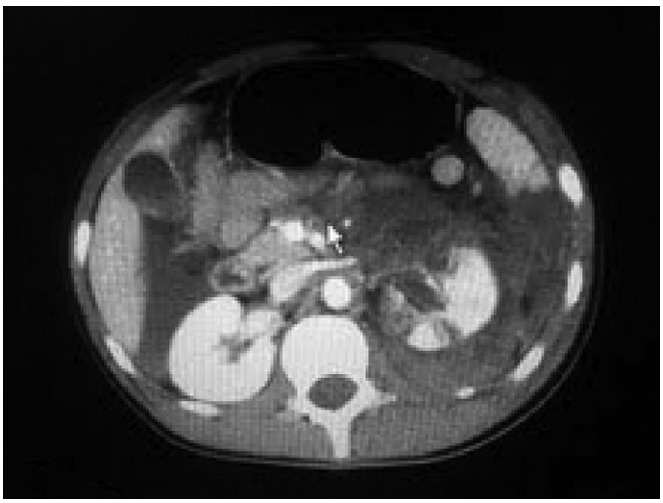


FIGURE 9-6 Left renal laceration.

similar to those for adults but should be performed by pediatric surgeons because interpretation further affects operative intervention. Nonoperative management is a frequent treatment option in blunt abdominal trauma, especially with blunt solid organ injury. Guidelines of bed rest and avoiding contact activity have been extended to the adult population from various pediatric trauma studies.[16]

OCCULT ABDOMINAL TRAUMA

Head trauma, bruising, abrasions, burns, falls, and fractures are common presentations of occult abdominal trauma (OAT) from abusive physical injuries. Abusive abdominal trauma has been found to have significant morbidity in various studies. As a facial injury is reviewed, clinicians should have a suspicion regarding unexplained falls, delayed attention, and a changing history of the injury and screen for occult abdominal trauma. Liver transaminase levels are more specific in screening than pancreatic enzyme levels, urine occult blood (dipstick and microscopic analysis), or hemoccult blood.[20] Imaging with ultrasound and CT can be used to diagnose visceral lacerations and injuries. Most of these injuries may be conservatively treated.

ABDOMINAL TRAUMA IN PREGNANCY

Trauma is a leading cause of nonobstetric maternal morbidity and mortality.[21] Pregnancy does not predispose to trauma or mortality but alters the pattern of injury. Advanced gestation makes the gravida more prone to abdominal trauma. Unrestrained motor vehicle accident (MVA) victims have higher maternal and fetal mortality rates.[22] Diagnostic studies, even though limited, have similar indications as for nonpregnant patients. Ultrasound also helps in fetal heart rate confirmation and determination of gestational age and placental condition. The uterus should be shielded, with the greatest fetal risk at 2 to 15 weeks of gestation. Hypotension, respiratory failure, and head and neck trauma cause maternal morbidity, even though these are uncommon in blunt abdominal injuries. Injuries to the spleen, liver, and cephalad bladder with pelvic injury are common blunt abdominal injuries. Gunshot and knife wounds are more common penetrating injuries. These cause preterm delivery, rupture of membranes, abruption, and hemorrhage. The patients present with hypotension, abdominal pain, back pain, vaginal bleeding, and uterine contractions. Uterine rupture is rare but, if it occurs, can cause fetal mortality and intra-abdominal hemorrhage, necessitating emergent surgical management. All Rh-negative pregnant abdominal trauma victims should receive appropriate Rh immune globulin within 72 hours. Upper abdominal injury in advanced pregnancy is critical because of upward peritoneal content displacement. Laparotomy is typically performed but, in stable patients, observant management is done after appropriate imaging. DPL is challenging in advanced pregnancies. Exploratory laparotomy is not an indication for cesarean delivery unless direct penetrating trauma has occurred, causing fetal death. Maternal stability takes precedence and physiologic changes of pregnancy have to be considered in management.

SUMMARY

Abdominal trauma may often occur concurrent with maxillofacial trauma; hence, a thorough understanding of relevant clinical findings, investigation, and management approaches of these injuries is vital to every maxillofacial surgeon. A thorough understanding of ATLS principles and their application is imperative, allowing for rapid systematic evaluation and triaging of trauma patients, as well as avoiding misdiagnoses that could lead to unnecessary significant morbidity and mortality.

Serial clinical abdominal examinations and a high index of suspicion are the key to abdominal trauma management. Any change with expectant trends must be managed with indicated imaging or surgical intervention. Trauma units, which can manage facial and abdominal injuries, are preferred for a multisystem injury patient. Delayed manifestations, as late as 36 to 48 hours with hemorrhagic shock and sepsis, should always be a consideration and emergency trauma care should be available. Urine output and vital signs are essential, with ongoing monitoring of trauma resuscitation, as is repeated clinical examination. Prolonged ileus frequently follows abdominal trauma and prolonged NPO status may be indicated. Nutritional support is initiated as part of early trauma diagnosis in the first 48 hours, because facial and abdominal injury can prevent normal intake of diet and the increased caloric demands of trauma can lead to a negative caloric balance. Nasoenteric feeding aids, gastrojejunostomy, and central venous hyperalimentation are frequently used. Enteral alimentation is preferred to other forms. Deep vein thrombosis prevention is vital because trauma patients are deemed at high risk for this, but actual data on prophylactic measures are scarce.

Massive transfusion protocols and early anesthesiology involvement, monitoring and managing hypothermia, and cardiac perfusion parameters are key to successful trauma management. Therapists and trauma nurses with a systems-based approach to error reduction can have a huge impact on missed injuries, as well as monitoring dressings, ambulation, and review of patients on prolonged bed rest with spleen and liver injuries. There is ample evidence that the trauma level designation of the surgical unit has a positive impact on outcome.

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PART THREE

Management of Head and Neck Injuries

CHAPTER 10

Applied Surgical Anatomy of the Head and Neck

Janelle E.K. Meuten | Katharine Powers | David E. Frost | Barry D. Kendell

OUTLINE

Skin Lines and Lines of Langer
Scalp
Skin of the Face
Osteology
Midface
Lower Face
Mandibular Surgical Approaches
Extraoral Surgical Approaches
Risdon and Submandibular Approaches
Intraoral Surgical Approaches
Angle
Parasymphysis and Body
Muscles
Muscles of Facial Expression
Muscles of Mastication
Suprahyoid Muscles
Infrahyoid Muscles
Soft Palate Musculature
Pharyngeal Musculature
Arterial Blood Supply to the Head and Neck
External Carotid Artery
Veins of the Head and Neck
Internal Jugular Vein
Common Facial Vein
Anterior Facial Vein
Retromandibular Vein
External Jugular Vein
Anterior Jugular Vein
Neurologic Anatomy
Trigeminal Nerve
Facial Nerve
Hypoglossal Nerve
Regional Anatomy
Orbital Anatomy
Orbital Nerves
Nasal Anatomy
External Nasal Anatomy
Nasal Cavity Anatomy
Parotid Region
Parotid Gland
Submandibular Gland
Floor of the Mouth

Because traumatic injuries disrupt the anatomy, the surgeon who is to repair and replace these traumatized structures must have an in-depth knowledge of normal anatomy. In addition, the operator must consider possible variations of normal and other associated structures that may be in close relationship to the traumatized area. Although numerous texts have been written on basic anatomy,[1-11] it is thought that for completeness, this textbook should include a review of major head and neck anatomy. Details on specific problems and treatment modalities are found in the appropriate chapters. It is the intent of this chapter to discuss the general anatomy, its inherent relationships, and some technical problem areas that should be considered in the management of traumatic facial injuries.

SKIN LINES AND LINES OF LANGER

The natural skin lines and wrinkles are major factors in determining the final soft tissue aesthetic result for the patient with facial trauma. The character and aesthetics of a scar are affected by its relationship to the location and direction of normal skin lines.

The natural skin lines and wrinkle lines are different from the lines of Langer, which denote the direction of the collagen fibers within the dermis. Langer believed that the skin was less extensible in the direction of the lines of tension that cross them.[2,12] In the face, Langer's lines have been shown to run across natural creases and flexion lines, thus making scars generated by incisions and trauma along those lines more noticeable. It is therefore recommended that elective incisions be made in or parallel to the lines of facial expression or the natural skin lines, when possible (Fig. 10-1).[13] Skin wrinkles provide an excellent template for areas of relaxed skin tension. Old scar lines and hairlines can also direct a surgeon to the most appropriate placement of an incision. Considerations as the ethnicity of the patient must also be taken into account because hyperpigmentation and hypopigmentation issues might arise.



FIGURE 10-1 Natural skin lines and wrinkle lines are recommended for elective incisions.

SCALP

The scalp is made up of five layers, three of which are closely bound together. These are the skin, dense connective tissue, and galea aponeurotica. Beneath these layers are the loose connective tissue and the periosteum or pericranial layer.[1,6,8] The scalp bleeds freely because the vessels are bound firmly in the dense connective tissue layer (Fig. 10-2). This firm union and the extensive blood supply frequently make bleeding excessive and often difficult to control rapidly with hemostats. Pressure usually controls the open bleeders, and the rapid application of Raney's clips controls full-thickness lacerations or

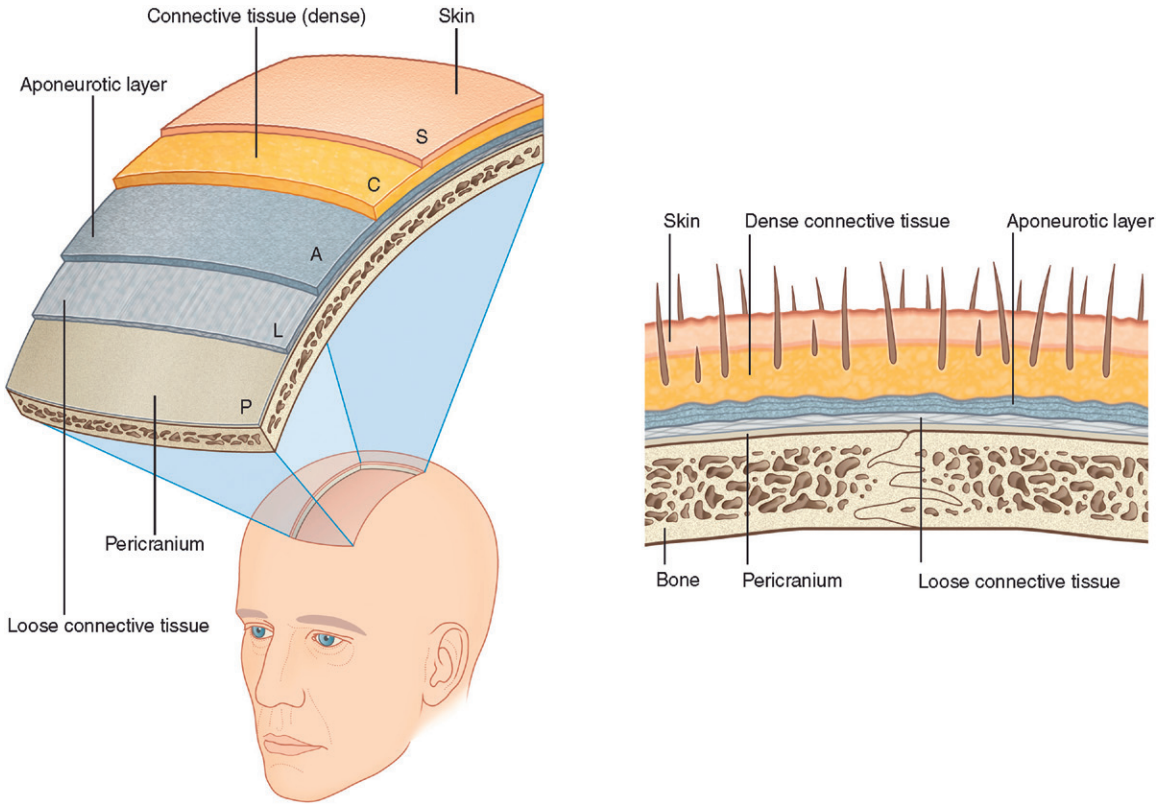


FIGURE 10-2 Layers of scalp. (From Aehlert B: *Paramedic practice today*, St. Louis, 2010, Mosby/JEMS.)

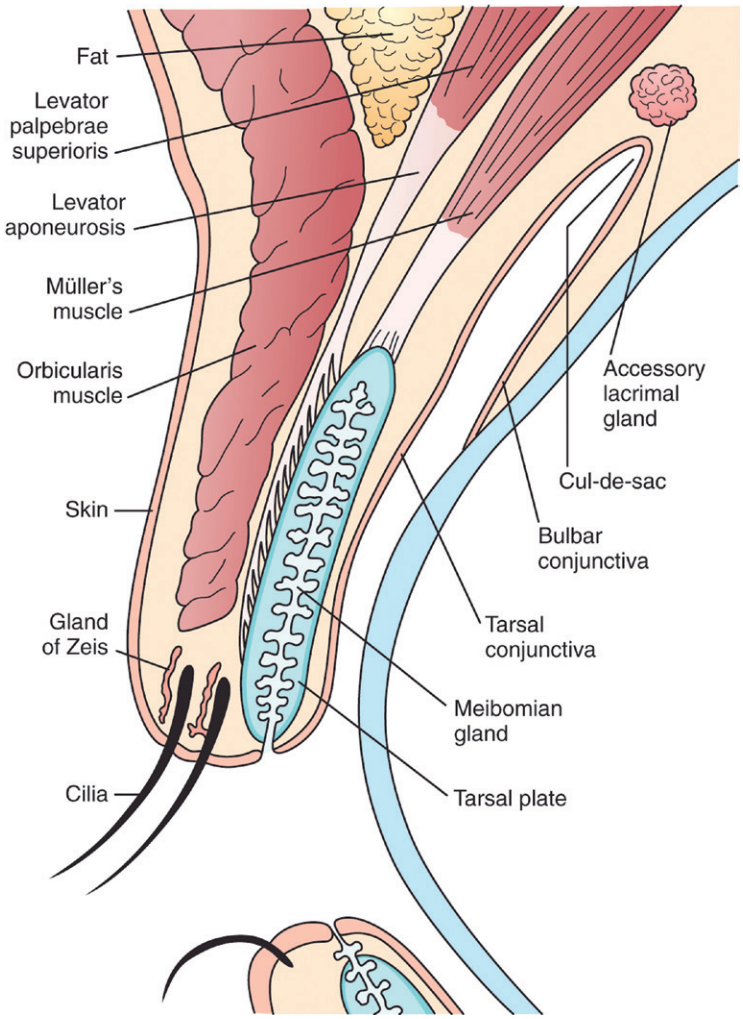


FIGURE 10-3 Cross-section of eyelid. (From Zitelli B, McIntire SC, Nowalk AJ: *Atlas of pediatric physical diagnosis,* ed 6, Philadelphia, 2012, Mosby.)

elective incisions. Because of the nature of the loose connective tissue layer, dissection of the scalp is rather easy in this tissue plane. In a similar manner, however, the effusion of fluid spreads rapidly in this plane, leading to a boglike edema.

The innervation of the scalp comes from the trigeminal nerve anteriorly and laterally and from the cervical nerves (C2 and C3) posteriorly.[1,4] If dissection is kept within the loose connective tissue layer, these nerves are avoided. In the supraorbital region, the superior orbital branch of the trigeminal nerve passes through a notch or foramen to innervate this area of the scalp. The supratrochlear nerve is located slightly medially and innervates the upper lid and the medial area of the forehead.[14] Care should be taken when elevating flaps and managing lacerations in this area. As with most areas of the anatomy, when the skeleton makes angles or muscles insert, there is a denser attachment of the skin and soft tissue. In the scalp, this attachment is most notable in the glabella and supraorbital regions.

SKIN OF THE FACE

The skin of the face becomes specialized in the area of the eyelids, which are comprised of two structural lamellae: the external lamellae formed by the orbicularis muscle and its overlying skin and the internal lamellae of the tarsal plate and conjunctiva.[15] The skin of the eyelid is extremely thin and delicate and contains small lacrimal, sweat, and sebaceous glands and hair follicles (Fig. 10-3).[16]

The skin of the nose is tightly attached to the lower lateral cartilage in the tip area. In other areas, the skin is less tightly adhered to the underlying infrastructure. The skin is thin in the nasal root and tip areas and thicker in the supratip region.[4]

OSTEOLOGY

The bones will be considered in the traditional facial thirds (Fig. 10-4).[3,5]

MIDFACE

The maxilla, zygoma, lacrimal, nasal, palatine, inferior nasal concha, and vomer bones are collectively referred to as the middle third of the facial skeleton.[4,17,18] Although the sphenoid, frontal, and ethmoid bones are not classically facial skeleton bones, they are frequently traumatized in midfacial fractures and thus should be considered in the midfacial skeleton. The bones will be discussed

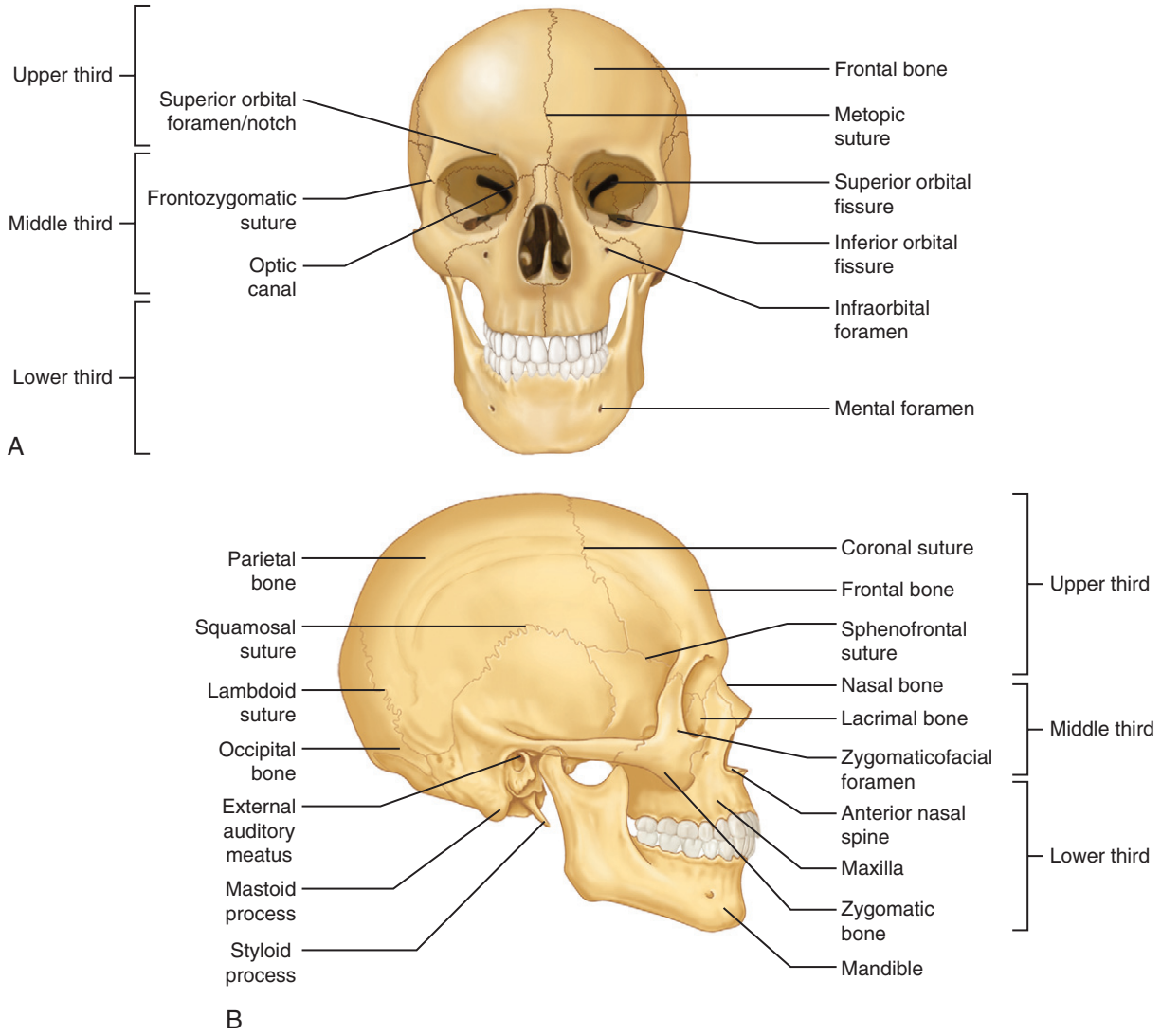


FIGURE 10-4 **A,** Frontal view of skull. **B,** Lateral view of skull. Facial thirds are noted.

separately, but their interconnections are of utmost importance.

Maxilla

The maxilla (Figs. 10-5 and 10-6) is a paired bone of the upper jaw,* fused to form one bone, and is the central focus of the middle third of the face. Each hemimaxilla contains a large pyramid-shaped body, the maxillary sinus (antrum of Highmore), and four prominent processes—the frontal, alveolar, zygomatic, and palatine processes.

The body of the maxilla is hollow and contains the maxillary sinus. The anterior wall of the sinus is the facial surface of the maxilla and is usually thin. The medial wall is the lateral nasal wall. The sinus opens superiorly and medially into the nasal cavity at the semilunar hiatus in the middle meatus. The superior wall or roof of the sinus is the orbital floor, and the floor of the sinus is the palatine and alveolar processes of the maxilla.

The frontal process arises from the anteromedial corner of the body and articulates with the frontal bone to form the medial orbital rim. The medial portion of the frontal process fuses with the nasal bone and may therefore be termed the *nasofrontal process*. Posteriorly, the process articulates with the lacrimal bone to form the anterior portion of the medial orbital wall. This area of articulation with the frontal bone, nasal bone, and lacrimal bone is prominent in the facial skeleton and is frequently fractured by blunt trauma.

The inferiorly extending portion of the maxilla is the alveolar process, which contains the maxillary teeth. The teeth are key to the accurate management of many midfacial fractures. The alveolar process may be fractured by direct trauma and therefore may be functionally separate from other portions of the maxilla.

The palatine process arises horizontally from the lower edge of the medial surface of the body. It joins the opposite process and forms the major portion of the hard palate.

*References 1, 2, 4, 6, 8, 10, and 19.

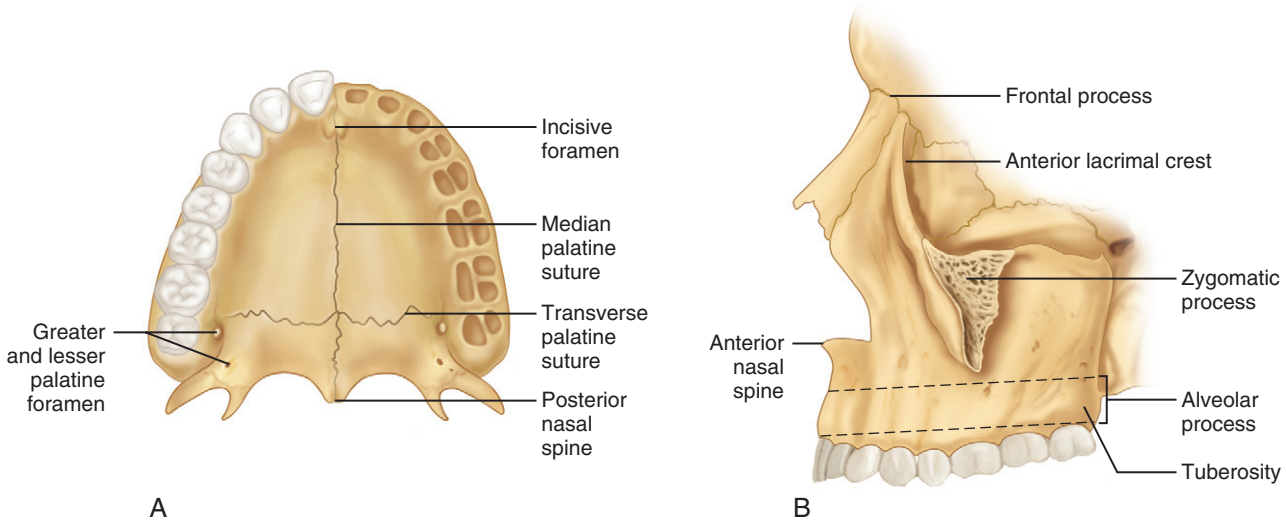


FIGURE 10-5 **A,** Maxilla and horizontal portion of palatine bone. **B,** Lateral aspect of maxilla.

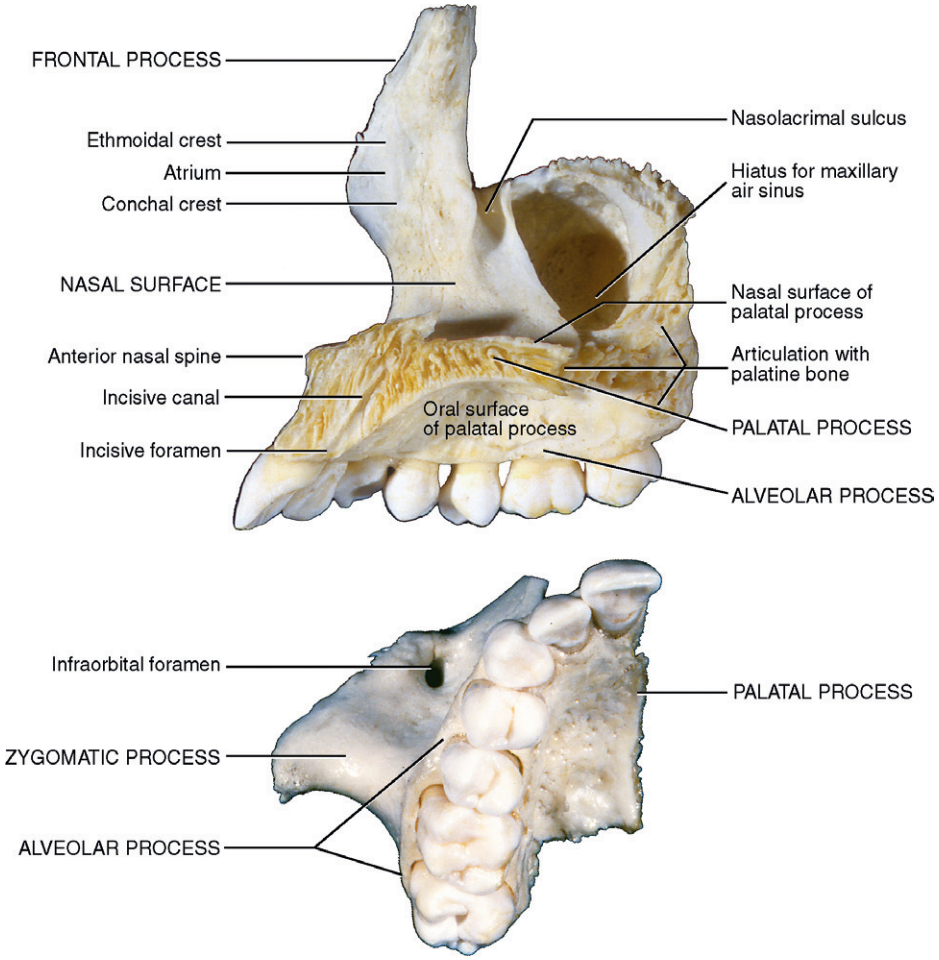


FIGURE 10-6 Medial aspect of maxilla and palatine bones. (From Liebgott B: *The anatomical basis of dentistry,* ed 3, St. Louis, 2009, Mosby.)

The zygomatic process of the maxilla arises from the anterolateral corner of the maxilla and articulates laterally with the zygoma. Together, they form the inferior orbital rim and the greatest portion of the orbital floor. The infraorbital foramen is on the anterior surface of the zygomatic process of the maxilla.

Surgical Note: The classic Le Fort I fracture passes through the anterior wall of the maxilla, extending posteriorly to the pterygoid plates. It is important to remember that this is a paired bone and, even though it is fused in the midline, in adults it behaves like two separate bones when manipulated. It may often be separated

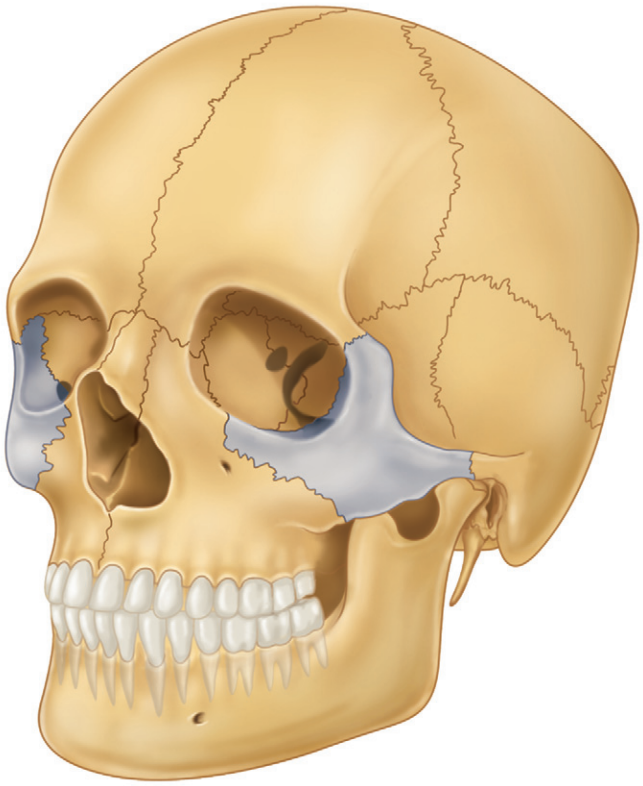


FIGURE 10-7 Zygoma (malar) showing articulations.

along the midpalatal suture in the more extreme facial fractures.

Zygoma

The zygoma (zygomatic bone, malar bone) is a paired bone that makes up the essence of the cheek prominence (Fig. 10-7; see also Fig. 10-4).* This thick, strong, diamond-shaped bone forms the lateral and anterior projections to the midface and is composed of four processes. The frontal process forms the lateral orbital wall and articulates with the frontal bone at the frontozygomatic suture. It is this articulation that is separated or rotated in isolated zygomatic fractures. The temporal process forms the zygomatic arch and articulates with the temporal bone. The maxillary process articulates with the maxilla to form the infraorbital rim and part of the floor of the orbit. Finally, the fourth process joins the maxilla on the lateral wall, producing the zygomatic eminence. This is an area of thickened bone that is usually available for fixation in the treatment of zygomaticomaxillary complex (ZMC) fractures. Along the crest of the zygoma, on the inferior aspect, is the insertion of the masseter muscle. The direction of force for this muscle is down and backward and its contraction contributes to displacement of the complex fracture of the zygoma, which may precipitate redisplacement in the improperly fixated fracture.

The zygoma articulates with the sphenoid bone on the posterior aspect of the frontal process. This articulation is with the greater wing of the sphenoid bone and forms the lateral wall of the orbit.

The only foramina of the zygomatic bone are the zygomaticofacial foramen, which opens from the orbital surface of the bone and passes to the eminence, and the zygomaticotemporal foramen, which opens to the infratemporal fossa. The zygomaticofacial and zygomaticotemporal branches of the second division of the trigeminal nerve pass from within the orbit to the surface and give sensory innervation to the associated structures.

Surgical Note: The classic zygomaticomaxillary fracture (tripod, zygomaticomaxillary complex, and trimalar) involves this bone and its articulations. Details on the fracture and its management are found in Chapter 16. The options of surgical manipulation for reduction are discussed in the following sections.

Temporal Fossa Approach. The Gillies approach is made via an incision in the hair-bearing area of the scalp, approximately 2 cm above and 1 cm anterior to the ear.[14,20-23] This incision is carried down to the level of the temporalis fascia. The only structure of anatomic importance is the superficial temporal artery. This vessel courses across the area of the incision and can be identified through palpation and thus avoided by the properly placed incision. If it is encountered, it can be ligated and cut without complications. The temporalis fascia runs to the arch of the zygoma. An incision is made through this fascia to expose the muscle and develop a path for the passage of instruments to manipulate the zygoma. The main anatomic concern is being too superficial to the fascia, thus introducing the elevator lateral to the arch.

Lateral Brow Approach. The lateral brow incision* is made to allow easy access to the frontozygomatic suture, because this area frequently requires open inspection, manipulation, and stabilization. Again, no major anatomic structures lie in close approximation. The incision is made full thickness to the bone, and the periosteum is reflected to expose the fracture. Generally, some form of elevator is placed posteriorly in the infratemporal fossa to aid in elevation of the fracture. Care should be taken not to lever against the temporal bone and displace any nondiagnosed, nondisplaced skull fractures. The incision is generally placed in the hair of the brow. The usual rule of making an incision perpendicular to the skin margin can be altered in this area because the incision should be made with the long axis of the hair follicle to prevent damaging the follicles, which could prevent the regrowth of hair. This hair should not be shaved for numerous reasons. The hair will grow back, but slowly, and it will be of a different texture and may often be sparse. The most notable problem is that of aligning the hair-bearing skin margins during suturing. If this is not performed properly, there will be an unsightly step in the brow when the hair does regrow.

Approaches to the Inferior Rim. The multiple surgical approaches to the inferior rim† and articulation of the zygoma and maxilla are described in Chapter 16. These approaches provide access to the inferior orbital rim,

*References 1, 2, 4-6, 11, 15, and 20.

*References 14, 15, 20, 21, and 24.
†References 12, 14, 15, 18, 20, and 25.

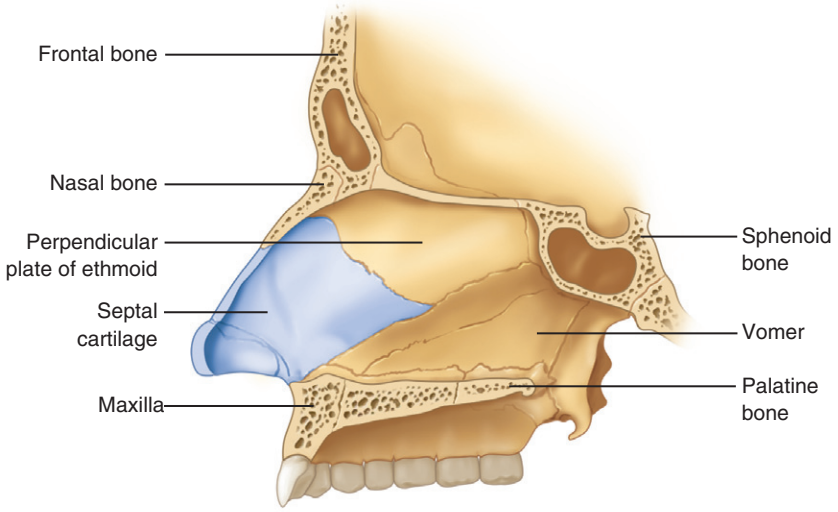


FIGURE 10-8 Nasal septum, nasal bones, vomer, and ethmoid bone.

orbital floor, lacrimal duct area and, in some cases, medial and lateral orbital walls. There are few anatomic problems if care is used to locate the layers of the inferior lid, most specifically the orbicularis oculi muscle, orbital septum, and inferior orbital rim. The approaches from the skin include the infraciliary incision and a lower incision through an existing skin crease. The infraciliary incision is aesthetically pleasing but may result in excessive and prolonged edema of the eyelid. The lateral extent of this incision, combined with the frequently needed lateral brow incision, compromises the lymphatic drainage of the lower lid. The skin is thin in this area and the skin flap must be developed carefully. The incision through the orbicularis oculi muscle is performed longitudinally to expose the inferior orbital rim. The incision through the periosteum should be on the facial aspect of the bone but above the infraorbital nerve.

Transoral Approach. The floor of the orbit and infratemporal fossa can be approached through a buccal mucosa incision in the posterior maxillary vestibule.[15,17-19] The eminence of the zygoma becomes available for stabilization via this approach. The maxillary sinus can be entered and the floor of the orbit, inferior rim, and eminence of the zygoma can be elevated from within. The infratemporal fossa can be entered posteriorly and superiorly, and the zygomatic arch and zygomatic body may be elevated. The buccal fat pad often interferes with visualization but is generally of no notable anatomic concern.

Transconjunctival Approach. The final approach to the orbital floor is via the transconjunctival incision.[5,14,21] Its only advantage is the avoidance of the slight facial scar from the infraciliary approaches. In the case of trauma, this is usually not a major factor. The incision is made through the conjunctiva at the lower border of the inferior tarsal plate, with the lower lid retracted inferiorly. A preseptal or retroseptal dissection can be made, although the preseptal dissection offers better control of the orbital fat. The approach to the orbital floor is otherwise the same and there are no notable anatomic problems associated with any of these approaches.

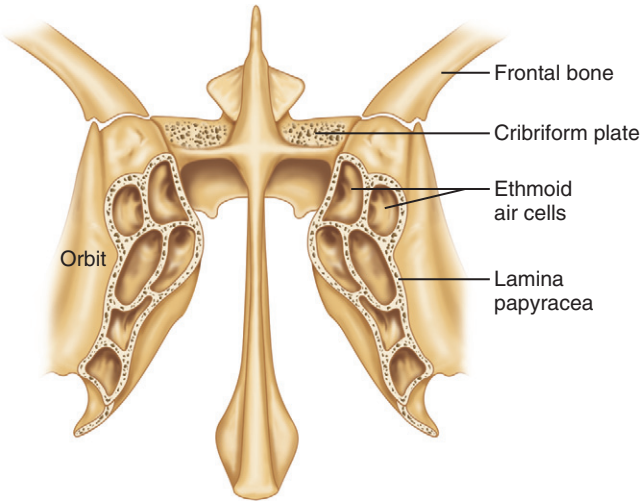


FIGURE 10-9 Frontal section of ethmoid bone.

Nasal Bones

The nasal bones (Fig. 10-8; see also Fig. 10-4)[1,2,4,6] are rectangular and articulate with the frontal bone superiorly and with each other at the midline. At the superior articulation, they are relatively thick, but inferiorly, they are much thinner. It is in this area that most fractures occur. The nasal bones articulate posteriorly with the frontal process of the maxilla.

Ethmoid Bone

The ethmoid bone is an unpaired bone that is central to the facial structure (Fig. 10-9),* and it is an integral part of the nasal structure, to both orbits and to the anterior cranial base. The perpendicular plate forms the superior and anterior portions of the nasal septum and attaches to the cribriform plate. It articulates posteroinferiorly with the vomer and posterosuperiorly with the sphenoid bone.

*References 1, 2, 4-6, 11, and 19.

The cribriform plate articulates anteriorly and laterally with the frontal bone and posteriorly with the sphenoid bone. Hanging bilaterally from the cribriform plate are the superior and middle nasal conchae. The middle concha has thin-walled ethmoidal air cells, which extend laterally to it. The multiple septa, which pass relatively perpendicular to the conchae, extend laterally to the thin plate of bone that constitutes most of the medial orbital wall. This bone is the lamina orbitalis of the ethmoid bone. It is extremely thin; hence the term *lamina papyracea*.

Surgical Note: The thin lamina orbitalis may be fractured in blunt orbital trauma.[11,15,17] The anterior ethmoid artery is a point for ligation as it passes from the orbital to the nasal aspect of the ethmoid bone. Because this artery is one of the terminal branches of the ophthalmic artery, which is a branch of the internal carotid artery, it is not affected by the usual measures to control facial bleeding and may require direct ligation via a medial canthal approach. The anterior ethmoid foramen is approximately 1.5 cm deep, measured from the medial orbital rim. Rarely is any surgical manipulation of this bone necessary or possible.

Vomer

The vomer (see Fig. 10-8)[1,4,7] is a plow-shaped bone that is located in the midline of the nasal fossa and forms the posterior portion of the nasal septum. It articulates with the palatine, maxillary, and ethmoid bones and rarely is of notable concern in the primary management of facial trauma.

Palatine Bones

The paired palatine bones connect the maxilla with the sphenoid bone (Fig. 10-10; see also Figs. 10-5*A* and 10-6).[1,2,4,5] This extremely irregularly shaped bone is composed of a major horizontal portion and vertical perpendicular plates. The horizontal plate articulates anteriorly with the maxilla and with the palatine bone of the opposite side in the midline to form the posterior aspect of the hard palate.

The vertical plate passes superiorly behind the maxilla and articulates posteriorly with the lateral pterygoid plate of the sphenoid bone. A ledge of the vertical plate terminates in a small contribution to the orbital floor at the posteromedial aspect. The junction of the sphenoid and palatine bones forms the sphenopalatine foramen. This foramen attaches the posterior aspect of the nasal cavity with the pterygopalatine fossa.

Surgical Note: Manipulation of the maxilla generally accomplishes adequate reduction of the palatine bones.[8,17] It is important to remember the small contribution of the palatines to the orbital floor because extreme trauma to the maxilla and palate may cause some displacement or involvement of the orbital contents.

Inferior Nasal Concha

The inferior nasal concha is a paired bone[2] that forms the bony support of the inferior turbinate bilaterally. It is of surgical importance only when it obstructs the inferior meatus and the nasal lacrimal duct.

Frontal Bone

The frontal bone (Fig. 10-11; see also Fig. 10-4)* is a cranial bone that is unpaired and forms the anterior

*References 1, 4, 14, 26, and 27.

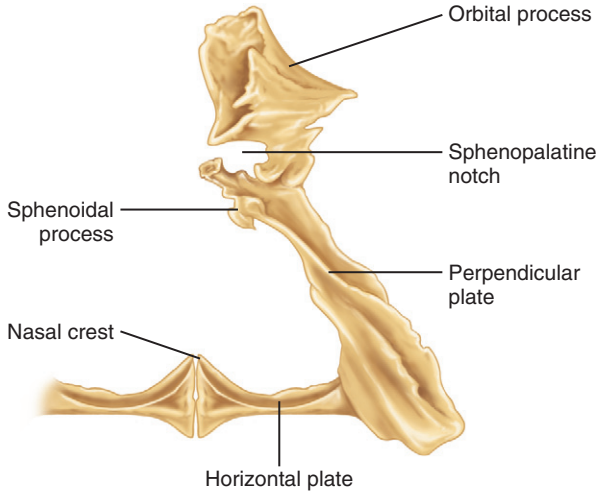


FIGURE 10-10 Palatine bone. Note orbital surface and nasal and palatal aspects.

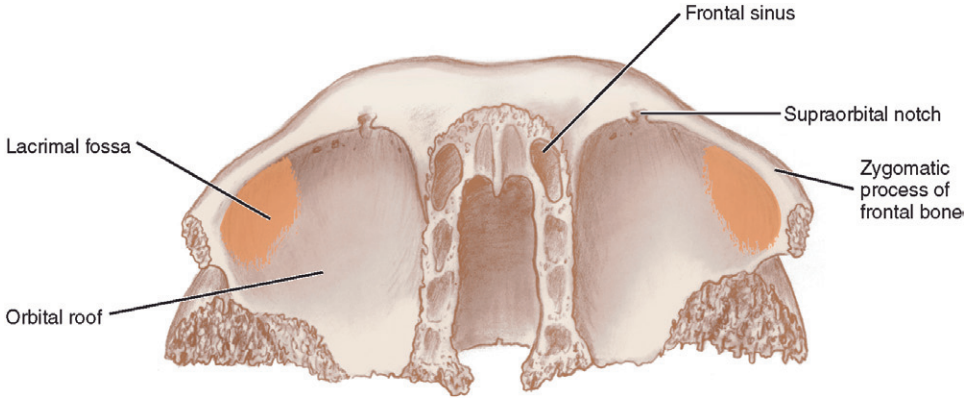


FIGURE 10-11 Frontal bone from inferior view. Note articulation with the nasal and ethmoid bones. (From Fehrenbach M, Herring S: *Illustrated anatomy of the head and neck*, ed 4, St. Louis, 2012, Saunders.)

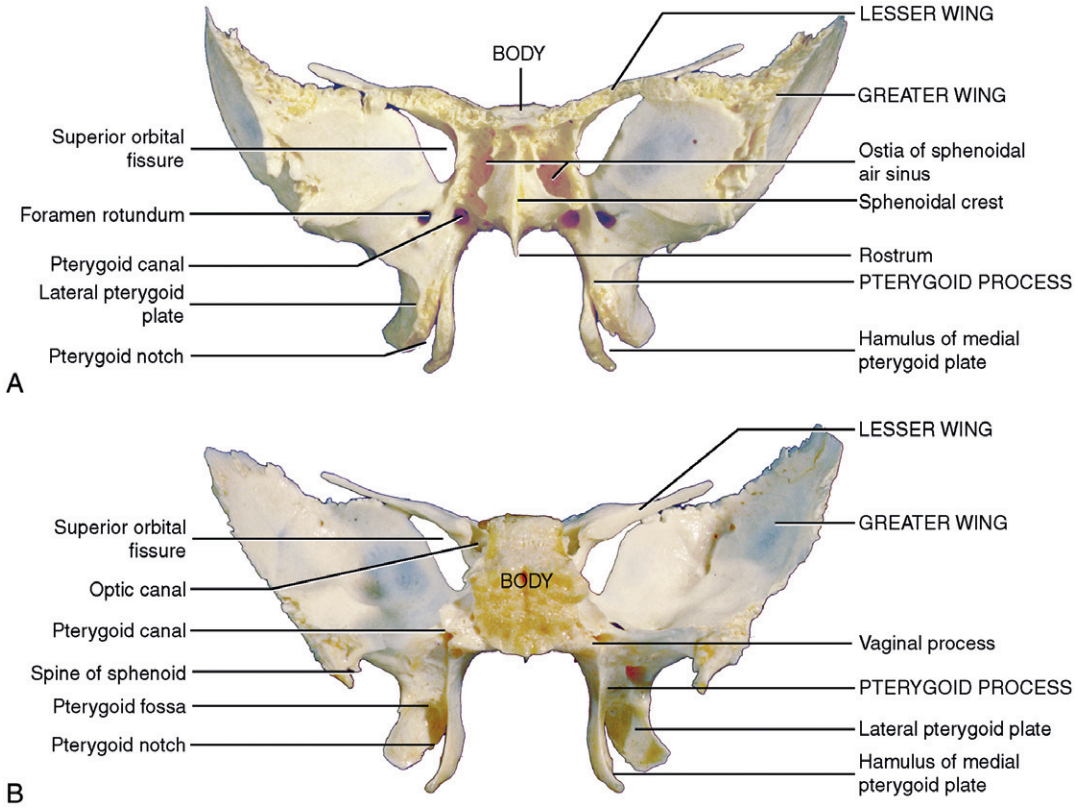


FIGURE 10-12 Sphenoid bone (frontal view). (From Liebgott B: *The anatomical basis of dentistry*, ed 3, St. Louis, 2009, Mosby.)

portion of the calvaria. The importance of this bone in facial trauma is its relationship to the anterior midfacial skeleton and the paranasal sinuses. The frontal bone articulates laterally with the zygoma and medially with the maxilla and nasal bones. Inferiorly and deep in the middle of the face, it articulates with the ethmoid and lacrimal bones and posteroinferiorly articulates with the wings of the sphenoid bone. The frontal bone articulates posterolaterally with the parietal bones.

The frontal bone forms a great portion of the roof of the orbit. Its thickened projections articulate laterally with the zygoma at the frontozygomatic suture and form the lateral orbital walls. The thickening of the frontal bone in the anterior region forms the supraorbital ridges. These curved elevations connect the zygomatic portion of the frontal bone with its midportion, articulating with both the maxilla and nasal bones. The supraorbital notch or foramen crosses this rim and transmits the frontal vessels and nerves.

The frontal sinus lies in the frontal bone in an area superior to the articulation with the nasal bones. Approximately 4% of the population do not have a frontal sinus. The sinus is not a simple chamber but rather is subdivided into compartments or recesses by incomplete bony partitions. There is usually an intrasinus septum that divides the left from the right. Drainage into the nose is by a well-formed duct, the nasofrontal duct. The duct itself is soft tissue and may follow a serpentine course to the anterior middle meatus of the nose, where it empties. The frontal sinuses are protected somewhat from injury by the supraorbital ridges. The anterior wall of the sinus has low resistance, but the ridges are highly resistant.

Surgical Note: There are multiple incisions and techniques of management for the frontal sinus. The major anatomic point of concern is the inner table, which, when fractured, demands a neurosurgical evaluation. Other areas of concern are the supraorbital nerves, which can usually be saved with careful dissection and removal from the supraorbital foramen by the use of a small osteotome. The neurovascular bundle can then be retracted with the orbital contents.

Sphenoid Bone

The sphenoid bone (Fig. 10-12)* is a single midline bone situated at the base of the skull that creates the anteroinferior extent of the cranial base and the posterior transition from facial bones to cranial bones. This complex bone has many processes that have delicate articulations with the adjacent cranial and facial bones.

The sphenoid bone articulates with the temporal and occipital bones to form the cranial base. It joins the parietal and frontal bones anteriorly and superiorly to complete the cranial complex. It meets the vomer and ethmoid bones in the midline anteriorly and meets the zygoma, palatine bones, and sometimes the tuberosity of the maxilla to complete its articulation with the facial skeleton.

The body of the sphenoid bone is hollow and forms two cavities separated by a thin bony septum. The hollow cavities are the sphenoidal sinuses; these drain into the sphenoethmoid recess above and behind the superior nasal concha. Although air-fluid levels can frequently be noted on radiographs, surgical management in the trauma patient is rarely necessary.

*References 1, 2, 4, 6, and 19.

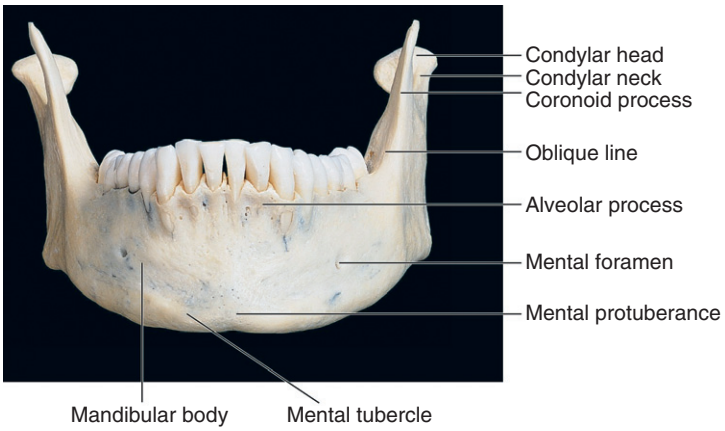


FIGURE 10-13 Mandibular osteology (anterior view). (Modified from Abrahams PH, Hutchings RT, Marks SC Jr: *McMinn's color atlas of human anatomy*, ed 6, St. Louis, 2008, Mosby.)

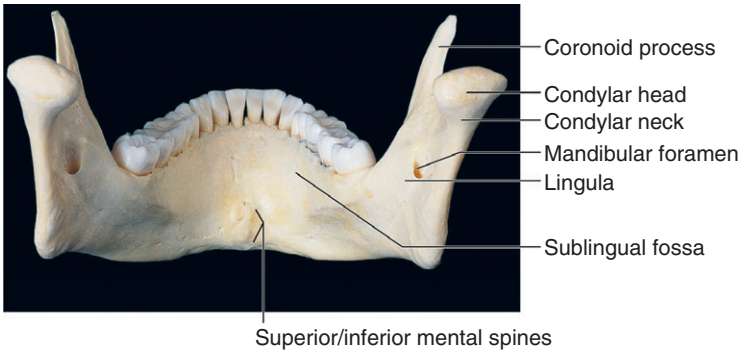


FIGURE 10-14 Mandibular osteology (lingual view). (Modified from Abrahams PH, Hutchings RT, Marks SC Jr: *McMinn's color atlas of human anatomy*, ed 6, St. Louis, 2008, Mosby.)

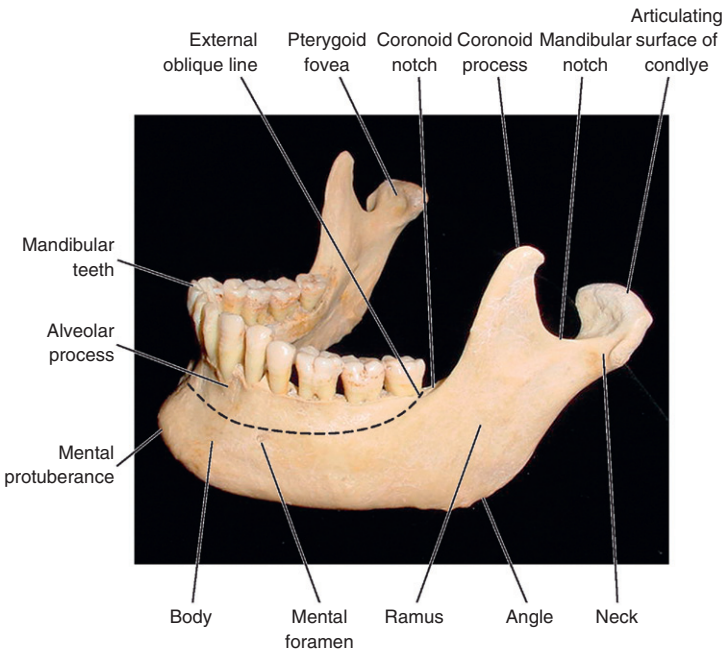


FIGURE 10-15 Mandibular osteology (oblique view). (From Fehrenbach M, Herring S: *Illustrated anatomy of the head and neck,* ed 4, St. Louis, 2012, Saunders.)

LOWER FACE

Mandible

Despite the fact that the mandible (Figs. 10-13 to 10-15)* is the largest and strongest facial bone by virtue of its position on the face and its prominence, it is commonly fractured when maxillofacial trauma has been sustained. Mandibular fractures occur twice as often as midfacial fractures[29,32]; however, it has been shown in cadaver experiments that almost four times as much force is required to fracture the mandible versus the maxilla.[30] The osteology of the mandible, various muscle attachments and their influence, and the presence of developing or completed dentition all play a notable role in producing inherent weaknesses. Therefore, fractures are seen more frequently in certain isolated areas.

*References 4, 6, 10, 16, 18, and 28-31.

The mandible is composed of the body and two rami, with their junction or angle forming the prominent gonion. The angle formed may vary between 110 and 140 degrees (mean, 125 degrees).[15] The angle decreases slightly during growth because of changes in the condylar process, shape, and size. With aging, the angle becomes more obtuse.[30] The body is U-shaped and has an external and internal cortical surface. The external cortical plate is thickest at the mental protuberance and in the region of the third molar. There is also a thickened triangular mental protuberance bounded laterally by the mental tubercles. The mental foramen is located on the external surface in the vicinity of the root apices of the first and second premolars. There are variations in the exact location of the foramen, as noted by Tebo and Telford.[33] The opening is directed backward and laterally and transmits the mental nerves and vessels.[18,34] The oblique line runs from just inferior to the mental foramen posteriorly and superiorly to the ascending ramus.

The internal cortical surface is elevated in the midline near the inferior border by the mental spine. Associated with this may be two pairs of discrete bone prominences termed the *genial tubercles*. They represent the origin of the geniohyoid muscles inferiorly and the genioglossus muscles superiorly. Running horizontally and slightly superior from front to back is an oblique ridge, the mylohyoid line, which represents the attachment of the mylohyoid muscle. Below this is the shallow depression created by the submandibular gland, called the submandibular fossa. Superior to the mylohyoid line and located anteriorly is the sublingual fossa, where the sublingual gland is found in close approximation.

The ramus of the mandible, when viewed from the side, is a quadrilateral structure. The lateral surface may be rough and thickened in the region of the angle by the insertion of the masseter muscle. On the medial surface is the mandibular foramen, which leads downward and forward into the mandibular canal and transmits the inferior alveolar nerve and vessels. The lingula is a medial bony projection to which the sphenomandibular ligament is attached. The mylohyoid groove extends from the lingula and runs anteriorly and inferiorly to the submandibular fossa. Below this is a roughened area created by insertion of the medial pterygoid muscle.

The mandibular notch is located on the superior edge of the ramus. It is bounded anteriorly by the coronoid process and its temporalis attachments, while also bound posteriorly by the neck and head of the mandibular condyle. A detailed description of condylar head anatomy is given later (see "Temporomandibular Joint"). Attached anteriorly to the neck of the condyle is the insertion of the lateral pterygoid muscle and attached laterally is the lateral ligament.

The body of the mandible supports the alveolus and dental structures. The body and alveolus have dense cortical outer and inner tables of bone, with central spongy or cancellous bone.

The strengths of the mandible are apparent when one evaluates the thick, round inferior border and the mental protuberance. The periodontal ligament and bone alveolus also combine with the trabecular pattern in the cancellous bone and are directed in a parallel fashion up the ramus to transmit pressures up to the condylar region.[35,36] The thickening on the inner aspect of the condylar neck or crest of the neck apparently acts as a main buttress of the mandible as it transmits pressure to the temporomandibular joint (TMJ) and the base of the skull.[29,32] The temporal crest runs from the coronoid process to the retromolar triangle distal to the terminal molar. The thickened posterior border of the mandible may act as an additional crest.[28]

Major structural forces are created at the angle of the mandible because of the cantilevered nature of its shape. The bone height at this angle is therefore critical in determining its strength and the presence of the perfectly aligned muscle sling created by the masseter and medial pterygoid muscles.[37] Thus, aging, with its potential for bone and alveolar resorption, weakens this area.

Areas that exhibit weakness include the area lateral to the mental protuberance, mental foramen, mandibular angle, and condylar neck.[29] If teeth are present, the socket is a weak zone, especially if teeth are impacted or unerupted. This would seem to indicate that a child in the mixed dentition stage may be highly susceptible; the fact that the child's bones are so resilient and flexible offsets the disadvantage of the unerupted teeth.[30]

Temporomandibular Joint

The TMJ (Figs. 10-16 and 10-17)* is a freely movable synovial joint located between the glenoid fossa of the temporal bone and the head of the mandibular condyle below. The anatomic classification of the TMJ is a diarthrodial joint, with independent discontinuous movement between the two bones. An articular disc (meniscus) divides the joint into two cavities. The superior compartment permits hinge or rotational movement, whereas the inferior joint space permits translatory motion. The bony surfaces within the joint spaces are lined by synovial membrane, which is responsible for secreting synovial

*References 1, 6, 10, 17, 18, 35, and 38-42.

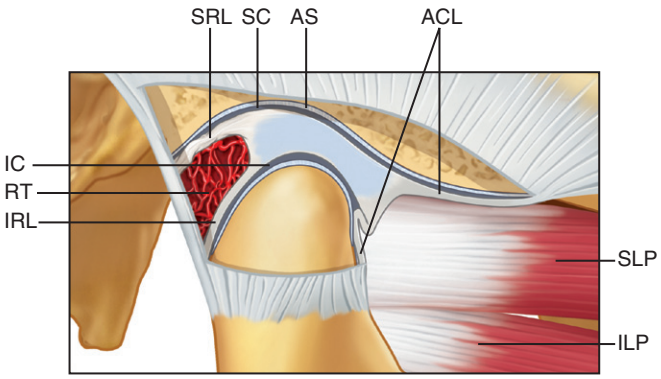


FIGURE 10-16 Temporomandibular joint showing the anatomic components. *ACL,* Anterior capsular ligament (collagenous); *AS,* articular surface; *IRL,* inferior retrodiscal lamina (collagenous); *RT,* retrodiscal tissues; *SC* and *IC,* superior and inferior joint cavity; *SLP* and *ILP,* superior and inferior lateral pterygoid muscles; *SRL,* superior retrodiscal lamina (elastic); the discal (collateral) ligament has not been drawn. (From Okeson JP: *Management of temporomandibular disorders and occlusion*, ed 7, St. Louis, 2012, Mosby.)

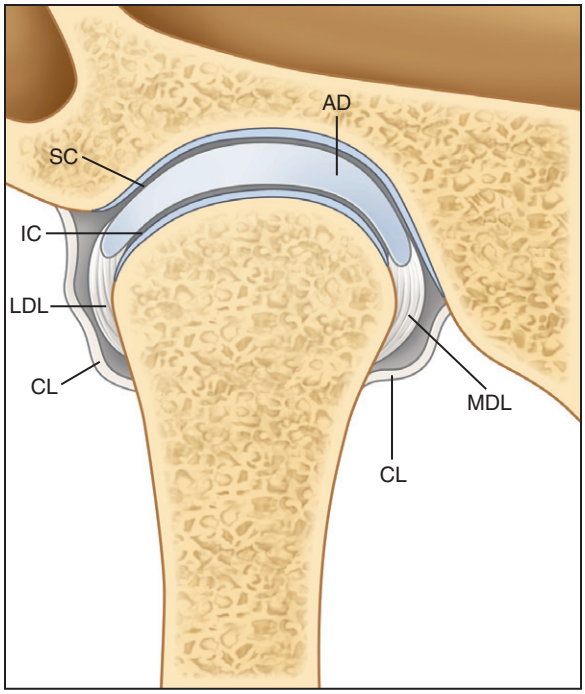


FIGURE 10-17 Temporomandibular articulation (anteroposterior view). *AD,* Articular disc; *CL,* capsular ligament; *IC,* inferior joint cavity, *LDL,* lateral discal ligament; *MDL,* medial discal ligament; *SC,* superior joint cavity. (From Okeson JP: Management of Temporomandibular Disorders and Occlusion, ed 7, St. Louis, 2012, Mosby.)

fluid that functions as nutritional support and a lubricating medium. This membrane is extremely thin, smooth, and highly vascular. Cells in the synovial membrane have the ability to differentiate into chondrocytes and provide the synovium's ability to regenerate following injury. The articular surfaces of the joint and condyle and the central portion of the meniscus are composed of collagen. This feature differentiates this joint from most other articulations because the surfaces are not covered by hyaline cartilage. Histologically, this avascular fibrous tissue may contain cartilage cells and therefore may be termed *fibrocartilage.*[10,17] This tissue has the ability to regenerate and to remodel under functional stress or following anatomic alteration, such as fracture.

The condylar head is a semicylindroid process 15 to 20 mm long and 8 to 10 mm thick. The long axis of the condyle is related to the position of the ramus of the mandible and not to the skeletal or frontal plane. The angle formed by the two condylar axes varies between 145 and 160 degrees. The articulating surface of the condylar head faces superiorly and forward, giving the condylar neck an appearance of being bent forward. When viewed anteriorly, the condylar head notably projects medially to the inner surface of the ramus, but less so laterally.

The temporal bone provides the articulating surface to the skull. This bone is located anterior to the tympanic bone. The fossa is composed of a posterior slope and the convex part of the articular eminence. The squamotympanic suture forms the boundary between the tympanic bone and temporal squama. The posterior part of the fossa has a raised crest joining the articular tubercle and postglenoid process. The roof is relatively thin, separating the fossa from the middle cranial fossa. The anterior portion of the fossa or articular eminence is a broad horizontal ledge that is convex in the anteroposterior direction and concave in the transverse direction.

The fibrous capsule surrounding the joint is thickest on the lateral surface and is considered a separate, distinct ligament—the temporomandibular ligament. It extends from the tubercle on the root of the zygoma to the lateral surface of the neck of the mandible, behind and below the lateral pole of the condyle. The temporomandibular ligament has a superficial fan-shaped layer of obliquely oriented connective tissue fiber and a deeper narrow band of fibers that are horizontally oriented.

The articular capsule in general attaches from the temporal bone to the neck of the condyle. There is a loose attachment between the temporal bone and meniscus, with considerable ability to move. The attachment from the meniscus to the condyle, however, is much stiffer and is reinforced medially and laterally. These paired ligaments act to restrict movement of the meniscus away from the condyle. This stabilizes the complex and aids in synchronizing the condyle and meniscus. Other ligaments to consider in this region are the stylomandibular and sphenomandibular ligaments, which serve as passive restraints to mandibular movement. The stylomandibular ligament originates from the styloid process and extends downward to the posterior border of the ramus of the mandible. The sphenomandibular ligament runs from the spine of the sphenoid bone and squamotympanic fissure to the lingula on the medial aspect of the mandibular ramus. The maxillary artery, its middle meningeal and inferior alveolar branches, and auriculotemporal and inferior alveolar nerves pass between this ligament and the mandible.

The articular disc or meniscus, a dense fibrocartilaginous structure, histologically may show varying degrees and locations of cartilage cells.[1] The meniscus is nonvascular and has no sensory innervation. When viewed from the lateral direction, there are three anatomic divisions or zones—the anterior band, intermediate zone, and posterior band. The thin intermediate or central zone corresponds to the functional area between the mandibular condyle and posterior slope of the articular eminence. When viewed from above, the meniscus is an oval biconcave structure that is thicker posteriorly. The posterior tissues are highly vascularized and innervated and are referred to as the bilaminar zone. The superior elastic fibers of the retrodiscal tissue attach to the tympanic plate and function to restrict anterior meniscus movement during extreme translation.[43] The inferior fibers of the retrodiscal tissue are composed of collagen and attach to the articular surface of the condyle. They act as a check ligament to prevent extreme rotation of the meniscus during rotational movement of the mandible. The anterior attachment of the meniscus is contiguous with the lateral pterygoid muscle. The overall shape and size of the meniscus, therefore, vary depending on age, functional remodeling, and pathologic condition.

The vascular supply to the TMJ arises anteriorly from the masseteric artery and posteriorly from branches of the superficial temporal and maxillary arteries. The

retrodiscal region has a rich venous plexus that fills and empties under the influence of mandibular movement.

The nerve supply to the joint is principally from the auriculotemporal nerve, with some additional innervation via the masseteric nerve and the deep temporal nerve.

Mandibular Fracture Location

The mandible has various strengths and weaknesses, as noted.* The common sites of mandibular fracture, therefore, are the mandibular condyle region, mandibular angle region (especially in the presence of an impacted or semierupted third molar), mental foramen or body region, mandibular parasymphysis, and any component of the dental alveolus. In the very young and older patient with mandibular atrophy,[30] other factors such as tooth buds developing in the child or a decrease in the cancellous-to-cortical ratio in older adults come into play. Because of the U shape of the mandible, eccentric forces often create bilateral fractures—one at the site of injury and the other contralaterally. Nahum[36] has shown that forces in excess of 800 lb are required to fracture the symphysis and both condylar necks, and further demonstrated that the mandible is more sensitive to lateral impact than to impact from the front.

*References 31, 28, 32, 35-37, and 44.
†References 4, 6, 18, 30, 33, 36, 41, and 45.

Anatomic Factors in Fracture Displacement

The direction of the causative blow, the direction of the line of fracture, and muscle pull all influence the amount and direction of bone displacement.† Muscle forces acting in the anterior region of the mandible, including those inserting in the mental region on the inner surface, are the geniohyoid, genioglossus, digastric, and mylohyoid muscles. These forces act to displace anterior segments inferiorly and posteriorly, with some possible medial component.[13,41]

In the posterior mandible, the muscles of mastication generally cause upward and forward displacement. This observation is especially true in the pterygomasseteric sling region. The medial pterygoid muscle also creates a medial component of pull. The temporalis muscle has two components of attachment and creates elevating forces and retraction forces. In a similar way, the lateral pterygoid muscle has two attachments. The internal component is responsible for superior, anterior, and medial forces, whereas the external component pulls the condyle down, anteriorly, and medially. If the balance is disrupted because of a fracture, displacement results.

The fracture angulation or direction of the mandibular angle and body region can vary. Depending on its orientation, muscle influence may be enhanced or prevented from actively displacing the proximal segment (Fig. 10-18). A fracture is regarded as favorable if it is in

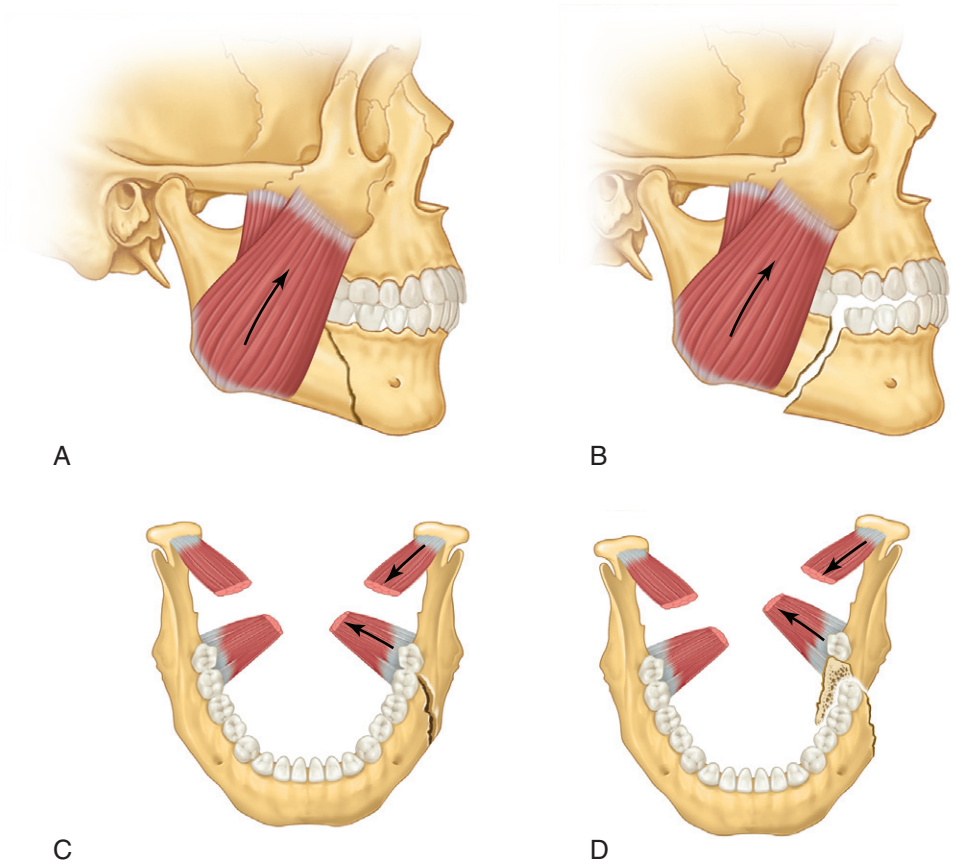


FIGURE 10-18 Mandibular angle fracture. **A,** Horizontally favorable. **B,** Horizontally unfavorable. **C,** Vertically favorable. **D,** Vertically unfavorable.

a downward and forward direction horizontally because of locking effects at the fracture site. If the horizontal direction is downward and posterior, the active pull of the posterior elevator muscles, such as the temporalis, masseter, medial pterygoid, and lateral pterygoid muscles, will displace the proximal segment superiorly. The vertical angulation of the fracture or bevel will inhibit the medial forces of the elevator group if the fracture direction is posterior and medial. This condition is regarded as vertically favorable. The opposite is true of the vertically unfavorable fracture traveling anteriorly and medially.

Other factors affecting the amount of displacement are the presence or absence of teeth in occlusion, muscle protection, such as in the pterygomasseteric sling region, and the exact relationship of the fracture position to muscle insertion. This last factor is especially apparent in condylar neck injuries and their relationship to the lateral pterygoid muscle insertion. If a fracture occurs above the insertion of this muscle on the neck of the condyle, little displacement occurs. If a lower level fracture occurs, displacement of the condyle will be medial and anterior because of the action of the lateral pterygoid muscle.

Surgical Note: Fractures that may compromise the patient's airway are important to the treating surgeon. Whenever the fracture creates an unstable situation for the tongue, consideration must be given to immediate temporary stabilization, intubation, or other means of supporting the airway. Mandibular fractures that may create airway problems include bilateral subcondylar fractures, bilateral parasymphysis fractures, and any maxillofacial trauma with massive edema or oral lacerations with subsequent bleeding.

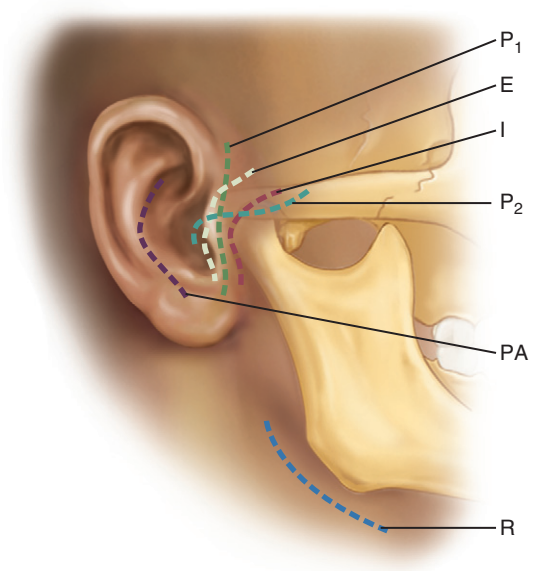


FIGURE 10-19 Surgical approaches to the posterior mandible and temporomandibular joint. *E,* Endaural approach; *I,* inverted hockey stick approach; P_1 and P_2, preauricular approach; *PA,* postauricular approach *R,* Risdon approach.

MANDIBULAR SURGICAL APPROACHES

Mandibular surgical approaches are as follows (Fig. 10-19): extraoral approaches include the (1) Risdon, (2) condylar, and (3) symphysis approaches; intraoral approaches are the (1) angle and (2) mental or parasymphysis approaches.

EXTRAORAL SURGICAL APPROACHES

An absolute description of various surgical procedures is possible in an elective situation. However, when dealing with a trauma patient, each approach is designed while taking into consideration the location of the injury, extent of the injury, method of stabilization to be used, possible coexisting soft tissue injuries, and potential anatomic factors.* The basic principles of making soft tissue incisions in natural skin folds may be more difficult to apply in the presence of severe edema and lacerations. In dealing with mandibular trauma in this section, the more common surgical approaches used for open reduction and exploration are discussed.

Preauricular and Condylar Approaches

Several soft tissue incision designs have been described, including the inverted L, as described by Blair,[38] the T, as described by Wakely,[46] and the endaural approach advocated by Lempert.[47] Dingman and Moorman[1] slightly modified Lempert's approach and reported it in 1951. The facial nerve, when damaged, poses the most notable obstacle and potentially the most serious complications. A detailed description of the facial nerve appears later in this chapter (see "Facial Nerve"). Other anatomic structures that must be considered in the overall dissection in the preauricular approach include the parotid gland, superficial temporal vessels, and auriculotemporal nerves.

The parotid gland is located below the zygomatic arch in front of the external auditory meatus, with the superficial pole of the gland lying directly on the TMJ lateral capsule. The parotid gland is enclosed in fascia derived from the superficial layer of the deep cervical fascia or parotideomasseterica fascia.

The superficial temporal vessels, along with the auriculotemporal nerve, emerge from the superior aspect of the parotid gland. Bleeding from these vessels can be brisk and often requires ligation. The vein lies superficial and posterior to the artery; the auriculotemporal nerve is located posterior and superficial to the artery. By keeping dissection close to the cartilaginous portion of the external auditory meatus, trauma to the auriculotemporal nerve can be minimized.

The endaural incision is started in the skin crease immediately adjacent to the anterior helix and is carried downward to the level of the tragus. The incision can then be placed in the gap between the spine of the helix and tragus, which is filled with fibrous attachments for the lamina of the tragus. (While in the auditory meatus, the incision remains in contact with the bony tympanic plate.) This incision results in a better cosmetic appearance. It is important not to extend the incision or

*References 16, 31, 32, 29, 35, 37-41, and 45.

dissection inferiorly because damage to the facial nerve as it exits the stylomastoid foramen may result.

The initial incision is made through skin and subcutaneous connective tissues, which include the temporoparietal fascia to the depth of the superficial layers of the temporalis fascia. In the upper aspect of the incision, the superficial temporal vessels may be encountered, as may the auriculotemporal nerve. The nerve is retracted and the vessels are retracted or ligated. The temporalis fascia is incised by an oblique incision above the zygomatic arch. The intervening fat is visualized between the two layers of temporalis fascia and blunt dissection is carried out inferiorly beneath the superficial layer of the temporalis muscle. The periosteum is stripped off the zygomatic arch from above. A sharp incision posteriorly along the plane of the initial incision can safely be made down to the periosteal level. The flap is elevated anteriorly, exposing the articular eminence. The temporomandibular capsule is now visualized totally. The condyle is palpated with the help of manual movements of the body of the mandible. Scissors or a scalpel can be used to enter the upper joint space horizontally, if necessary, to evaluate the condylar head surface or meniscus integrity. Depending on the extent of the dissection necessary at this point, the operator should be cognizant of the medial structures, including the maxillary artery, middle meningeal artery, auriculotemporal nerve inferiorly, and pterygoid plexus of veins lying anteromedially. It may be necessary in some cases to use a Risdon approach as well when performing an open reduction in low fractures.

RISDON AND SUBMANDIBULAR APPROACHES

During the procedures to reduce and stabilize mandibular angle fractures and, in some cases, low subcondylar fractures, some form of approach from the inferior mandible is required (Fig. 10-20). Parameters used to establish an incision include the following:

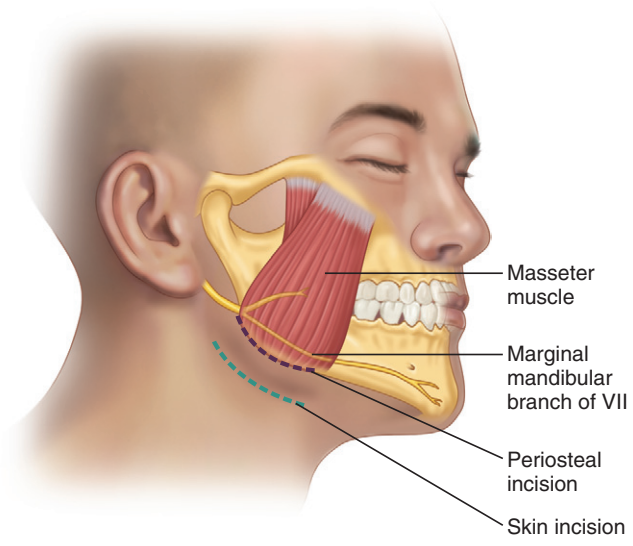


FIGURE 10-20 Risdon approach illustrating the relationship of the marginal mandibular branch of the facial nerve (VII) to the inferior border of the mandible and periosteal incision *(dotted line).*

- Anteroposterior fracture location
- Natural skin folds
- Langer's skin lines
- Position of the marginal mandibular branch of the facial nerve

For this reason, the incision is usually located approximately 2 cm or two fingerbreadths below the inferior border of the mandible. It is necessary to make an incision long enough to expose and identify anatomic structures, such as the facial artery and vein, and to obtain sufficient access to the fracture. The incision may be extended posteriorly to within 0.5 cm below the lobe of the ear. After marking the skin, the head is extended and turned to one side. The incision may be cross-hatched to reapproximate soft tissues during final suturing in an anatomic manner without making a so-called dog ear at one end.

The initial incision is made through the skin and subcutaneous tissue. Any bleeding is controlled by electrocautery. If a local anesthetic is administered, it is important not to inject it deep to the platysma muscle if electrical nerve stimulation is used to detect facial nerve function. The skin and subcutaneous tissue are then undermined adequately. At this point, the operator should visualize the well-demarcated muscle lines of the platysma. The muscle may be entered carefully at one end of the incision by a mosquito hemostat and bluntly dissected from beneath toward the other end of the incision, staying parallel to the inferior border of the mandible. The corner of the patient's mouth should be carefully observed during this procedure because the marginal mandibular branch of the facial nerve travels just below the platysma muscle. The surgeon can check the undermined muscle by carefully clamping portions or using an electrical nerve stimulator, followed by complete sectioning by knife or scissors. If the marginal mandibular branch of the facial nerve is encountered, it should carefully be dissected free and retracted.

Next, the facial artery should be located by palpation initially and then by blunt dissection, if necessary. The marginal mandibular nerve should run directly over this artery. The artery is usually found anterior to the vein. If necessary, these structures can be isolated and ligated. The submandibular gland should be visible at this point. It may be necessary to separate the lower pole of the parotid gland from the submandibular gland. In other individuals, these structures may be separated by the stylomandibular ligament.

Palpation and isolation of the inferior border of the mandible reveal the thickened pterygomasseteric sling. This can be sharply divided, along with periosteum, and can be elevated from the bony mandible until adequate visualization and mobilization of the mandible are achieved. Bleeding will be reduced if the sling is incised in the posterior portion rather than laterally through the masseter muscle.

The Risdon modification of this incision involves a more posterior and vertical incision, posteroinferior to the angle of the mandible. The advantage to this approach is that there is less likelihood of damage to the marginal mandibular branch of the facial nerve, but good exposure is maintained for most procedures, except angle fractures located more anteriorly.

INTRAORAL SURGICAL APPROACHES

ANGLE

Often, when the presence of an impacted or partially impacted lower third molar in the line of fracture at the angle necessitates removal, an intraoral approach is reasonable for fixation by wire osteosynthesis or with plates. Generally, the anatomic considerations in flap design are based on the position of the tooth (e.g., buccal, lingual, erupted), amount of bone displacement, and type of fixation that will be used (e.g., rigid internal fixation may require a more extensive incision). It may be necessary to strip some of the superficial tendon of the temporalis muscle off the ascending ramus. When dissection is carried down to the inferior border of the mandible, the operator must stay within the periosteal sheath to prevent damage to branches of the facial nerve and to the facial artery and vein as they course around the mandible in the antegonial notch region.

PARASYMPHYSIS AND BODY

The intraoral approach to the parasymphysis and mental nerve region of the mandible affords the operator the following advantages—excellent visualization, the ability to observe the occlusion at the same time as reduction and stabilization are performed, and an extraoral scar can be avoided. Two types of incisions can be used, a vestibular or gingival sulcus incision. In the parasymphysis and midline area, a vestibular incision should be placed far enough into the unattached mucosa to prevent postoperative gingival stripping by scar contracture. Care should also be taken to avoid the mental foramen region with the incision. Careful dissection of the mental nerve and, if necessary, nerve relocation can be carried out safely if access to the fracture is restricted. Stripping of the attachment of the various muscles of facial expression, such as the depressor anguli oris and platysma, does not seem to produce any deleterious effects on the postoperative result. However, release of these attachments does increase the chances of postoperative hematoma formation, and pressure dressings are recommended. The mentalis muscle is the only elevator of the lower lip and chin and, if not properly repositioned during final closure, may lead to drooping of the lower lip. The paired mentalis muscles begin at the root of the lower incisor teeth. Their fibers pass inferiorly and insert into the skin at the soft tissue pogonion. With proper incision placement, there should be sufficient mentalis muscle mass in superior and inferior tissues to suture deeply when closing.

MUSCLES

MUSCLES OF FACIAL EXPRESSION

Although the muscles of facial expression (Fig. 10-21) do not have a strong influence on the displacement of fractures, soft tissue damage found in major maxillofacial trauma and the various surgical approaches necessary for repairing deeper structures invariably affect these muscles. If consideration is not given to their presence during repair and treatment planning, unsatisfactory aesthetic results will be encountered.

Muscles in this group all lie superficially and hence influence the skin.[18] They perform major activities, such as closing the eyelids and moving the lips. All muscles in this group are innervated by the facial nerve. They can be categorized into the following areas:

- Muscles of the scalp and auricle

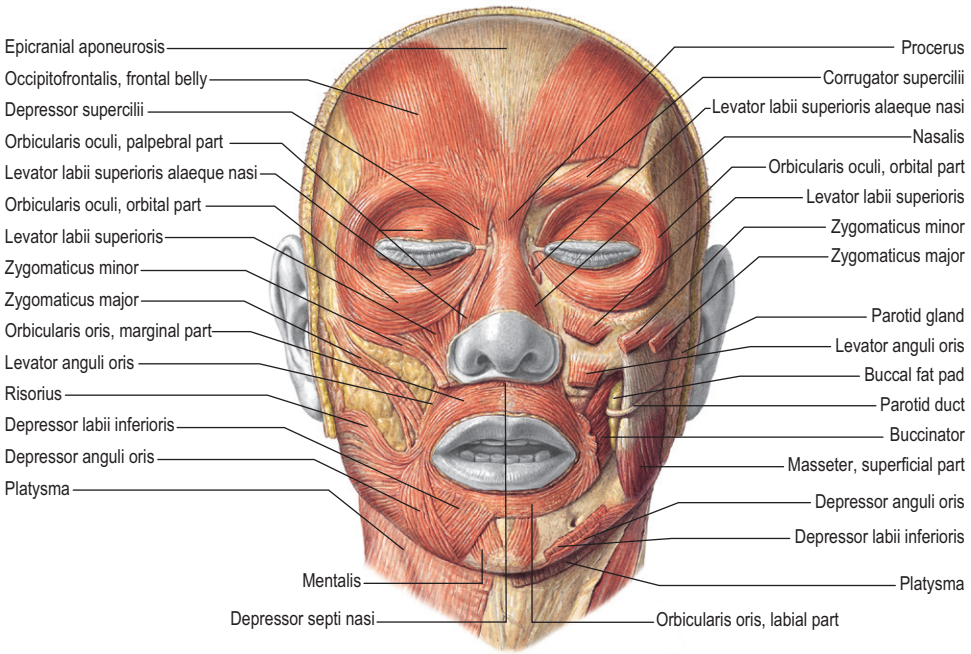


FIGURE 10-21 Muscles of facial expression. (From Standring S, editor: *Gray's anatomy: The anatomical basis of clinical practice*, ed 40, Philadelphia, 2008, Churchill Livingstone.)

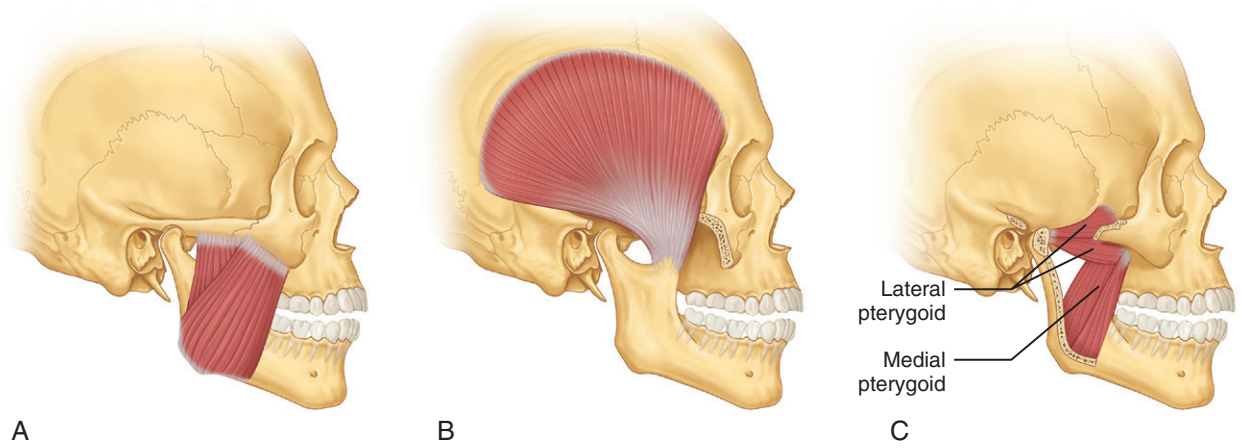


FIGURE 10-22 Muscles of mastication. **A,** Masseter muscle illustrating two heads. **B,** Temporalis muscle. **C,** Medial pterygoid muscle and lateral pterygoid muscle.

- Muscles around the orbit
- Muscles of the nose
- Muscles of the mouth
- Platysma muscle extending down the neck

Anatomically, these muscles blend together at various points, and it is impossible to dissect or differentiate individual muscles at these spots. This is especially true near the corners of the mouth, where the modiolus consists of a convergence of six muscles. Included in this group is the buccinator muscle, which has no bone origin but instead arises from the pterygomandibular raphe and forms a continuous sheet with the orbicularis oris muscle. The other muscles arise from bone attachments.

MUSCLES OF MASTICATION

As discussed in prior sections, the muscles of mastication (Fig. 10-22)* play a notable role in bone displacement following mandibular fracture. Their actions must also be considered during treatment planning because the type and direction of placement of fixation devices may be influenced by future muscle pull. All muscles of mastication are innervated by branches of the mandibular nerve, which is a division of the trigeminal nerve.

Masseter Muscle

The masseter muscle is a large, rectangular, superficial muscle composed of superficial, middle, and deep portions (see Fig. 10-22*A*). The superficial portion arises from the lower border of the zygoma and the most anterior fibers arise from the zygomatic process of the maxilla. These fibers generally run downward and posteriorly. The middle part of the masseter muscle arises from the medial posterior third of the zygomatic arch, whereas the deep part arises from the medial surface of the zygomatic arch and from the fascia over the temporalis muscle. These fibers are directed more vertically downward compared with the superficial fibers. All three portions insert together into the lateral surface of the mandible. The attachments extend to include the lower third of the posterior border of the ramus in addition to the lower border as far anteriorly as the second molar. The masseter muscle is innervated by the masseteric nerve, which reaches the deep surface of the muscle through the mandibular or coronoid notch. The blood supply is furnished by the masseteric artery, which is a branch of the internal maxillary artery.

This muscle acts as a powerful elevator. The deep fibers are also involved in mandibular retraction.

Temporalis Muscle

The temporalis muscle is a fan-shaped muscle lying in the temporal fossa (see Fig. 10-22*B*). Its origin is from the floor of the fossa below the inferior temporal line. It also arises from the deep surface of the temporal fascia. The temporal muscle bundles converge toward the deep surface of the zygomatic arch and insert into the coronoid process medially at the apex and along its anterior border.

The attachment extends down to the ramus of the mandible. There are some fibers of the posterior part that may radiate into the articular disc of the TMJ.[17]

The innervation to the temporalis muscle is via the deep temporal branches of the mandibular nerve. The blood supply is furnished by the middle and deep temporal arteries, branches of the superficial temporal artery, and internal maxillary artery, respectively.

The action of the temporalis muscle is primarily elevation, although there are some retracting capabilities of the posterior fibers.

Medial Pterygoid Muscle

The medial pterygoid muscle is found on the medial side of the mandibular ramus. It is considered the counterpart of the masseter muscle; however, overall, it is weaker. It possesses two heads of origin (see Fig. 10-22*C*). The larger deeper head arises from the medial surface of the lateral pterygoid plate and from the pyramidal process of the palatine bone. The superficial head arises from the pyramidal process of the palatine bone and from the tuberosity of the maxilla. The two heads unite, pass downward and backward, and insert into the medial surface of the mandible near the angle.

*References 4, 6, 18, 32, and 36.

The innervation of the medial pterygoid muscle is via the masseteric and buccal nerve branches of the mandibular nerve. A branch of the maxillary artery provides the blood supply.

The medial pterygoid muscle acts mainly as a synergist of the masseter muscle during elevation of the mandible. It can also act along with the lateral pterygoid muscle to cause the mandible to protrude.

Lateral Pterygoid Muscle

The lateral pterygoid muscle occupies the infratemporal fossa (see Fig. 10-22*C*). It has two heads; the large inferior head arises from the lateral surface of the lateral pterygoid plate, whereas the upper head arises from the infratemporal surface and crest of the greater wing of the sphenoid bone. The muscle fibers are oriented posteriorly and converge and insert partly into the capsule of the TMJ, mainly into the front of the mandibular condylar neck.

Innervation comes from the masseteric and buccal nerves. The blood supply is via a branch of the maxillary artery.

SUPRAHYOID MUSCLES

The suprahyoid muscles connect the hyoid bone with the skull (Fig. 10-23). Their basic functions are elevation of the hyoid bone and depression of the mandible, depending on the activity of these muscles and coincident activity of the infrahyoid muscle group.

Digastric Muscle

The digastric muscle consists of two bellies united by an intermediate tendon. The posterior belly arises from the mastoid notch of the temporal bone, from which the fibers are directed forward and downward. The anterior belly is shorter and attaches to the lower border of the mandible at the digastric fossa close to the symphysis. Fibers from here are directed downward and posteriorly. A tendon between these two bellies is attached to the body of the muscle and greater horn of the hyoid bone by fibers of the cervical fascia, which form an aponeurosis. The tendon can slide in the formed loop.

Innervation to the posterior belly is by a branch of the facial nerve and innervation to the anterior belly is by

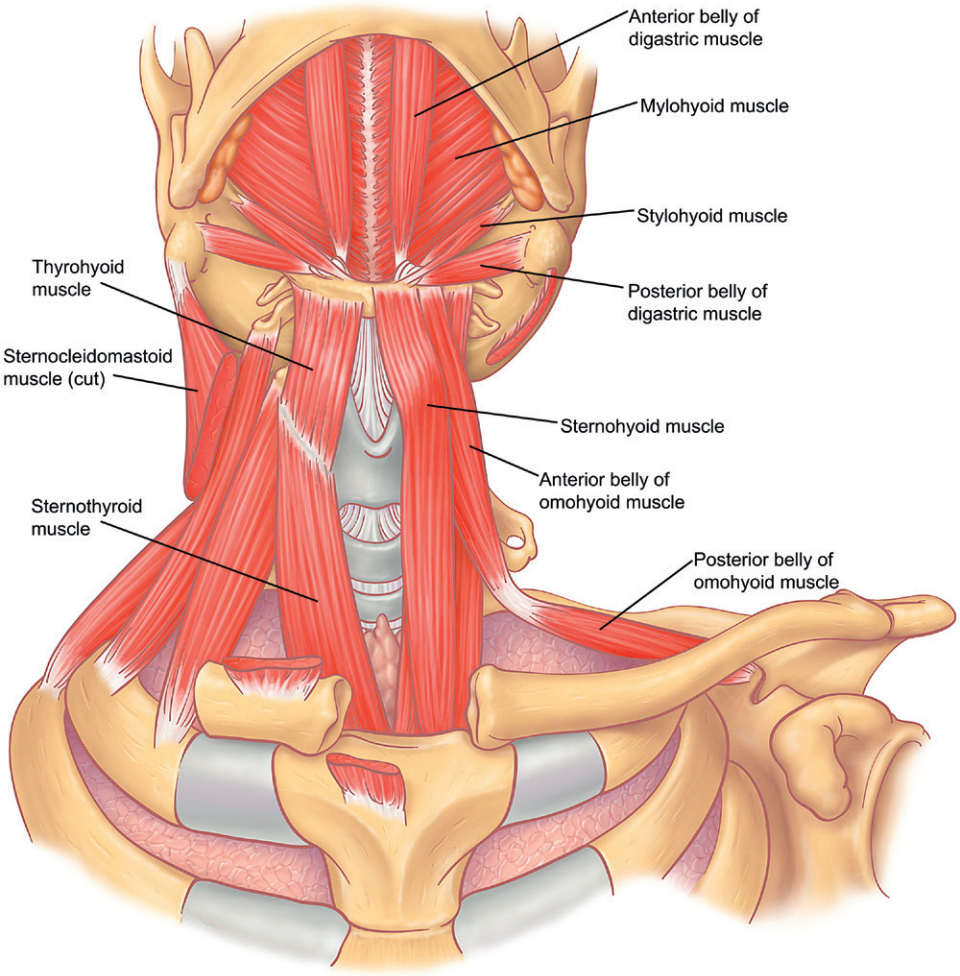


FIGURE 10-23 Suprahyoid muscles. (From Deslauriers J: Anatomy of the neck and cervicothoracic junction, *Thorac Surg Clin* 17:529, 2007.)

the mylohyoid branch of the inferior alveolar nerve. The digastric muscle pulls the chin backward and downward, which assists the lateral pterygoid muscle in rotating the mandible into an open mouth position.

Mylohyoid Muscle

The mylohyoid muscle is found above the anterior belly of the digastric muscle, arising from the mylohyoid line on the internal surface of the mandible from the third molar region posteriorly to almost the symphysis anteriorly. The direction of the fibers is toward the midline, where they form a tendinous raphe. The posterior fibers insert into the body of the hyoid bone. The mylohyoid therefore forms the floor of the oral cavity. The diaphragm formed is thicker in the free posterior margin, resulting in an important surgical landmark. The lingual nerve, deep process of the submandibular gland, and hypoglossal nerve pass deep to the posterior border.

Innervation is by the mylohyoid branch of the inferior alveolar nerve; vascular supply is via the submental artery, which is a branch of the facial artery. The principal action of the mylohyoid muscle is elevation of the tongue.

Geniohyoid Muscle

The geniohyoid muscle is situated above the mylohyoid muscle and arises from the inferior genial tubercle behind the mandibular symphysis. It inserts into the front of the body of the hyoid bone. The muscles of the right and left make contact and may fuse. The fibers proceed downward and slightly posterior to attach to the upper half of the hyoid bone.

Innervation is provided by the hypoglossal nerve, which consists of branches of the first and second cervical nerves. The action of the geniohyoid muscle is to pull the hyoid bone up and forward, or to pull the mandible down and posteriorly.

Stylohyoid Muscle

The stylohyoid muscle is a slender muscle arising from the lateral and inferior surfaces of the styloid process. Fibers insert into the hyoid bone at the junction between the body and greater horn. The tendon of the digastric muscle commonly splits the stylohyoid muscle near its insertion.

Innervation is provided by the facial nerve. The muscle functions as an elevator and retractor of the hyoid bone or as a stabilizer of the hyoid bone during other muscle functions.

INFRAHYOID MUSCLES

The infrahyoid muscles are four straplike muscles that anchor the hyoid bone to the sternum, clavicle, and scapula (Fig. 10-24). Their function is to depress the hyoid and larynx or to stabilize and fix the hyoid bone in position during contraction of the suprahyoid muscle group.

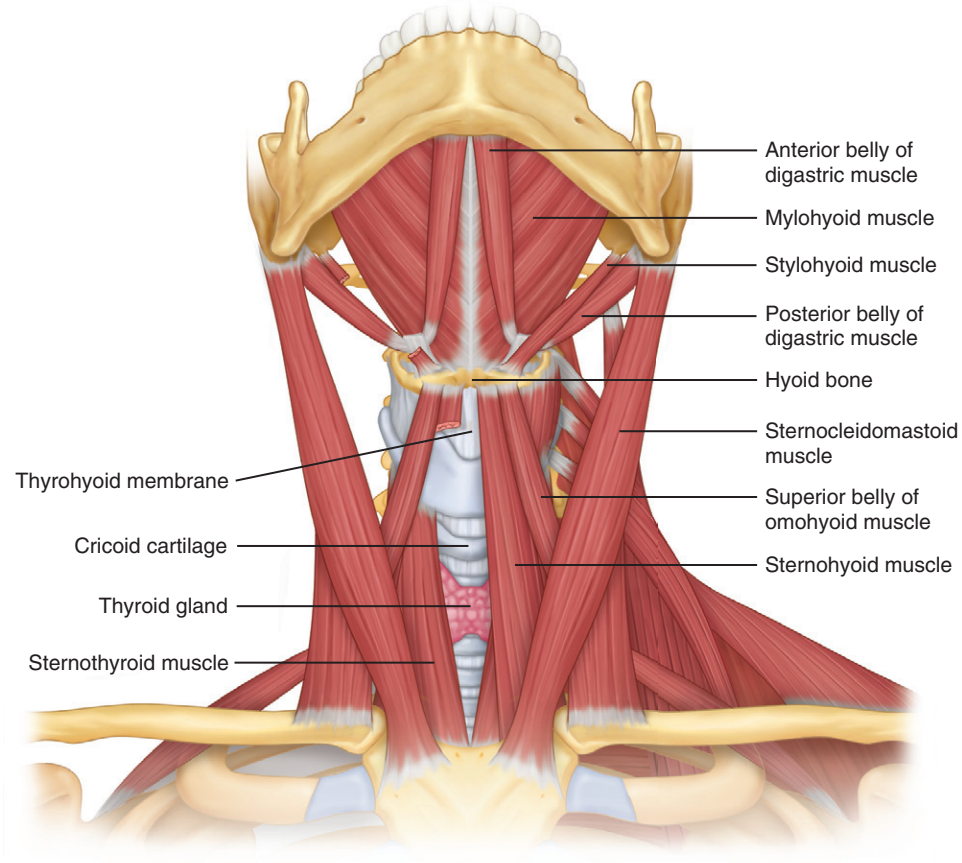


FIGURE 10-24 Infrahyoid muscles.

Sternohyoid Muscle

The sternohyoid muscle arises from the back of the manubrium and medial end of the clavicle. The muscle fibers run superiorly and converge, but remain separated by the fascia termed the linea alba colli. The sternohyoid fibers attach to the inferior border of the hyoid bone.

Omohyoid Muscle

The omohyoid muscle consists of two bellies united by a tendon. The muscle origin is the upper border of the scapula near the suprascapular notch. The inferior belly runs forward and upward under the sternomastoid muscle to the tendinous attachment. The superior belly runs upward and inserts into the lower border of the hyoid bone. The intermediate tendon is attached to the manubrium and to the first costal cartilage by a facial sling.

Sternothyroid Muscle

The sternothyroid muscle lies under the sternohyoid muscle, where it arises from the back of the manubrium and inserts into the thyroid cartilage.

Thyrohyoid Muscle

The thyrohyoid muscle originates where the sternothyroid muscle attaches at the oblique line of the thyroid cartilage and, therefore, may be considered an extension of this muscle. It inserts into the lateral part of the greater horn of the hyoid bone.

Innervation of the infrahyoid muscles is by the first, second, and third cervical nerves. The sternohyoid, omohyoid, and sternothyroid muscles are supplied by the ansa cervicalis, whereas the thyrohyoid muscle receives innervation from the hypoglossal nerve.

SOFT PALATE MUSCULATURE

The soft palate and pharynx are functionally and anatomically related (Fig. 10-25).[2-4,6,10] Although rarely involved in minor traumatic episodes, they are frequently involved in gunshot wounds and more severe traffic accidents. Their anatomic description will be addressed here.

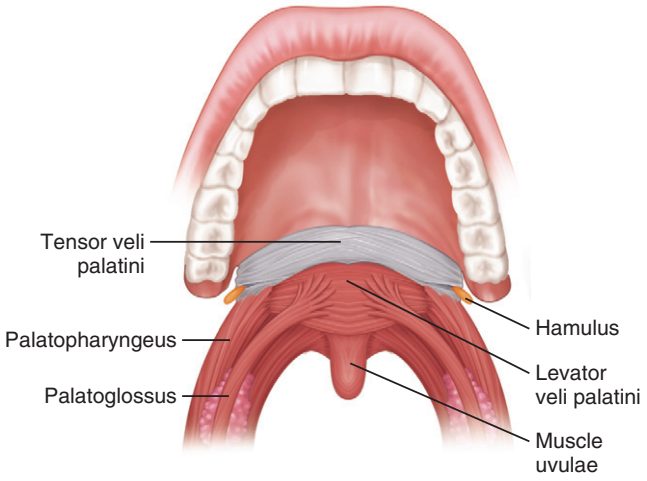


FIGURE 10-25 Soft palate musculature.

Levator Veli Palatini

Arising from the petrous portion of the temporal bone and eustachian tube, the levator veli palatini also extends medially and inferiorly to join its counterpart from the opposite side. The levator veli palatini functions to elevate the vertical posterior portion of the soft palate. It functions in harmony with the tensor veli palatini and pharyngeal constrictors to close the oral pharynx from the nasal pharynx.

Motor innervation is via the vagus nerve (cranial nerve [CN] X); sensory innervation is from the pharyngeal plexus and CN IX.

Tensor Veli Palatini

Taking its origin from the base of the medial pterygoid plate, spine of the sphenoid bone, and eustachian tube, the tensor veli palatini then courses vertically to the pterygoid hamulus, where it passes around and courses horizontally into the soft palate. Its function is to raise or tense the soft palate and open the eustachian tube during swallowing. Innervation is via the mandibular division of the trigeminal nerve (CN V).

Muscle of Uvula

The muscle of uvula arises from the posterior nasal spine and aponeurosis of the palate. The muscle passes posteriorly and inserts on the mucous membrane of the uvula. Its function is to shorten the uvula. The nerve supply is from the vagus.

PHARYNGEAL MUSCULATURE

See Figure 10-26.[2-4,6,10]

Superior, Middle, and Inferior Constrictors

The superior, middle, and inferior constrictor muscles make up the lateral and posterior walls of the pharynx and function in harmony to close or constrict the pharynx. Their origins, from superior to inferior respectively, are the medial pterygoid plate, pterygomandibular raphe, which is the upper border of the greater cornu of the hyoid, and larynx. They all insert on the fibrous median raphe. The three are paired and overlap each other vertically. Innervation is via the vagus nerve by the pharyngeal plexus.

Dilators and Elevator Muscles

Palatopharyngeus Muscle. This is the posterior tonsillar pillar. It originates in the soft palate and passes vertically to insert on the posterior border of the thyroid cartilage. Innervation is via the pharyngeal plexus.

Salpingopharyngeus Muscle. This is generally considered part of the palatopharyngeus muscle. It runs vertically from the auditory tube to blend with the palatopharyngeus muscle. It is also innervated by the pharyngeal plexus.

Palatoglossus Muscle. This muscle forms the anterior tonsillar pillar, arising from the anterior aponeurosis of the soft palate and inserting on the lateral side of the tongue, where it merges with the transverse fibers of the

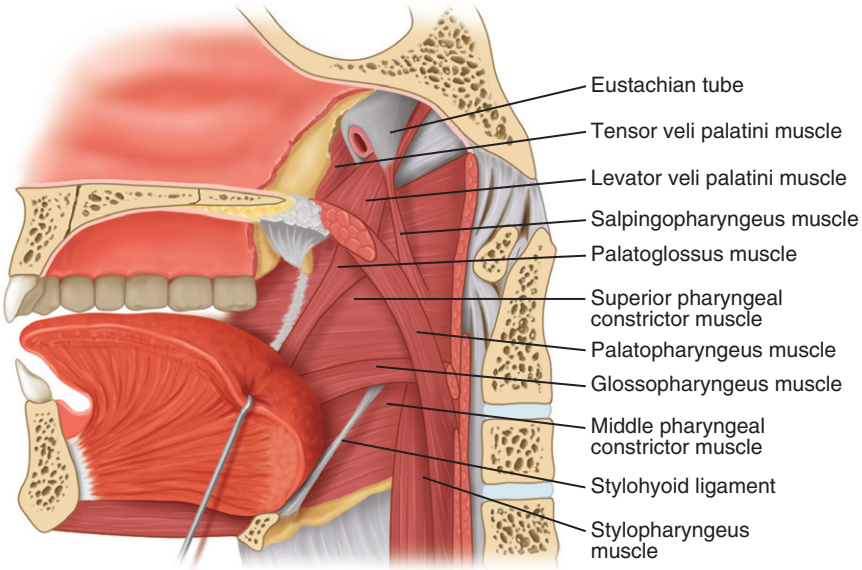


FIGURE 10-26 Pharyngeal musculature.

tongue. Innervation is from the pharyngeal plexus of nerves.

Stylopharyngeus Muscle. Arising from the styloid process, this cylindrical muscle passes inferiorly between the superior and middle pharyngeal constrictors. As it passes behind the middle constrictor, it spreads and unites with its counterpart from the opposite side. Its function is to dilate or widen the pharynx. The stylopharyngeus muscle is innervated by a branch of the glossopharyngeal nerve.

ARTERIAL BLOOD SUPPLY TO THE HEAD AND NECK

Generally speaking, the external carotid artery and its branches are responsible for the arterial blood supply to the facial region (Fig. 10-27).[4,6,16,18] Exceptions include those areas supplied by branches of the internal carotid artery to the upper face and portions of the nasal cavity.

The aortic arch is the origin of the common carotid artery on the left side. The right common carotid artery is a branch of the brachiocephalic or innominate artery.

Surgical Note: When performing ligation of the external carotid artery, it is important that the surgeon be cognizant of the relationship of the external and internal carotid arteries at their origin. At this point, the internal carotid artery is posteromedial and the external carotid artery is anterolateral. The level of this division is generally at the superior border of the thyroid cartilage.

EXTERNAL CAROTID ARTERY

During its earliest course, the external carotid artery is superficial, lying below the investing layer of deep cervical fascia, platysma muscle, superficial fascia, and skin. As it progresses superiorly, it runs through the submandibular triangle to the retromandibular fossa, through the substance of the parotid gland to the level of the mandibular neck. Here it gives off its branch, the superficial temporal artery, and continues deep to the condyle, turning medially and anteriorly as the internal maxillary artery (see Fig. 10-27).

Branches of the External Carotid Artery

Superior Thyroid Artery. The superior thyroid artery arises from the common carotid artery or from the front of the external carotid artery below the hyoid bone and under the sternocleidomastoid muscle. Its course is downward and forward, deep into the infrahyoid muscles to the apex of each lobe of the thyroid gland, where it divides into infrahyoid, superior laryngeal, cricothyroid, and glandular branches.

Lingual Artery. The lingual artery arises from the front of the external carotid artery as a common vessel with the facial artery or as a separate entity. The artery lies level with or above the hyoid bone. Its course begins horizontally forward of the posterior border of the hyoglossus muscle, continuing forward deep into the hyoglossus muscle along the upper border of the hyoid bone lying on the middle constrictor muscle. Superiorly, it reaches a space between the genioglossus and inferior longitudinal muscles of the tongue. At this point, it turns horizontally and runs along the lower surface of the tongue, toward its tip, in a tortuous fashion. In severe facial trauma with penetrating wounds, ligation of the lingual artery may be necessary. Branches include the following.

Suprahyoid Artery. The suprahyoid artery runs along the upper border of the hyoid bone, sending branches to muscles attached to the bone and anastomosing with the opposite side.

Dorsales Linguae Artery. The dorsales linguae artery arises under the hyoglossus muscle and ascends to the dorsum of the tongue.

Sublingual Artery. The sublingual artery arises at the anterior border of the hyoglossus muscle. From there, it

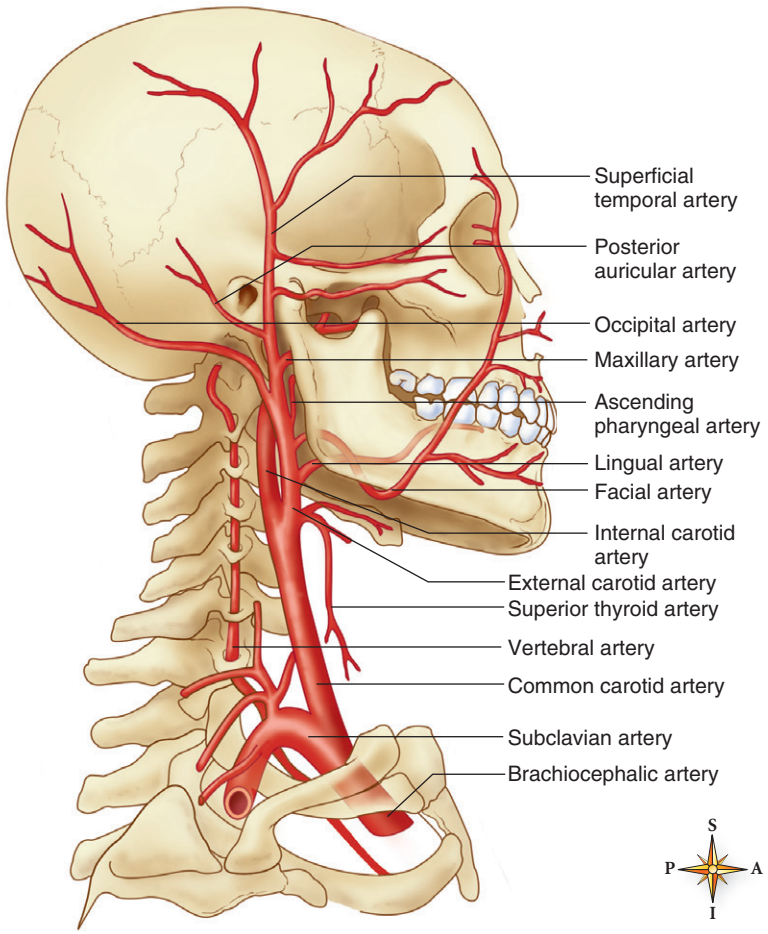


FIGURE 10-27 Arterial blood supply to the head and neck. (From Thibodeau: *Anatomy and physiology*, ed 7, St. Louis, 2010, Mosby.)

runs forward in the floor of the mouth medially to the sublingual gland, which it supplies, and along with mucous membranes of the floor of the mouth and mylohyoid muscle.

Deep Lingual Artery. This is actually the terminal continuation of the lingual artery in the substance of the tongue. It supplies the tongue and then forms an anastomosis with the opposite side, the arcus raninus.

Facial Artery. The facial artery arises from the front of the external carotid artery in common with the lingual artery (linguofacial trunk) or as a separate branch just below the posterior belly of the digastric muscle. It ascends in the carotid triangle and enters a groove on the posterior border of the submandibular gland. It turns downward and forward between the submandibular gland and medial pterygoid muscle, where it then winds around the lower border of the mandible at a point in front of the anterior border of the masseter muscle. At this point, the facial artery is usually located anteriorly to the facial vein. The facial artery runs upward and forward on the face, ending as the angular artery at the medial angle of the eye by anastomosing with branches of the ophthalmic artery. Its general course is tortuous throughout the face, with considerable individual variations.

Surgical Note: Ligation of the facial artery and vein is often required during the open approach to the inferior mandible. The facial artery is generally considered to have cervical and facial divisions.

Cervical Division

1. *Ascending palatine branch.* The origin of this branch is close to the lateral pharyngeal wall and runs along the outer surface of the superior pharyngeal constrictor. It accompanies the levator veli palatini and supplies the soft palate, portions of the pharynx, and the tonsils.
2. *Tonsillar branch.* This is the main artery to the tonsil. It may arise from the anterior border of the masseter muscle. The facial artery also runs upward with the ascending palatine branch.
3. *Glandular branch.* This branch supplies the submandibular gland.
4. *Submental branch.* This is the largest branch given off by the facial artery in the neck. Its course is forward on the mylohyoid muscle, where it supplies muscles in the area; it then turns upward over the lower border of the mandible.

Facial Division

1. *Inferior labial artery.* This artery penetrates the orbicularis oris muscle, supplying the skin, muscles, and mucous membranes of the lower lip. From here it anastomoses with the opposite side.
2. *Superior labial artery.* This branch is larger than the inferior branch. It has a course and distribution

similar to the inferior labial artery, although to the upper lip.

3. *Lateral nasal branch.* This branch supplies the ala and dorsum of the nose.
4. *Angular artery.* This is the termination of the facial artery. It anastomoses with the dorsal nasal and palpebral branches of the ophthalmic artery, thereby establishing a possible route of communication between the external and internal carotid arteries.

Occipital Artery. The occipital artery arises from the back of the external carotid artery at about the same level as the facial artery; however, its origin can vary in either direction. Near its origin, the hypoglossal nerve winds around it. The artery runs through the carotid triangle backward and upward to the lower border of the posterior belly of the digastric muscle and then crosses the internal carotid artery and internal jugular vein. Under the sternocleidomastoid muscle, it occupies the occipital groove on the temporal bone. Posterior to the sternocleidomastoid muscle, it pierces the trapezius muscle and divides into various scalp branches. Branches include a sternomastoid branch, a mastoid branch, a descending branch, which can be important because of anastomoses with the opposite side during external carotid ligation, meningeal branches, and occipital terminal branches.

Posterior Auricular Artery. The posterior auricular artery arises from the back of the external carotid artery above the posterior belly of the digastric muscle. It follows the stylohyoid muscle upward under cover of the parotid gland and terminates between the mastoid process and the auricle. Branches include the stylomastoid, posterior tympanic auricular, and occipital branches.

Ascending Pharyngeal Artery. The ascending pharyngeal artery is a small vessel that is the only medial branch of the external carotid artery. It arises low, just before the division above the common carotid artery, and ascends between the internal carotid artery and pharyngeal wall to the base of the skull. Branches are given off to the wall of the pharynx and adjacent muscles. Near the base of the skull, there is an anastomosis with the pterygoid artery of the maxillary artery.

Superficial Temporal Artery. The superficial temporal artery is the smaller terminal branch of the external carotid artery, with its origin in the parotid gland behind the neck of the mandible. It crosses the zygomatic arch and divides into frontal and parietal branches. The auriculotemporal nerve runs posteriorly to the superficial temporal artery. Branches include the transverse facial artery, which arises in the parotid gland and runs forward across the masseter muscle between the zygomatic arch above—and the duct of the parotid gland below—and is accompanied by zygomatic branches of the facial nerve. There it supplies the parotid gland and duct, the masseter muscle, the skin, the auricle, and the joint capsule. The terminal branches divide above the zygomatic arch into the frontal and parietal branches. There are also deep branches during its course to the middle temporal artery.

Surgical Note: The identification and protection of the superficial temporal artery are important during open procedures involving the condyle. Incision and flap design are usually such that the artery is anterior to the approach and is reflected in the soft tissue flap.

Maxillary Artery. The maxillary artery is the larger of the terminal branches arising in the parotid gland behind the neck of the mandible. At this point, the maxillary artery turns at right angles to the superficial temporal branch, where it courses anteriorly and upward through the infratemporal fossa. There is a varying relation between the artery and lateral pterygoid muscle.[14] In more than 50% of individuals, the artery is on the outer side of the muscle, passing between the mandible and sphenomandibular ligament. In the rest, the artery is located medially to the lateral pterygoid muscle.

Branches of the maxillary artery are numerous and complicated (Fig. 10-28). In an attempt to group the branches for ease of understanding, they are divided into the following parts.

Mandibular Part of the Maxillary Artery. The first part runs forward between the neck of the mandible and the sphenomandibular ligament. Most of its branches accompany those of the mandibular nerve.

Deep Auricular Artery. This artery ascends through the parotid gland to supply the meatus and tympanic membrane.

Anterior Tympanic Artery. This artery supplies the tympanic membrane.

Middle Meningeal Artery. Clinically, this is the most important branch of the maxillary artery. It runs upward between the sphenomandibular ligament and lateral pterygoid muscle. It passes between the two roots of the auriculotemporal nerve and enters the cranium via the foramen spinosum, which is located in the sphenoid bone (Fig. 10-29). It then divides into a tympanic branch and splits into anterior and posterior branches.

Surgical Note: This artery, because of its medial location to the condyle, could be potentially damaged during open procedures in the condylar region or directly damaged by severe condylar displacement.

Inferior Alveolar Artery. This artery descends between the sphenomandibular ligament and ramus of the mandible. The nerve lies anteriorly, and enters the mandibular canal via the mandibular foramen. The branches of the inferior alveolar artery include the lingual and mylohyoid branches before entering the canal and the dental, mental, and incisor branches after entering the canal. In the mandibular canal, the artery sends branches into the marrow spaces and the teeth via apical branches and periodontal branches, with the mental artery branching off and exiting via the mental foramen to supply the soft tissue of the chin. The continuation terminal branch is the incisive artery, which continues its course within the mandible to anastomose with the incisive artery of the opposite side.

Second Part of the Maxillary Artery. The second portion of this artery consists of the following.

Anterior and Posterior Deep Temporal Arteries. These arteries ascend between the temporalis muscle and skull to supply the temporalis muscle.

Masseteric Artery. This artery travels with the corresponding masseteric nerve behind the temporalis muscle to pass through the coronoid notch and enter the deep

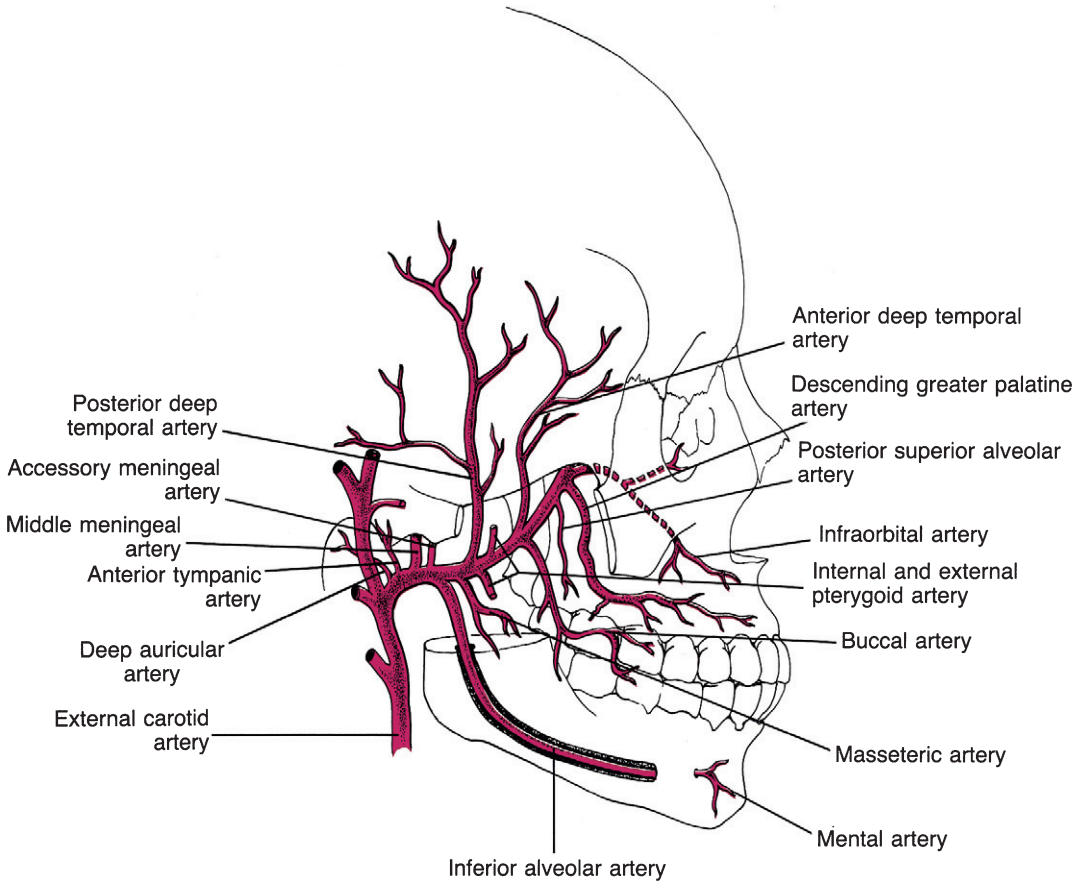


FIGURE 10-28 Maxillary artery and branches.

surface of the masseter muscle. It also has branches supplying the joint capsule.

Pterygoid Branches. These branches supply the pterygoid muscles.

Buccal Artery. This artery accompanies the buccal nerve to the buccinator, which it supplies, along with the skin and mucous membranes of the cheek.

Third Part of the Maxillary Artery. This portion of the maxillary artery branches extensively and supplies the upper teeth, portions of the face and orbit, palate, and nasal cavity.

Posterior Superior Alveolar Artery. This artery takes a tortuous course down in the infratemporal fossa and onto the posterior surface of the maxilla. Here, small branches supply the gingiva and dental branches, entering canals to the molars and premolars and to the maxillary sinus lining.

Infraorbital Artery. This artery arises in the pterygopalatine fossa; it enters the orbit through the inferior orbital fissure and courses anteriorly in the infraorbital sulcus, proceeding through the infraorbital canal, and finally through the infraorbital foramen with the corresponding nerve. Before leaving the canal, the anterior and superior alveolar branches form. Branches develop and supply various orbital muscles throughout its route through the orbit.

Descending Palatine Artery. This artery is one of the terminal branches arising in the pterygopalatine fossa. After descending through the pterygopalatine canal, the greater palatine artery develops and passes through the greater palatine canal, running forward to the roof of the mouth and supplying the hard palate. The lesser palatine artery passes through the lesser palatine canal to supply the soft palate.

Artery of the Pterygoid Canal. This vessel frequently arises from the descending or greater palatine artery, which runs backward through the pterygoid canal along with the corresponding nerve.

Sphenopalatine Artery. This is the last of the terminal branches. It enters the nasal cavity through the sphenopalatine foramen and gives off the posterior and lateral nasal arteries, which then proceed to the conchae, meatus, and paranasal sinuses. It eventually terminates in the nasal septum and, as such, is important in epistaxis.

Internal Carotid Artery

The origin of the internal carotid artery[4,6] is approximately at the level of the thyroid cartilage (see Fig. 10-27). It is at first behind and medial to the external carotid artery but, during its course, it moves away from the external carotid artery and eventually separates near the styloglossal and stylopharyngeal muscles. It enters the middle cranial fossa through the carotid canal and travels into the cavernous sinus, dividing into the anterior and middle cerebral arteries.

No branches form during the internal carotid artery's course through the neck. Its curved shape can follow neck movements without stretching. The internal carotid artery is closely related to the internal jugular vein and

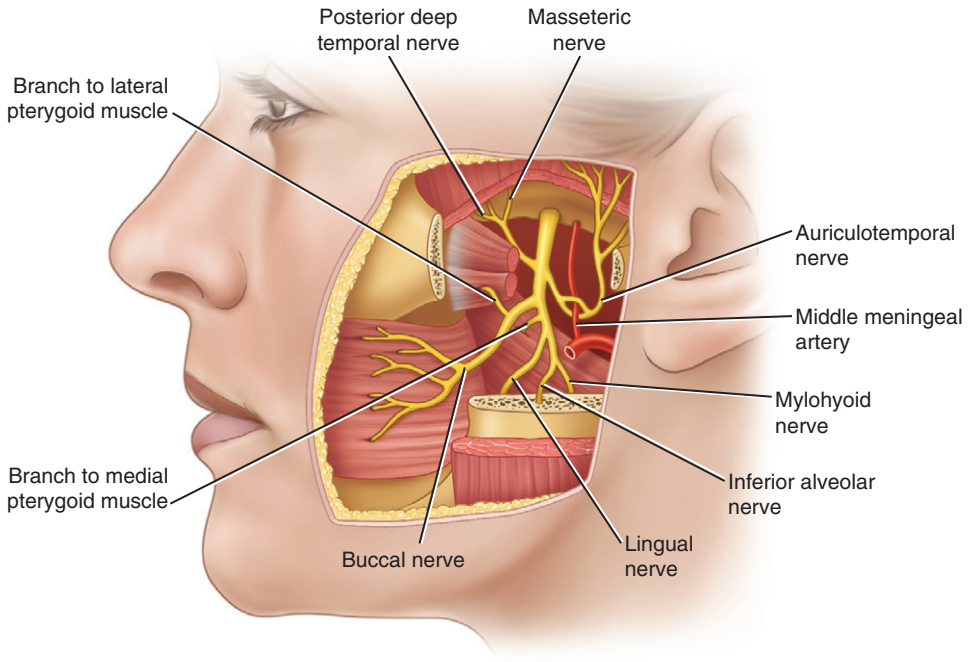


FIGURE 10-29 Mandibular division of the trigeminal nerve.

the vagus nerve. The internal jugular vein travels in a half-spiral trajectory around the artery.

After leaving the cavernous sinus, the internal carotid artery branches into several other arteries; the most significant is the ophthalmic artery, which follows the optic nerve into the orbit. This branch supplies the eyeball, muscles, lacrimal gland, and eyelids. Other branches of note include the central artery of the retina, posterior and anterior ethmoid branches to the nasal cavity, medial and lateral palpebral branches, and supraorbital branch. The final facial branch of the ophthalmic artery is the nasal branch.

VEINS OF THE HEAD AND NECK

Venous drainage to the head and neck (Fig. 10-30)[4,6,16] can be considered from the standpoint of being superficial and deep in function. The superficial drainage is mainly via the external and anterior jugular veins, whereas most of the deep venous drainage is via the internal jugular vein. There is significant anastomosis between all veins, intracranially and facially, and superficially and deep. The superficial veins empty into the internal jugular vein at a low point and, in turn, it joins the subclavian vein to form the brachiocephalic or innominate vein behind the sternoclavicular articulation.

Consideration of the potential disruption of a vein with subsequent bleeding is always important in the trauma patient, as is a potential retrograde spread of infection in the postoperative phase.

INTERNAL JUGULAR VEIN

Beginning at the jugular foramen, the internal jugular vein is located posteromedially to the internal carotid artery. During its descent, the vein is closely related not only to the internal carotid artery but also to the vagus nerve. Various deep structures drain into the internal jugular vein. Those of relevance here include the common facial vein, which drains the superficial and deep parts of the face, and lingual and sublingual veins.

COMMON FACIAL VEIN

The common facial vein drains from an area that corresponds more or less with the distribution of the facial, maxillary, and superficial temporal arteries. It originates from the intersection of the facial and retromandibular veins near the angle of the mandible. The common facial vein empties into the internal jugular vein at the level of the hyoid bone.

Surgical Note: The common facial vein is often violated in cases of traumatic hemorrhage or iatrogenic hemorrhage during various approaches to the maxilla, zygoma, condylar ramus, or body of the mandible. Ligation, pressure, and hemostatic cautery are all useful for controlling venous bleeding in these areas.

ANTERIOR FACIAL VEIN

The frontal vein, which drains the anterior scalp region, empties into the angular vein at about the bridge of the nose, where the angular vein continues downward toward the cheek. Near the commissure, the facial vein descends along with the facial artery, where it crosses the inferior border of the mandible close to the anterior edge of the masseter muscle attachment. The vein is normally posterior to the artery at this location.

Surgical Note: The vein is normally identified, ligated, and cut during submandibular approaches to the mandible.

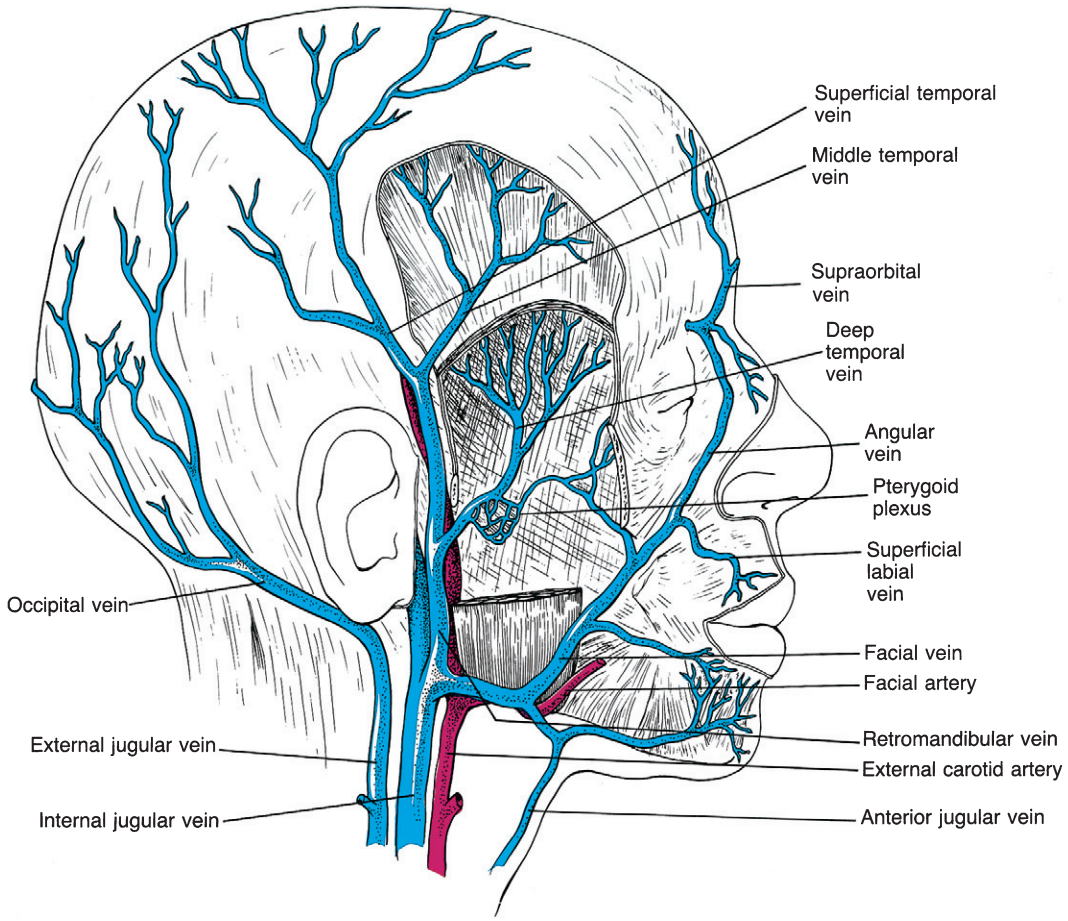


FIGURE 10-30 Veins of the head and neck.

RETROMANDIBULAR VEIN

At approximately the level of the neck of the mandible, the retromandibular vein is created by the junction of the superficial temporal and maxillary veins. The vein is located within the substance of the parotid gland, from which it descends to leave the gland at its lower pole. Initially, the vein and external carotid artery are closely related but, as it descends into the face, the vein and artery diverge. In the preauricular region, the superficial temporal artery is often closely related to two veins—the superficial temporal vein and the more anteriorly located middle temporal vein. Surrounding the maxillary artery in the infratemporal fossa is the pterygoid plexus of veins. The pterygoid plexus empties into the retromandibular vein below the level of the neck of the mandible medially.

Surgical Note: Many of the deep veins of the face drain into this plexus, which is an area for potentially significant bleeding during mobilization of Le Fort–type maxillary fractures, which separate at or through the pterygoid plates. Because this is essentially a blind area during the treatment of most fractures, pressure and local measures are the only methods of hemostasis available.

EXTERNAL JUGULAR VEIN

The posterior auricular and occipital veins join below the ear to form the superficial external jugular vein. From this point, the vein descends across the sternocleidomastoid muscle and eventually drains into the internal jugular vein.

ANTERIOR JUGULAR VEIN

There is notable variability in the position and presence or absence of one or both sides of the anterior jugular vein. This vein is located anteriorly, or even as a single midline vein, coursing inferiorly around the anterior aspect of the sternocleidomastoid muscle to empty into the internal jugular vein.

NEUROLOGIC ANATOMY*

Surgical Note: The most common injuries to nerves of the head and neck subsequent to major maxillofacial trauma involve the superficially positioned facial nerve or the

*References 4, 6, 16, 18, 32, 29, 34, and 47.

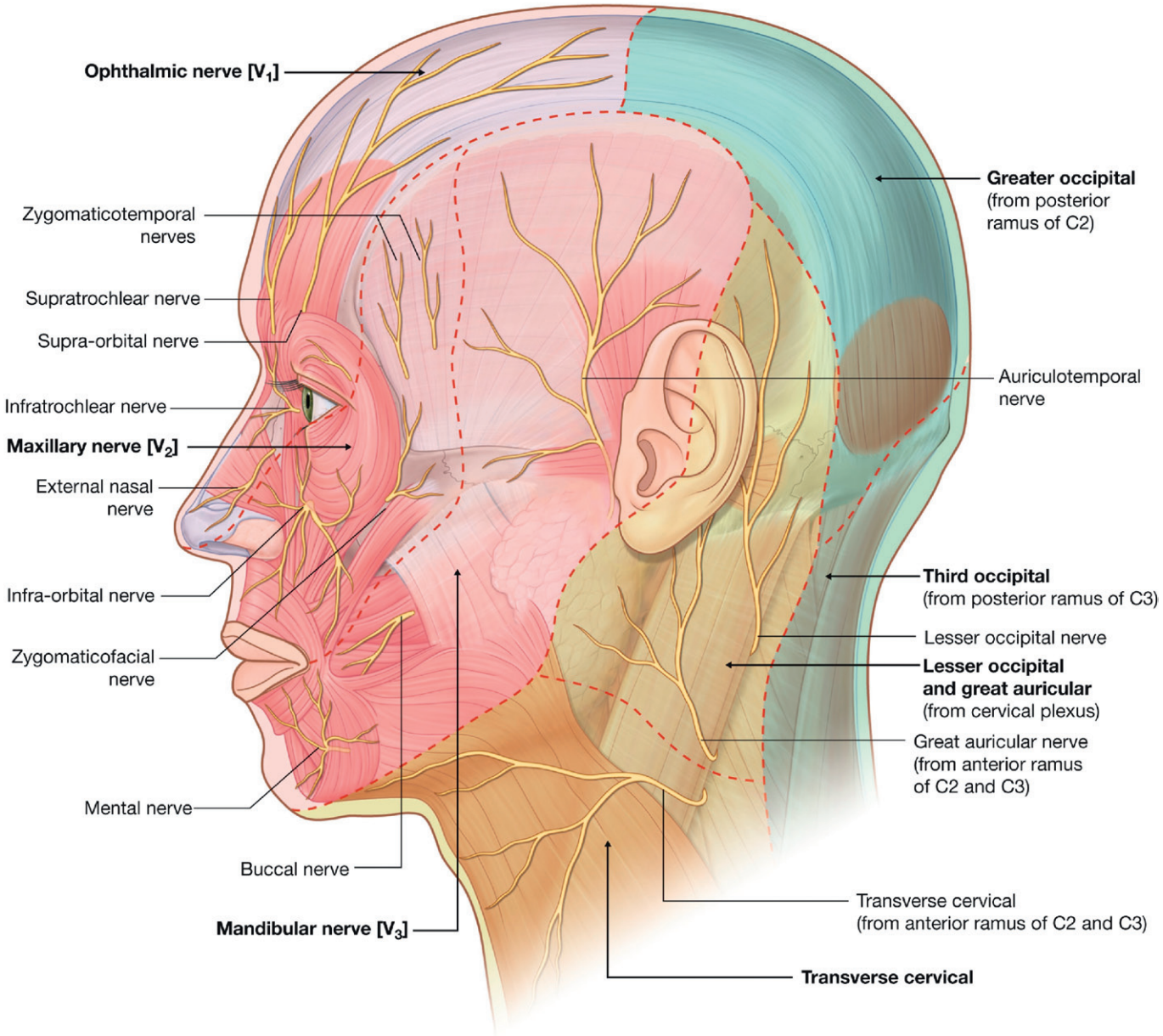


FIGURE 10-31 Cutaneous sensory distribution of the trigeminal nerve. (From Drake R, Vogl AW, Mitchell A: *Gray's anatomy for students*, ed 2, Philadelphia, 2009, Churchill Livingstone.)

cranial nerves found within bony canals that are fractured, such as the inferior alveolar nerve, infraorbital nerve, and optic nerve. The surgeon must also be cognizant of neuroanatomy when designing soft tissue flaps or applying any form of a stabilizing appliance. Discussion in this chapter is limited to the trigeminal, facial, and hypoglossal nerves.

TRIGEMINAL NERVE

The trigeminal nerve is composed of sensory and motor fibers. The sensory distribution essentially covers the entire anterior head and face, whereas the motor division innervates the muscles of mastication. The trigeminal nerve arises from the ventral surface of the cerebral pons. The semilunar or gasserian ganglion, which is the sensory root ganglion, is found in Meckel's cavity near the foramen lacerum. From this location, the three sensory divisions of the trigeminal nerve arise. The motor portion eventually joins the mandibular division in its course. The first division of the trigeminal nerve is the ophthalmic nerve, which enters the orbit through the medial part of the superior orbital fissure. The second division is the maxillary nerve, which exits the foramen rotundum and goes into the pterygopalatine space. The mandibular nerve, or third division, leaves the foramen ovale and continues into the infratemporal fossa. Figure 10-31 illustrates the normal cutaneous innervation of the three divisions of the trigeminal nerve. The mandibular angle region is variably innervated by branches of the two upper cervical nerves.

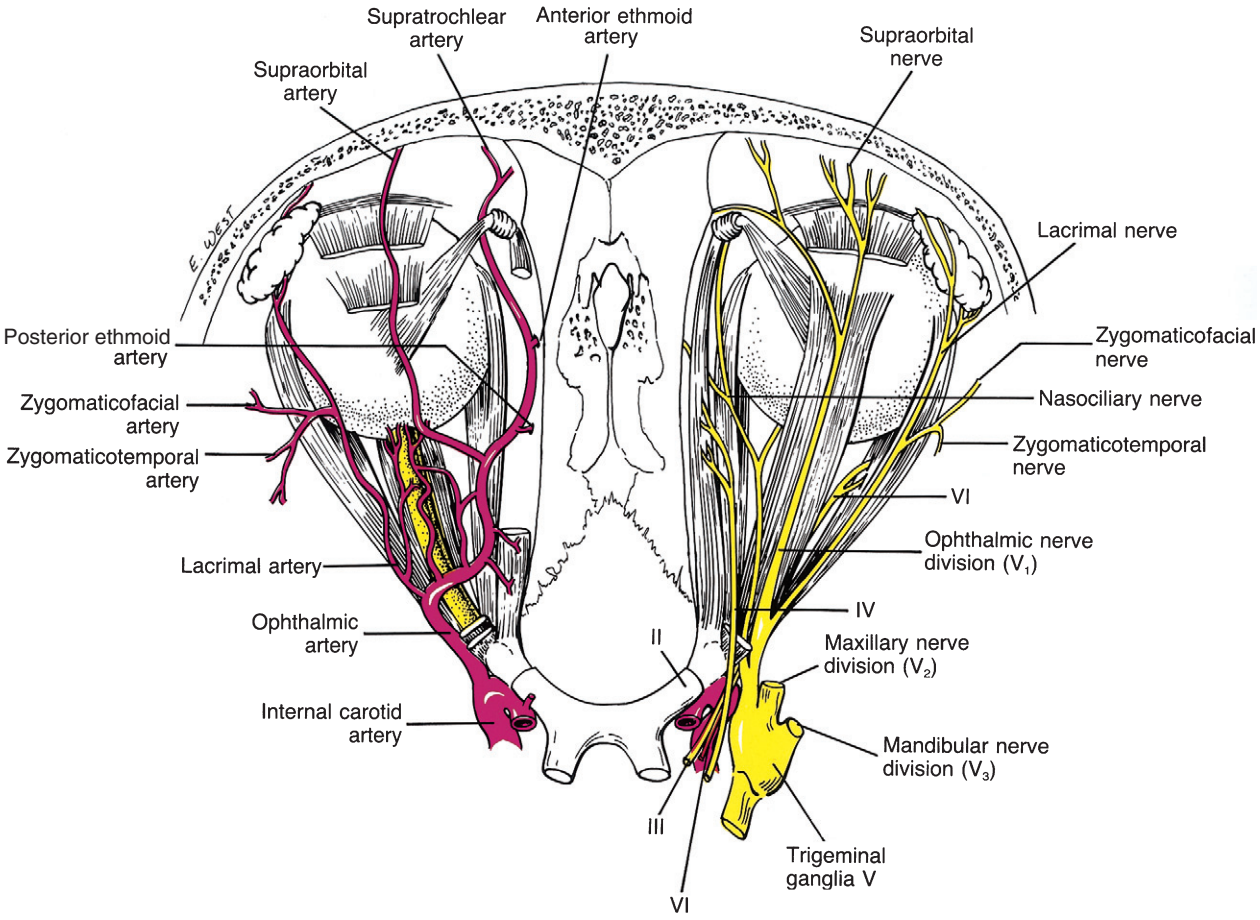


FIGURE 10-32 Superior orbital view. *Right,* Nerves and musculature. *Left,* Cutaway showing arterial supply.

Ophthalmic Nerve

In the orbit, the ophthalmic nerve (Fig. 10-32) divides into three branches. The nasociliary nerve travels along the medial orbital roof, where it branches into the nasal cavity. The frontal nerve proceeds anteriorly to the skin of the forehead and the lacrimal nerve courses along the lateral orbital roof to the lacrimal gland and the skin at the corner of the eye.

Maxillary Nerve

After exiting the skull through the foramen rotundum, the maxillary nerve (Figs. 10-33 and 10-34; see also Fig. 10-32) enters the pterygopalatine fossa, where it splits into three major branches.

Pterygopalatine Nerve. Shortly after leaving the main trunk of the maxillary nerve, the pterygopalatine nerve seems to enter the pterygopalatine ganglion, but instead the fibers are only closely adherent. Near the ganglion, the superior and posterior nasal branches enter the sphenopalatine foramen into the nasal cavity, where they supply the middle nasal concha. Lateral branches supply the upper and middle conchae, whereas medial branches supply the septum, and terminal branches enter the nasopalatine or incisive foramen to innervate the incisor teeth, gingiva, and palatal tissue.

The major portion of the pterygopalatine nerve continues through the pterygopalatine canal. While in the canal, some small branches enter the nasal cavity and supply the inferior nasal concha, along with the middle and inferior nasal meatus. Further into the canal, a branch enters the greater palatine foramen and sends branches anteriorly in the palate. Another branch passes through the lesser palatine foramen and supplies the tonsillar and soft palatine tissues.

Infraorbital Nerve. This nerve courses from the infratemporal fossa through the inferior orbital fissure and into the orbit, where it runs in the infraorbital groove, which transforms into the infraorbital canal and then exits through the infraorbital foramen to supply superficial structures of the face. Branches of the infraorbital nerve include the posterior and superior alveolar nerve, which exits from the infratemporal fossa. From here, it divides and sends branches through the posterior maxillary wall and into the maxillary sinus. The middle superior alveolar nerve leaves the infraorbital nerve while in the groove, where it enters the superior aspect of the maxillary sinus. The anterior and superior alveolar nerves leave while the infraorbital nerve is in the canal, which sends branches to the maxillary sinus and nose. Sensory distribution is provided to the maxillary teeth, alveolar bone, periodontal ligaments, and gingiva.

Zygomatic Nerve. In some cases, this nerve may be a branch of the infraorbital nerve. Its course is lateral through the orbit, where it sends a branch up to the

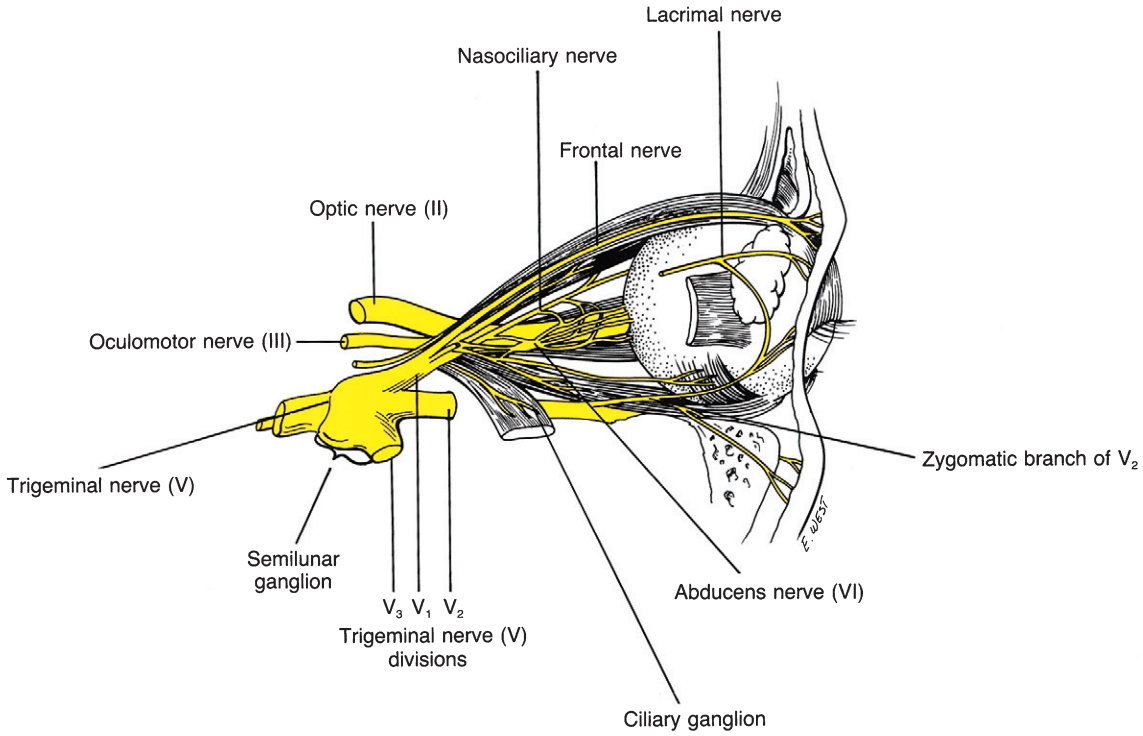


FIGURE 10-33 Lateral view of orbital nerves.

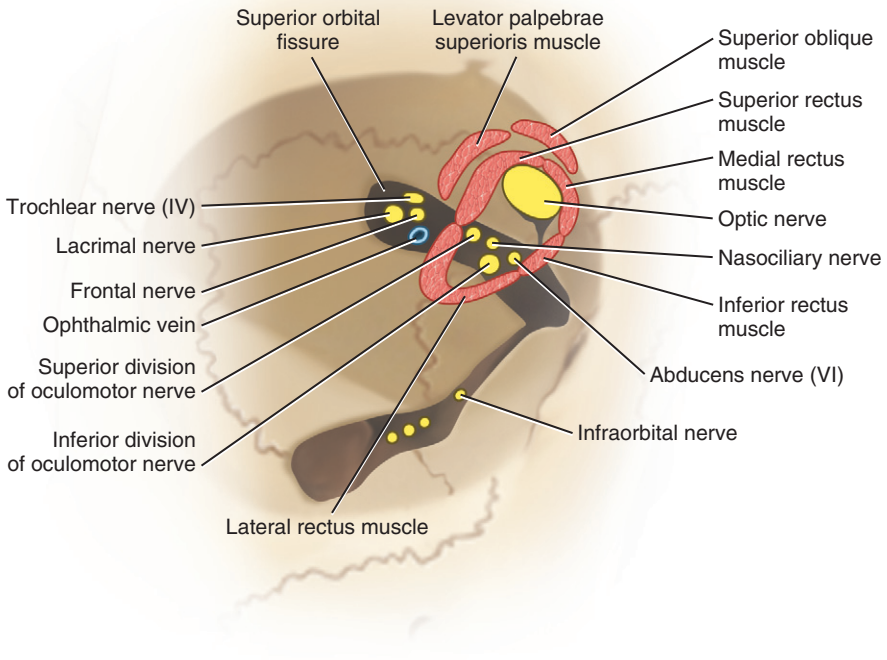


FIGURE 10-34 Orbital apex, superior orbital fissure, and relations of contents; right orbit.

lacrimal nerve, which consists of postganglionic parasympathetic fibers from the pterygopalatine ganglion. The zygomatic nerve then continues through the zygomatico-orbital foramen and into the zygomatic bone. Here, a branch, the zygomaticofacial nerve, exits the bone and supplies the skin over the cheek, whereas the zygomaticotemporal nerve exits into the temporal fossa, where it supplies the skin of the temple region.

Mandibular Nerve

The mandibular nerve (see Fig. 10-29) contains sensory and motor nerve fibers and exits the skull through the

foramen ovale and into the infratemporal fossa. The motor branches to the muscles of mastication are given off as the masseteric nerve, posterior and anterior temporal nerves, medial pterygoid nerve, and lateral pterygoid nerve.

Buccal Nerve. The course of the buccal nerve begins near the greater wing of the sphenoid bone and runs along the medial surface of the lateral pterygoid muscle, where it passes between its two heads. It innervates the lateral pterygoid and temporalis muscles. The buccal nerve then follows the anterior edge of the temporalis muscle and perforates the buccinator muscle. The sensory distribution of the buccal nerve includes the buccal gingiva in the posterior mandible, cheek, and a portion of the mucous membranes of the upper and lower lips.

Lingual Nerve. During its initial course, the lingual nerve is found in close proximity to the inferior alveolar nerve. Later, it separates and is found anteromedially in relationship to this nerve. The lingual nerve descends between the medial and lateral pterygoid muscles. At the inferior border of the lateral pterygoid muscle, the chorda tympani fibers connect. As the lingual nerve passes beyond the lateral pterygoid muscle, it proceeds laterally along the lateral surface of the medial pterygoid muscle and then turns sharply anteriorly on the superior surface of the mylohyoid muscle. In the floor of the mouth, the course of the lingual nerve is very close to the oral mucous membranes, especially posteriorly in the area of the superior pole of the submandibular gland. As it moves anteriorly, the nerve runs medially and proceeds below the submandibular, or Wharton's, duct and ends by sending fibers into the tongue substance. The lingual nerve provides sensory distribution to the gingiva and mucous membranes of the mandibular lingual and mucous membranes of the floor of the mouth, and the lower and upper surfaces of the body of the tongue back to the circumvallate papillae. The lingual nerve carries terminal taste fibers to the anterior two thirds of the tongue. These fibers originate from the facial nerve and relay impulses to the lingual nerve via the chorda tympani nerve. The regional anatomy of the floor of the mouth will be discussed later.

Inferior Alveolar Nerve. As noted, in its initial course, the inferior alveolar nerve has a close relationship to the lingual nerve. After separating approximately 5 mm below the cranial base, the inferior alveolar nerve travels between the lateral and medial pterygoid muscles. The inferior alveolar nerve passes around the lower border of the lateral pterygoid muscle and then proceeds to the medial aspect of the ramus of the mandible to enter the mandibular foramen. It has been shown that in approximately 34% of mandibles, the neurovascular bundle divides soon after the beginning of the inferior alveolar canal.[44] Other studies have not shown as high an incidence, varying from 0.96% to 8%.[34] The superoinferior course of the nerve in the mandible is such that it descends to the lowest point near the first molar and then rises again. In a lateromedial position, the canal and bundle are closest to the lateral cortical plate in the third molar area, but the nerve remains fairly constant in its relationship to the medial cortical plate throughout its course (Fig. 10-35).[34] The mylohyoid nerve branches off the inferior alveolar nerve before entering the canal. This nerve runs downward and anteriorly in the mylohyoid groove before innervating the mylohyoid muscle, anterior belly of the digastric muscle, and sensory fibers of the chin area. Exiting the mental foramen, the inferior alveolar nerve divides into three terminal mental nerves, which supply sensation to the chin, lower lip, and mucous membranes of the lower lip.

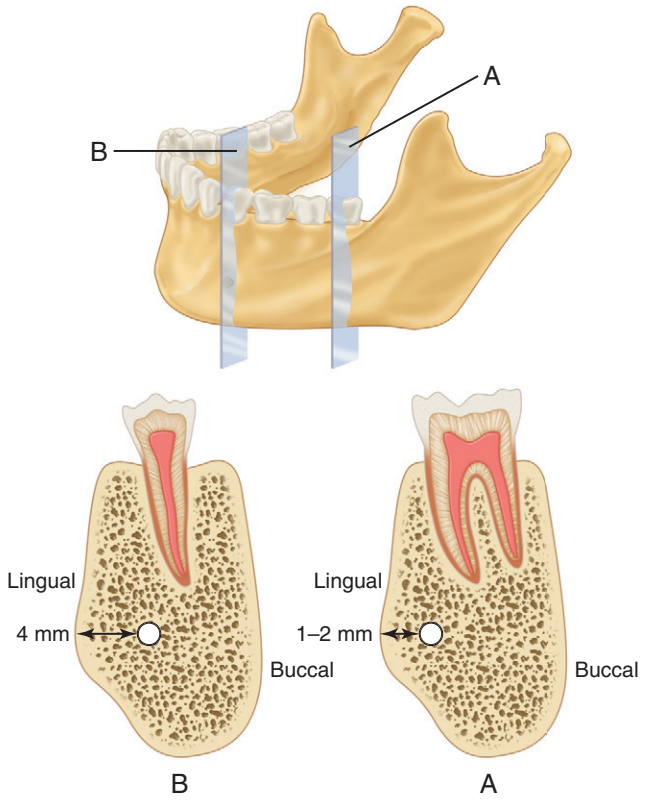


FIGURE 10-35 Position of the inferior alveolar canal as it travels anteriorly in the mandible.

Auriculotemporal Nerve. The auriculotemporal nerve separates from the mandibular nerve immediately below the cranial base and divides to encircle the middle meningeal artery. After reuniting, it courses downward and posteriorly, crossing the neck of the mandibular condyle. It enters the substance of the parotid gland and divides into two branches, one of which turns upward anterior to the outer ear—closely related to the superficial temporal artery crossing the zygomatic arch. Its position is important to consider in an open approach to the preauricular region. Terminal branches supply the outer ear, external auditory meatus, capsule of the mandibular joint, and areas of the parotid gland. Other terminal branches supply the skin in the greater posterior temple region.

FACIAL NERVE

The motor fibers of the facial nerve provide innervation to the muscles of facial expression, occipital and auricular muscles, platysma muscle, posterior belly of the

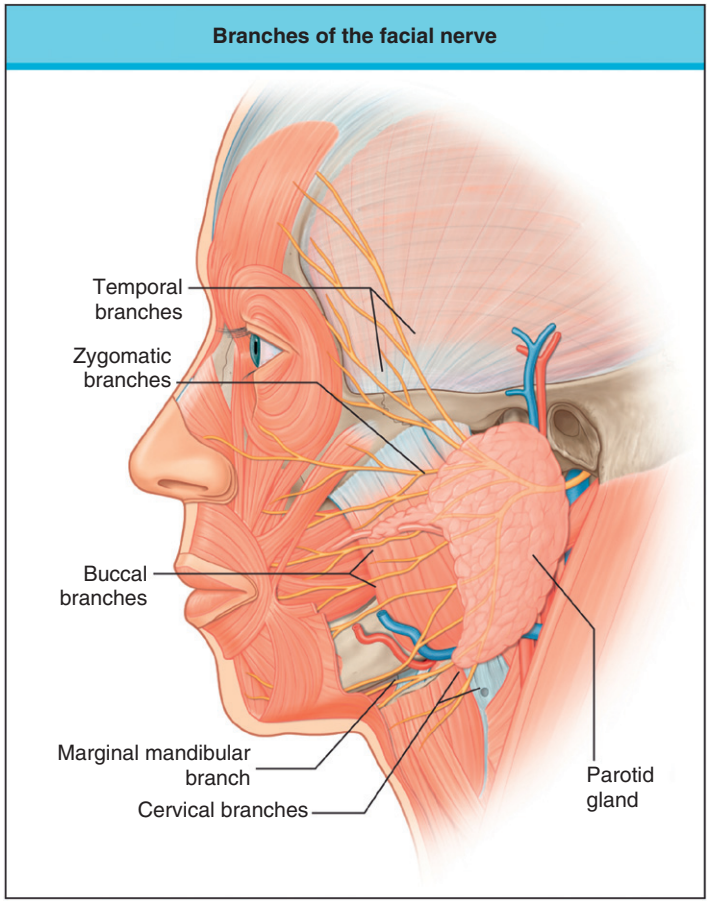


FIGURE 10-36 Distribution of the facial nerve. The exact configuration can vary considerably; this illustration is only an example. (From Langdon RC, Sattler G, Hanke CW: *Minimum incision face lift*. In Robinson JK, Hanke CW, Siegel DM, Sengelmann RD, editors: Surgery of the skin, St. Louis, 2005, CV Mosby.)

digastric muscle, and stylohyoid muscle. In addition, it supplies fibers for deep sensitivity of the face and for taste to the anterior two thirds of the tongue and palate. The facial nerve also transmits preganglionic visceral efferent fibers to the lacrimal gland, sublingual and submandibular glands, and other minor glands.

After leaving the brain, the facial nerve enters the inner auditory meatus. The nerve bends sharply at the tympanic cavity and travels posteriorly above the oval window, continuing to the posterior wall of the tympanic cavity. The facial nerve then leaves the canal and exits through the stylomastoid foramen.

Surgical Note: Damage to the facial nerve is possible in severe maxillofacial injuries with basilar skull fractures at any point near the pathway of the nerve and would result in ipsilateral paralysis of the muscles of facial expression.

Of concern to the surgeon is the close proximity of the main trunk of the facial nerve where it exits the stylomastoid foramen and mandibular condyle. After exiting the foramen, which is situated posterolaterally to the styloid process, the nerve enters the substance of the parotid gland, where it divides into its upper and lower divisions just posterior to the mandible. The approximate distance from the lowest point of the external bony auditory meatus to the bifurcation of the facial nerve is 2.3 cm. Posterior to the parotid gland, the nerve is at least 2 cm deep into the skin surface. From this point, the two branches curve around the posterior mandible, where they form a plexus between the parotid gland and masseter muscle. The terminal branches of the facial nerve then spread in a fanlike fashion as five separate nerves (Fig. 10-36). These branches are the temporal, zygomatic, buccal, mandibular, and cervical branches. There are often variations in the pattern of distribution.[16]

Temporal Branch

The temporal branch exits the parotid gland anterior to the superficial temporal artery. Muscles innervated via this branch include the auricular muscles, frontal muscle, superior portion of the orbicularis oculi muscle, and corrugator muscle to the eyebrows.

Surgical Note: During an open approach to the TMJ, violation of the nerve is possible. Often, temporary weakness is apparent because the patient cannot squeeze the ipsilateral eye together or wrinkle the forehead. This inability is usually caused by retractor trauma or soft tissue edema. If this is the case, it should resolve itself during the first postoperative week.

Zygomatic Branch

The course of the zygomatic branch is anterosuperior, crossing the zygomatic bone. Innervation is to the orbicularis oculi muscle.

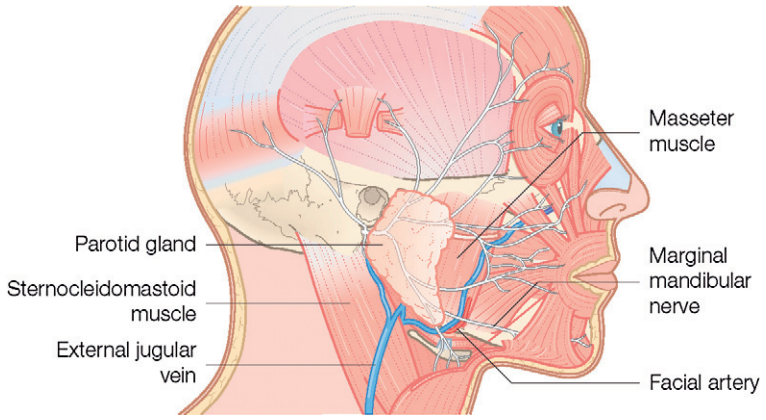


FIGURE 10-37 Marginal mandibular branch of the facial nerve and its relationship to the facial artery. (From Hanke CW, Sattler G: *Procedures in cosmetic dermatology series: Liposuction*, Philadelphia, 2005, Saunders.)

Surgical Note: Inadvertent damage may occur during open reduction of the zygomatic arch or with the use of a Byrd screw or zygomatic hook during a closed approach.

Buccal Branch

Running almost horizontally, the buccal nerve will often divide into a separate branch above and below the parotid duct as it runs anteriorly.

Surgical Note: Injury is possible in association with soft tissue trauma to the cheek region.

Mandibular Branch

The marginal mandibular branch (Fig. 10-37) originates after the facial nerve divides into the temporofacial and cervicofacial division and extends anteriorly and inferiorly within the substance of the parotid gland. There may be two or even three branches of this nerve.

These branches run anteriorly parallel to the inferior border of the mandible. In some cases, the course of this nerve is above the inferior border but, in as many as 19% of cases, it is found below this border.[48] In essentially all cases, the nerve is located above the inferior border of the mandible beyond the facial artery.

Surgical Note: The marginal mandibular branch is an important structure encountered at the inferior border of the mandible just beneath the platysma muscle fibers during an open approach to the mandibular angle and body area. For this reason, an initial incision made approximately 1 to 1.5 cm below the inferior border should prevent direct exposure or trauma. The terminal innervation of this nerve is to the ipsilateral muscles of the lower lip and to the mentalis muscle.

Cervical Branch

The cervical branch exits the parotid gland above its inferior pole and runs downward underneath the platysma muscle, which it innervates.

HYPOGLOSSAL NERVE

After exiting the brain, the hypoglossal nerve passes through the hypoglossal canal and winds around the vagus nerve inferiorly; it is bound to this nerve with some connective tissue. The hypoglossal nerve then passes inferiorly across the medial aspect of the stylohyoid and digastric muscles. It curves slightly upward across the internal and external carotid arteries and the occipital and lingual arteries on their lateral side (Fig. 10-38). Continuing forward along with the sublingual vein, the nerve follows the lateral surface of the hyoglossus muscle. At the posterior extent of the mylohyoid muscle, the nerve runs along the superomedial surface to split into several fibers within the substance of the tongue in a fanlike fashion. The hypoglossal nerve is responsible for motor innervation to all the muscles of the tongue. The nerve, however, contributes fibers to the ansa cervicalis, which descends along the internal carotid artery and joins branches of the second and third cervical nerves to contribute to the hypoglossal ansa. From the loop, branches are sent to the omohyoid, sternothyroid, and sternohyoid muscles.

REGIONAL ANATOMY

ORBITAL ANATOMY

The configuration of the bony structure of the orbit (Figs. 10-39 to 10-42; see also Figs. 10-32 to 10-34)* is that of a pyramid with its base facing anteriorly, with each orbit having an intrabony volume of approximately 35 mL. Each bony orbit is composed of seven bones (see Fig. 10-39), as follows:

- Frontal bone
- Zygoma
- Maxilla
- Lacrimal bone
- Ethmoid bone
- Sphenoid bone
- Palatine bone

The medial walls (lamina papyracea of the ethmoid, lacrimal, and palatine bones) are almost parallel to the sagittal plane. The medial wall is the thinnest wall of the orbit, but derives some increased strength from the trabeculation of the ethmoidal air cells. The lateral walls (zygoma, sphenoid, and frontal bones) diverge from the apex at approximately 45 degrees. The lateral orbital rim is formed by the zygoma and is posterior to the medial, superior, and lateral orbital rims. The floor of the orbit (maxilla) is the roof of the maxillary sinus and is relatively thin and anatomically weakened by the passage of the

*References 1, 4, 11, 15, 17, and 49.

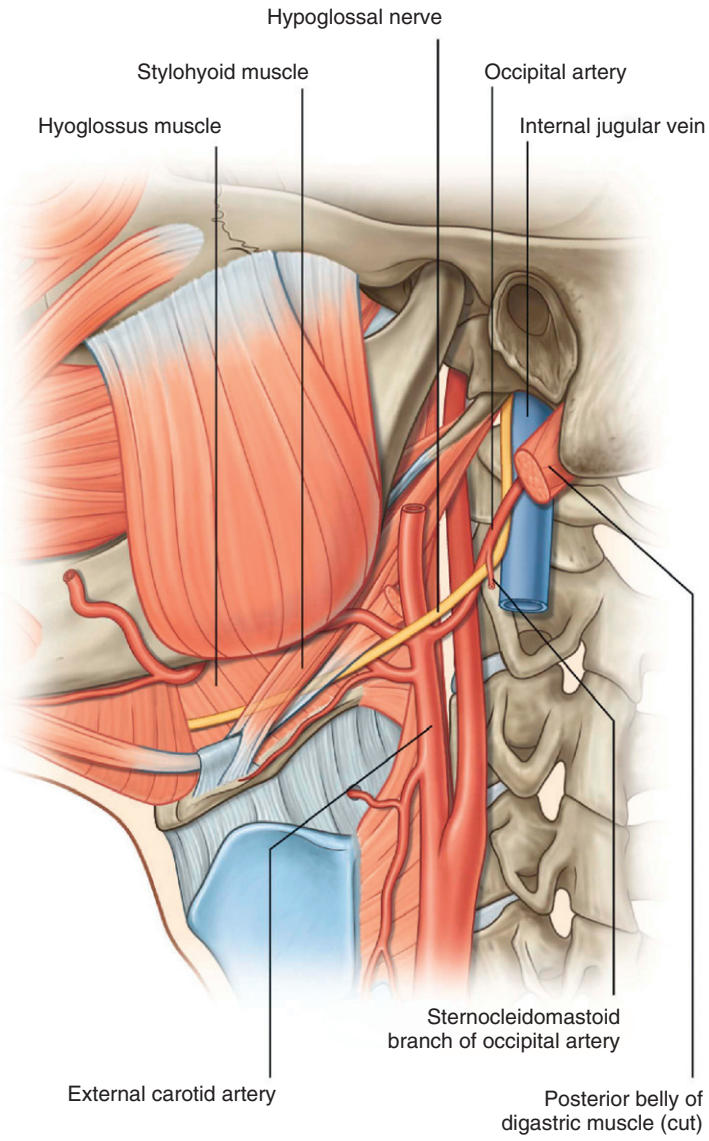


FIGURE 10-38 Hypoglossal nerve. (From Drake R, Vogl AW, Mitchell A: *Gray's anatomy for students*, ed 2, Philadelphia, 2009, Churchill Livingstone.)

infraorbital nerve. The roof of the orbit is formed mainly by the frontal bone and partly by the sphenoid bone. The trochlea, which transmits the tendon of the superior oblique muscle, is a special periosteal attachment in the area of the junction of the medial wall and roof of the orbit—approximately 4 mm posterior to the orbital rim—and its integrity must be maintained during medial orbital exploration. If it is disrupted, it must be securely reattached. The roof of the orbit continues forward to form the superior orbital rim, which has a notch or canal for the supraorbital and supratrochlear neurovascular bundles. As with the trochlea, the frontal orbital rim and its integrity must be maintained during medial orbital exploration; if it is disrupted, it must be reattached securely. The frontal bone articulates laterally with the zygoma at the frontozygomatic suture. The orbital rims are extremely strong and provide protection for the globe.

Consideration must be given to the position of the infraorbital nerve and terminal branches of the trigeminal nerve (CN V2) during any dissection of the inferior rim or orbital floor. The infraorbital nerve supplies sensation to the skin of the face in this region and to the conjunctiva of the lower lid. The anterior and superior alveolar nerve descends as a terminal branch in or along the maxilla. This nerve supplies the sensation to the anterior maxillary teeth and gingivae. The associated small arteries that run with these nerves generally are not of surgical concern and are rarely identified. Although their sacrifice is not problematic, every attempt should be made to maintain or decompress the sensory nerves in this area.

The orbital soft tissue is separated from the orbicularis oculi muscle and extraorbital soft tissue by the orbital septum. This septum constitutes a diaphragm at the entrance to the orbit that functionally separates the tissue spaces of the lid from those of the orbit. It essentially is a continuation of the periosteum of the orbit (periorbita) and the periosteum of the outer surface of the adjacent facial bones. From here it extends into the upper and lower lids. The septum fuses with the connective tissue anterior to the superior and inferior tarsus. Medially, it passes posterior to the posterior lacrimal

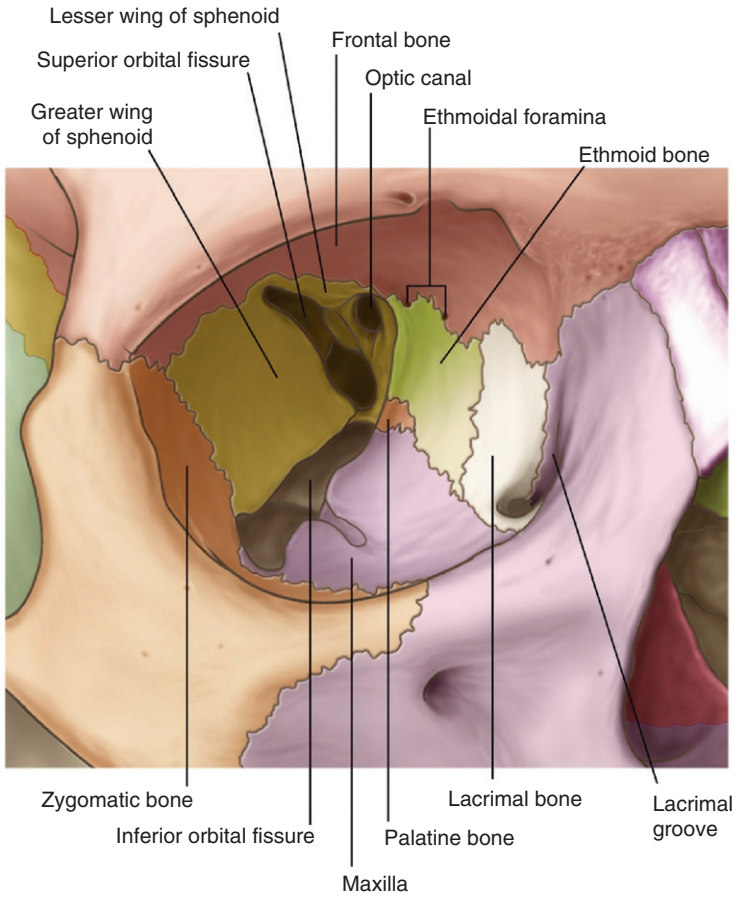


FIGURE 10-39 Orbital osteology. (From Drake R, Vogl AW, Mitchell A: *Gray's anatomy for students*, ed 2, Philadelphia, 2009, Churchill Livingstone.)

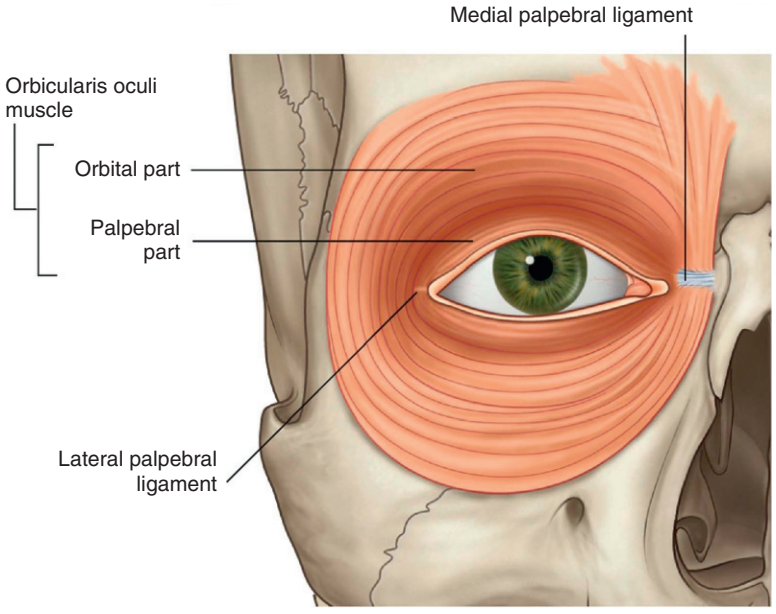


FIGURE 10-40 Orbicularis oculi muscle. (From Drake R, Vogl AW, Mitchell A: *Gray's anatomy for students*, ed 2, Philadelphia, 2009, Churchill Livingstone.)

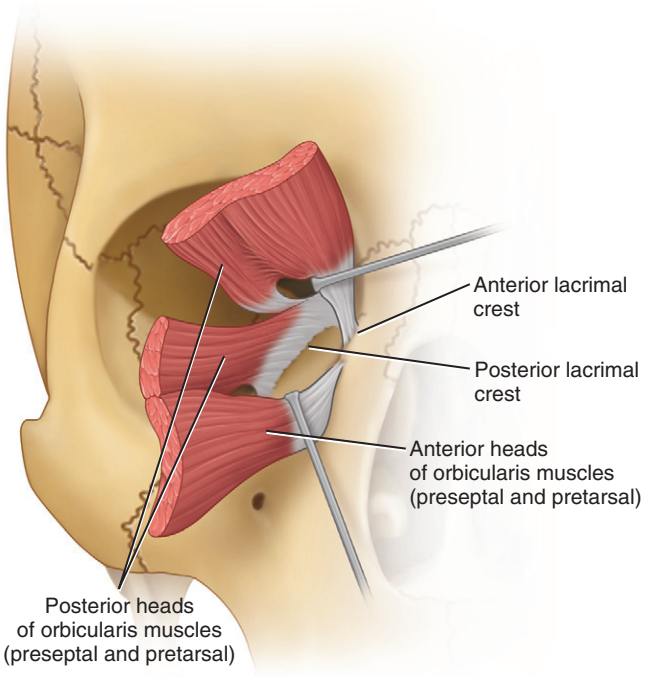


FIGURE 10-41 Medial palpebral ligament.

crest, thus eliminating the lacrimal sac from the orbit proper.

Orbital Muscles*

Orbicularis Oculi Muscle. The orbicularis oculi muscle (see Fig. 10-40) is composed of a palpebral division, which further divides into preseptal and pretarsal subdivisions, and an orbital division. The palpebral division arises from the medial palpebral ligament and inserts into the lateral palpebral raphe. A portion closest to the medial margin of the lid arises from the posterior lacrimal crest and is known as the pars lacrimalis or muscle of Horner. The larger portion of the medial palpebral ligament arises from the anterior lacrimal crest. The lateral palpebral raphe is attached to the osseous protuberance on the inner aspect of the lateral orbital rim of the zygoma (Whitnall's orbital tubercle) and passes medially to the lateral commissure of the eyelid, where it divides into two slips. The muscle is innervated by temporal and zygomatic branches of the facial nerve (CN VII) at the lateral and inferior raphe. Paralysis prevents tight closure of the eye and can predispose the patient to ectropion and epiphora.

*References 1, 4, 6, 11, and 49.

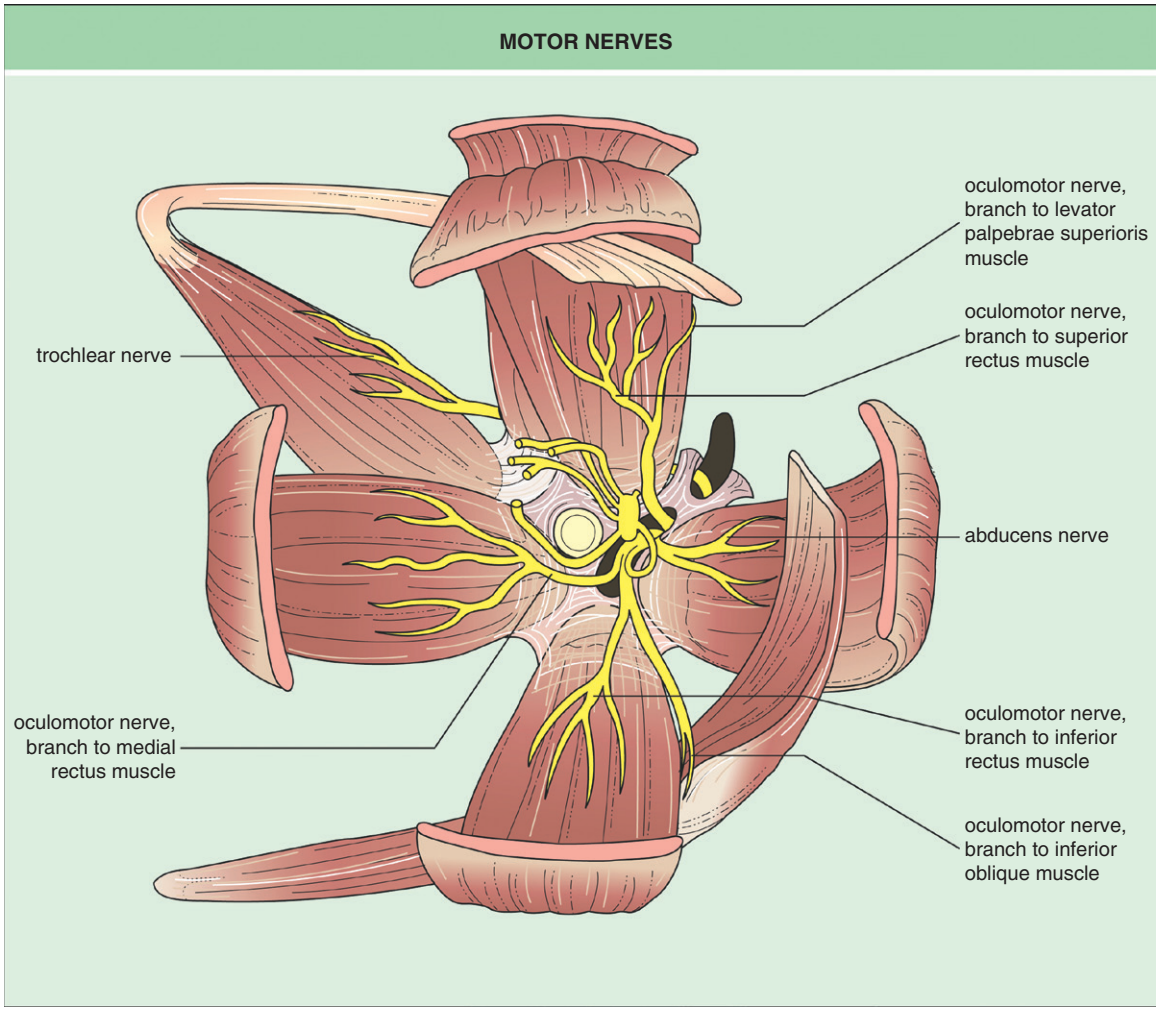


FIGURE 10-42 Frontal view of orbital musculature and its nerve relationship. (Adapted from Dutton JJ. *Atlas of clinical and surgical orbital anatomy*. Philadelphia, 1994, WB Saunders.)

Surgical Note: The medial palpebral ligament gives anatomic support to the lacrimal sac and is involved in emptying of the sac. Its position dictates the shape of this area (see Fig. 10-41). Every attempt to correct a traumatic displacement should be made and, in a similar fashion, care should be taken to avoid displacing this ligament in the reduction of fractures and treatment of soft tissue trauma.

Levator Palpebrae Superioris. The levator palpebrae superioris is a muscle of the upper lid and is a direct antagonist to the orbicularis oculi muscle; it raises the upper lid and exposes the globe. It arises deep in the orbit from the lesser wing of the sphenoid bone. It passes above the superior rectus muscle as a thin flat muscle and becomes broad as it ends anteriorly in an aponeurosis, which splits into three lamellae. Innervation is by the oculomotor nerve (CN III) as it enters the orbit through the superior orbital fissure (see Fig. 10-34).

Rectus Muscles. The rectus muscles arise from a fibrous ring that surrounds the superior, inferior, and medial aspects of the optic foramen (see Fig. 10-34). The ring continues as a tendinous band over the inferior and medial aspects of the superior orbital fissure. This fibrous ring in its lower division (tendon of Zinn) gives rise to the inferior rectus muscle, part of the medial rectus muscle, and inferior head of the lateral rectus muscle. The upper division of this fibrous band (superior ligament of Lockwood) gives rise to the superior rectus muscle, remainder of the medial rectus muscle, and superior head of the lateral rectus muscle. Each rectus muscle then passes anteriorly in the orbit in the position implied by its name and inserts as a tendinous area of the sclera, anterior to the equator of the globe and approximately 6 mm behind the margin of the cornea.

All except the lateral rectus muscle are innervated on their deep surface by the oculomotor nerve (see Fig. 10-32). The lateral rectus muscle is innervated by the abducens nerve (CN VI). These muscles form a cone that gives some protection to the optic nerve (CN II), which passes anteriorly within their confines (see Fig. 10-42). Between the two heads of the lateral rectus muscle, the two divisions of the oculomotor nerve, nasociliary nerve, abducens nerve, and ophthalmic vein enter the muscular cone. The optic canal, which lies within the confines of the origin of these muscles, transmits the optic nerve and ophthalmic artery (see Fig. 10-34).

Superior Oblique Muscle. The superior oblique muscle takes its origin immediately above the optic foramen, superiorly and medially to the superior rectus muscle. It passes anteriorly and ends in a tendon that passes through a fibrocartilaginous ring attached at the trochlear fovea of the frontal bone. The tendon bends posteriorly at this ring and passes beneath the superior rectus muscle to insert into the sclera posterior to the equator of the globe on the laterosuperior aspect (see Fig. 10-32). This muscle is innervated by the trochlear nerve (CN IV), which enters the orbit through the superior orbital fissure and passes above the other orbital nerves and enters the muscle from the inferior aspect.

Inferior Oblique Muscle. The inferior oblique muscle is a thin muscle that arises from the orbital surface of the maxilla, laterally to the lacrimal groove. It passes laterally on the bony side of the inferior rectus muscle and then inserts into the sclera posterior to the equator of the globe and between the superior and lateral rectus muscles. Innervation is supplied by the oculomotor nerve.

Medial Palpebral Ligament

Medial Canthal Ligament. The medial canthal ligament* attaches the tarsal plates to the medial wall of the orbit and aids in the attachment of the orbicularis oculi musculature to the medial orbit. It gives structure and configuration to the palpebral configuration (see Fig. 10-41). Traumatic disruption leads to pooling of lacrimal flow in the medial palpebral area.

The medial canthal ligament attaches to the anterior lacrimal crest of the maxilla and posterior lacrimal crest of the lacrimal bone. Between these slips of muscle runs the lacrimal sac. The orbicularis oculi muscle, which arguably contributes a portion of its musculature to the prominent posterior slip of the ligament, is referred to as the pars lacrimalis or Horner's muscle. The position of this muscle aids in the efficient collection of tears by creating a positive and negative pressure change on opening and closing the eyelids. This pressure change ensures the flow of tears through the lacrimal sac.

Proper replacement of the traumatically displaced medial canthal ligament is essential to restore proper function to this area. Options for the management of this ligament are discussed in detail in Chapter 17.

Orbital Blood Supply

The ophthalmic artery is the branch of the internal carotid artery that supplies the orbit (see Fig. 10-32).[6,7,11,17] This vessel enters the orbit through the optic canal with the optic nerve. It initially passes inferiorly and laterally to the optic nerve. The first branch is the central retinal artery. From the inferior lateral position, it passes over the optic nerve toward the medial orbital wall. As the ophthalmic artery passes around the optic nerve, it gives off branches to the lacrimal gland and long posterior ciliary branches to the lateral aspect of the globe. As the lacrimal artery passes anteriorly along the superior aspect of the lateral rectus muscle, it supplies muscular branches and terminates in the lateral palpebral artery and zygomatic branches. The lateral palpebral branches supply the lateral eyelids and the zygomatic branch passes through the zygomaticotemporal foramen to reach the temporal fossa.

As the artery crosses over the optic nerve, it gives off branches of short posterior ciliary arteries to the globe and large supraorbital branch. Numerous small muscular branches arise in this area as well. This branch passes anteriorly and superiorly along the superior rectus and levator palpebrae superioris muscles to the supraorbital foramen or notch. It supplies the muscles associated with its course.

The posterior and anterior ethmoidal arteries are the next branches of the ophthalmic artery as it continues on the medial and superior aspects of the orbit. These arteries give blood to the ethmoidal air cells and frontal sinus and finally terminate as they enter the cranium as

*References 4, 6, 7, 11, 13, 15, and 49.

small meningeal branches to the dura mater. The medial palpebral arteries arise slightly anteriorly and inferiorly to the pulley of the superior oblique muscle. They leave the orbit and supply the eyelids from the medial aspect.

The terminal branches are the supratrochlear and dorsal arteries. The former leaves the orbit at the medial angle and supplies the skin of the forehead in this area. The latter exits the orbit above the medial palpebral ligament and supplies the dorsum and root of the nose and skin in this area.

Orbital Nerves

The complex structure and function of the orbit necessitate an intricate neural system (see Figs. 13-32 to 13-34).[7,8,11,49] From a surgical anatomic view, the sympathetic and parasympathetic nerve functions are interwoven with the larger, more readily identified cranial nerves. Therefore, only the cranial nerves and their positions and anatomic considerations will be discussed here.

Sensory Nerves. CN II, the optic nerve, enters the orbit through the optic canal or foramen in the sphenoid bone and takes a direct route to the posterior aspect of the globe. As this nerve enters the orbit, it is immediately enclosed in the muscular cone of the extraocular muscles and is afforded some protection by these structures. There is also some protection given by the laxity of the nerve. It is approximately 5 mm longer than the distance from the orbital canal to the posterior aspect of the globe (2 to 2.5 cm).

The general sensory nerves of the orbit are all branches of the ophthalmic nerve, the first division of the trigeminal nerve (CN V). This nerve branches just before entering the orbit via the superior orbital fissure. Two branches enter the orbit superiorly and laterally to the ophthalmic foramen, whereas the third enters through the ophthalmic foramen.

The lacrimal nerve is the smallest branch and courses along the upper border of the lateral rectus muscle to the lacrimal gland and then down to the conjunctiva and skin of the upper eyelid. The frontal nerve is largest and runs above the levator palpebrae muscle. It divides into a supraorbital branch, which exits through the supraorbital notch and supplies the upper eyelid, forehead, and scalp in this area, and supratrochlear nerve, which passes over the trochlea of the superior oblique muscle to the conjunctiva of the upper eyelid and the forehead.

The third branch of the ophthalmic nerve is the nasociliary branch, which passes through the ophthalmic foramen, over the optic nerve, and below the superior rectus muscle to the medial wall of the orbit, where it distributes its many branches. These include communications to the ciliary ganglion, a long ciliary branch to the globe, an infratrochlear branch, which supplies the medial angle of the conjunctiva, lacrimal sac, and skin of the nose and eyelid in this area, and finally the anterior and posterior ethmoidal branches, which pass through the associated foramen to supply the ethmoidal, frontal, and sphenoidal sinuses and the nasal cavity.

Surgical Note: The optic nerve is protected in the orbit by the extraocular muscular cone and the fact that it is approximately 5 mm longer than the distance from the back of the globe to the optic canal (≈20 to 25 mm). Surgically, this allows a fair degree of forward displacement of the globe in retrobulbar surgery and manipulation; however, the nerve is tightly bound at the optic foramen, where the meninges blend with the periorbita. Careful attention to the position of the retractors when exploring the medial wall prevents trauma to the nerve, which exits the canal approximately 2.5 to 3 cm deep into the anterior lacrimal crest.

Motor Nerves. The remaining nerves of the orbit all enter the orbit through the superior orbital fissure (see Fig. 10-34). The abducens nerve (CN VI) and oculomotor nerve (CN III) pass through the oculomotor foramen, which is created by the tendinous ring of the rectus muscles crossing the superior orbital fissure, and thus are contained within the muscular cone. The trochlear nerve (CN IV) passes above the ring and stays outside the muscular cone throughout its course. The trochlear nerve then rises to the roof of the orbit and passes medially to the superior oblique muscle, which it supplies.

The abducens nerve supplies the lateral rectus muscle. It enters the orbit between the heads of the lateral rectus muscle and below the inferior division of the oculomotor nerve and passes along the inner surface of the lateral rectus muscle. As noted, the trochlear nerve supplies the superior oblique muscle, whereas the oculomotor nerve supplies all the other extraocular muscles.

As the oculomotor nerve enters the superior orbital fissure, it separates into two divisions—a small superior and larger inferior division. They both pass between the heads of the lateral rectus muscle. The superior division passes above the optic nerve and innervates the superior rectus and the levator palpebrae muscles, whereas the inferior division passes below the optic nerve and innervates the inferior and medial rectus muscles and the inferior oblique muscle.

Surgical Note: Direct or indirect trauma to the area of the confluence of anatomy at the posterior aspect of the orbit (accidental or surgical in nature) may cause the rarely seen complications of superior orbital fissure syndrome and orbital apex syndrome. The symptoms depend on the structures involved and localize the lesion anatomically. The advisability of surgical intervention in the presence of pretreatment complications such as these is discussed in Chapter 18.

NASAL ANATOMY

EXTERNAL NASAL ANATOMY

The prominence of the nose makes it a frequent target in interpersonal conflict and an often traumatized structure in other forms of facial injury. Injuries result in cosmetic and, if untreated, functional problems. Thus, an in-depth understanding of the anatomy of this area is important.

The substructure of the external nose* is composed of the cartilaginous lower half and nasal bones superiorly (Fig. 10-43). The inferior cartilaginous structure derives some of its support from the alveolar process of the

*References 1, 2, 4, 7, 16, and 17.

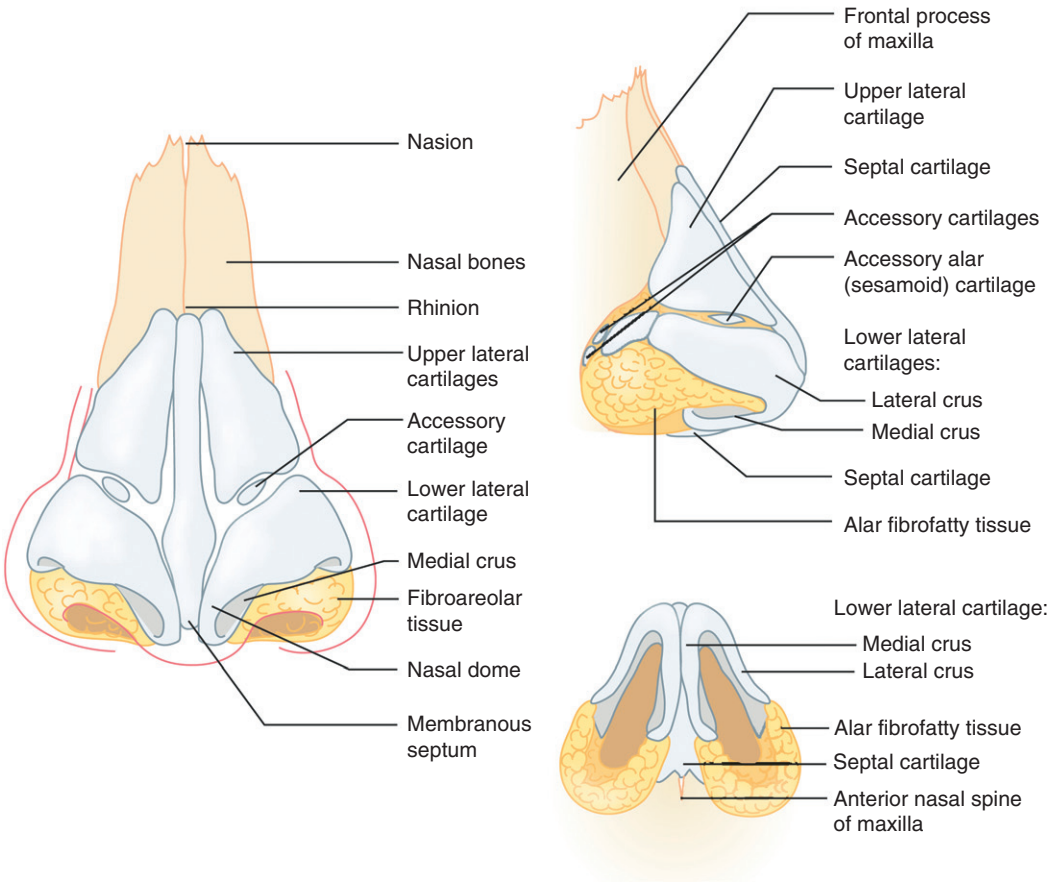


FIGURE 10-43 *Left,* Substructure of external nose. *Right,* Inferior view of nasal septum and alar cartilages. (From Nouri K: *Complications in dermatologic surgery*, Philadelphia, 2008, Mosby.)

maxilla in the piriform apertures. The area of union of the maxillas in the midline forms an anterior projection, the anterior nasal spine, which lends support to the nasal tip. The bony opening of the nose is composed of two paired bones, the maxilla inferiorly and the nasal bones superiorly.

The nasal bones are thinner toward the tip of the nose, gradually thickening as one proceeds toward the upper half. Similarly, the lower segments of the nasal bones are convex in shape, whereas the upper portions tend toward concavity. The cartilaginous portion of the external nose consists of the septal cartilage and lateral and alar cartilages. Termed the *keystone area,* this cartilaginous section extends 4 to 7 mm beneath the nasal bones. The paired upper lateral cartilages are attached to the septal cartilage in the midline and to the undersurface of the nasal bones superiorly. A portion of the upper lateral cartilage attaches to the septum via connective tissues. They are often continuous with each other at the superior margin and, along with the septal cartilage, are considered the nasoseptal cartilage.

The lateral cartilages are triangular. The base of the triangle is superior and articulates with the nasal bones. The lateral border thins to connective tissue, which separates it from the superior aspect of the alar cartilage. The medial border is thicker and blends into the septal cartilage superiorly; inferiorly, it has a free edge just lateral to the nasal midline.

The alar cartilages are paired curved cartilages that support the nasal openings. They have a medial and lateral crus. The crura are formed by a bending of the cartilage on itself, with the medial crus of one side loosely connected with the opposite medial crus (see Fig. 10-43). The part that forms the lateral crus curves gently to form the alae of the nose. Laterally and inferiorly, fibrofatty tissue makes up the remainder of the alar base.

The nasal septal cartilage articulates with the perpendicular plate of the ethmoid, vomer, maxilla, and nasal bones and the upper and lower nasal cartilages. This midline structure forms the support for much of the external nasal structure.

The arterial supply to the external nose is largely via the external carotid system by the branches of the facial and maxillary arteries. It is supplemented by a small portion from the internal carotid system via the ophthalmic artery as its terminal branches perforate the orbital septum and pass downward on the lateral aspect of the nose (see Fig. 10-27). The blood supply to the nose is found primarily in the soft tissue layers, necessitating that care be taken during surgical repairs. Dissection should be directed as close to the cartilage as possible.

The external nasal neural innervation is complex, being derived from the supratrochlear and infratrochlear branches of the ophthalmic nerve superiorly, infraorbital branch of the maxillary nerve laterally and inferiorly, and external nasal branch of the nasociliary

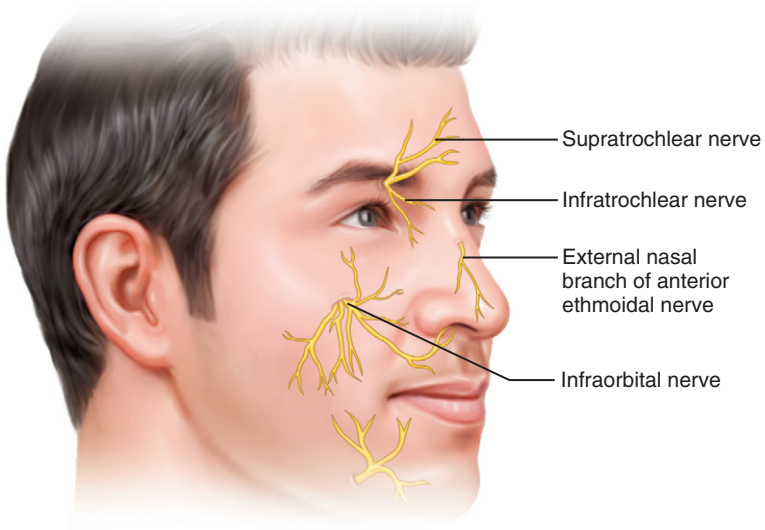


FIGURE 10-44 Sensory nerves to the external nose.

nerve from the ophthalmic nerve to the nasal tip and skin over the dorsum inferiorly (Fig. 10-44).

NASAL CAVITY ANATOMY

Internally, the nasal septum (see Fig. 10-8) divides the nasal cavity.[7,16,17] The nasal cavity is roughly teardrop-shaped in the frontal section, with a narrow area above. The walls of the internal nose are formed medially by the nasal septum, laterally by the maxilla, ethmoid bone, and nasal cartilages, inferiorly by the maxilla and palatine bones, and superiorly by the cribriform plate of the ethmoid bone.

The nasal cavity is lined by mucous membranes that are tightly attached to the underlying periosteum or perichondrium, except in the vestibule, where facial skin rolls into the nasal aperture. These mucous membranes are highly vascular.

The nasal septum is the common medial wall of the two nasal cavities (see Fig. 10-8). It is formed posterosuperiorly by the perpendicular plate of the ethmoid bone, by the vomer posteroinferiorly, and by the septal cartilage and medial crus of the alar cartilages anteroinferiorly. Below, the nasal crests of the maxilla and palatine bones complete the septum. The septum rests in the groove formed by these bones and, if displaced by trauma, requires replacement in the groove to prevent functional and aesthetic problems.

The lateral wall of the nose is formed inferiorly by the lateral wall of the maxilla and inferior nasal concha, which is an independent bone. Below the concha is the inferior meatus. This meatus contains the opening of the nasolacrimal duct. Superiorly, the lateral wall is formed by the segments of the ethmoid bone, which form the middle and superior conchae. Beneath the middle concha is the middle meatus, which receives the opening of the frontal sinus, anterior ethmoidal air cells, and maxillary sinus. Posterior to the middle concha on the lateral wall is the sphenopalatine foramen. Superior and posterior to the middle concha is the superior concha. It is much shorter and smaller than the others. Below it, in the superior meatus, is the opening to the posterior ethmoidal air cells. The supreme nasal concha, with its associated meatus, is not generally identifiable, but in this area, above and behind the superior concha, is the opening of the sphenoidal sinus and sometimes the posterior ethmoidal air cells.

The blood supply to the nasal septum is via branches of the sphenopalatine artery, anterior and posterior ethmoidal arteries, and facial artery. Thus, the blood supply is from the internal and external carotid systems.

The innervation of the internal nose is general sensory and special sensory. The special sensory innervation is via the olfactory nerve (CN II) and passes into the skull through the foramina of the cribriform plate of the ethmoid bone. The general sensory innervation of the internal nose is via the nasociliary branch of the ophthalmic nerve, filaments from the anterior alveolar branch of the maxillary nerve, nerve of the pterygoid canal, nasopalatine nerve, anterior palatine nerve, and nasal branches of the pterygopalatine nerve.

The nasociliary branch of the ophthalmic division of the trigeminal nerve gives filaments to the anterior part of the septum and lateral nasal wall (Figs. 10-45 and 10-46). The anterior alveolar nerve supplies the inferior meatus and concha, whereas the nerve of the pterygoid canal supplies the superior and posterior septa and superior concha. The superior branches from the pterygopalatine nerve have a distribution similar to that of the nerve of the pterygoid canal. The nasopalatine nerve supplies the middle of the septum, whereas the anterior palatine nerve supplies the lower nasal branches to the middle and inferior conchae.[1,4,7]

PAROTID REGION

PAROTID GLAND

The parotid gland is the largest of the salivary glands and occupies the parotid facial space (Fig. 10-47). This space generally extends from the ramus of the mandible anteriorly, from the tympanic bone and the mastoid process posteriorly, and from the zygomatic arch superiorly. The

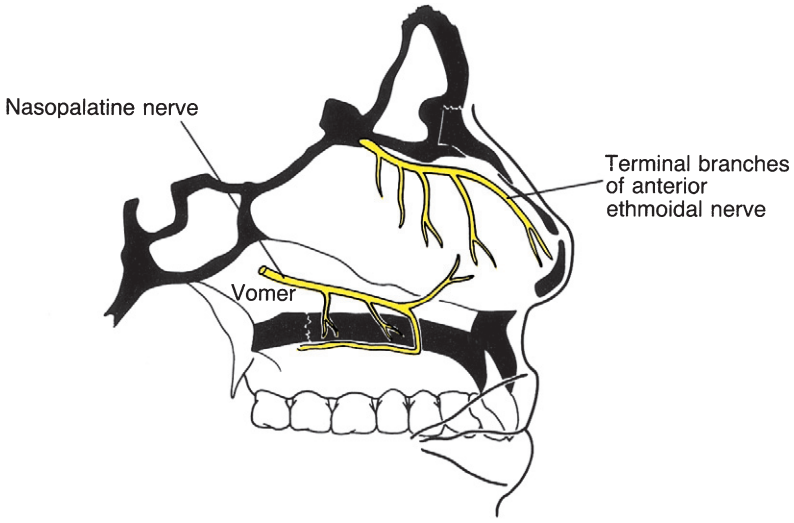


FIGURE 10-45 Innervation to the nasal septum.

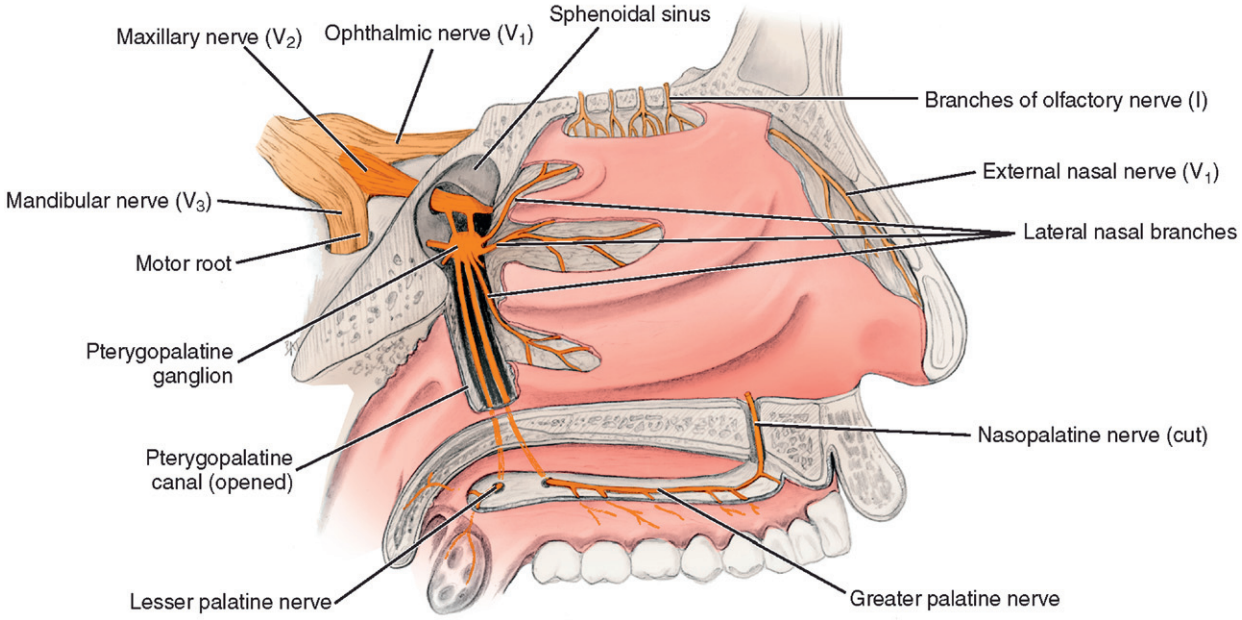


FIGURE 10-46 Lateral nasal wall innervation. (From Fehrenbach M, Herring S: *Illustrated anatomy of the head and neck*, ed 4, St. Louis, 2012, Saunders.)

inferior extent reaches to the angle of the mandible and a cervical portion reaches below, along the sternocleidomastoid muscle. There is a connective tissue investment that sends septa inward among the lobules of the gland.

The parotid gland is composed of a large superficial portion and is shaped like an inverted triangle lying on the ramus of the mandible. This portion may have an accessory glandular mass extending beyond the limits of the masseter muscle anteriorly. The smaller deeper portion of the gland is connected by a broad isthmus around the posterior border of the mandible. This deep portion may extend as far as the pharyngeal wall.

The parotid duct, or Stensen's duct, usually leaves from the apical portion of the superficial lobe and then runs forward on the masseter muscle to its anterior margin. The duct then turns medially, piercing the buccinator muscle obliquely to reach the mucous membranes of the cheek. At approximately the second molar, the duct opens into the mouth. The course of the duct approximates a line drawn from the concha of the ear to the commissure of the lips. Its length is normally about 5 cm.

The most important structures entering and leaving the gland are the facial nerve and its branches, posterior facial vein, external carotid artery with its branches, superficial temporal artery, and internal maxillary artery. The auriculotemporal nerve traverses the upper portion of the parotid gland.

Incisions to deep soft tissue and hard tissue should be horizontal rather than vertical, when possible, to prevent potential damage to the parotid duct and terminal branches of the facial nerve. Soft tissue trauma to the duct could lead to extraoral fistula formation.

SUBMANDIBULAR GLAND

The submandibular gland (Fig. 10-48)[3,6,18] occupies a space on the medial aspect of the mandible in the

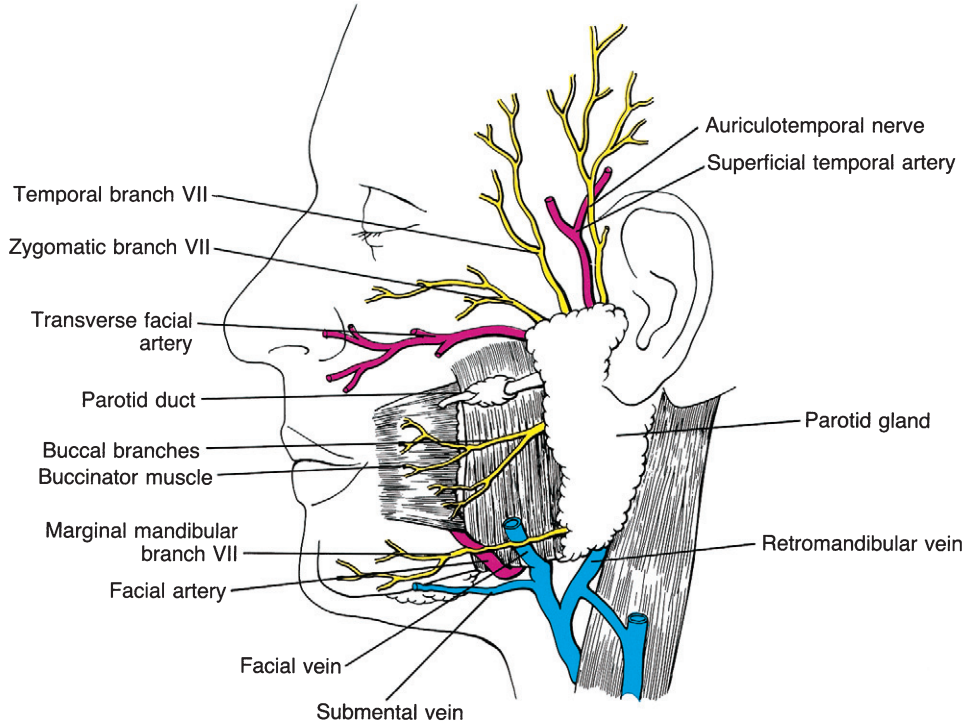


FIGURE 10-47 Parotid regional anatomy.

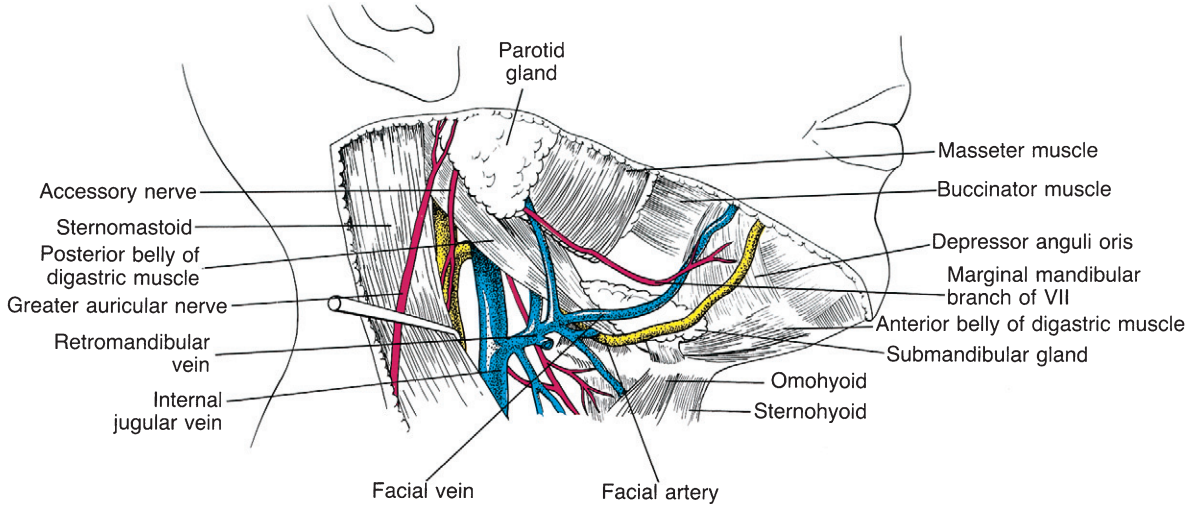


FIGURE 10-48 Submandibular regional anatomy.

submandibular fossa. Its lower pole extends inferiorly, covering the intermediate tendon of the digastric muscle. This gland is closely related medially to the stylohyoid, digastric, and styloglossus muscles and to the hyoglossus muscle and posterior border of the mylohyoid muscle anteriorly. The submandibular duct, or Wharton's duct, arises from the upper inner aspect of the gland and is often accompanied by the extension of the gland itself. The duct courses around the posterior border or free margin of the mylohyoid muscle and then runs anteriorly along the inner surface of the sublingual gland after crossing the lingual nerve superiorly. The duct ends at the base of the tongue near its tip.

Like the parotid gland, the submandibular gland is enclosed in a facial capsule, a derivative of the investing layer of the deep cervical fascia. Structures closely related to the gland include the facial artery, which may be embedded, but is at least closely related to its inner surface and upper border. During extraoral open reduction of the body or angle of the mandible, the fascia and

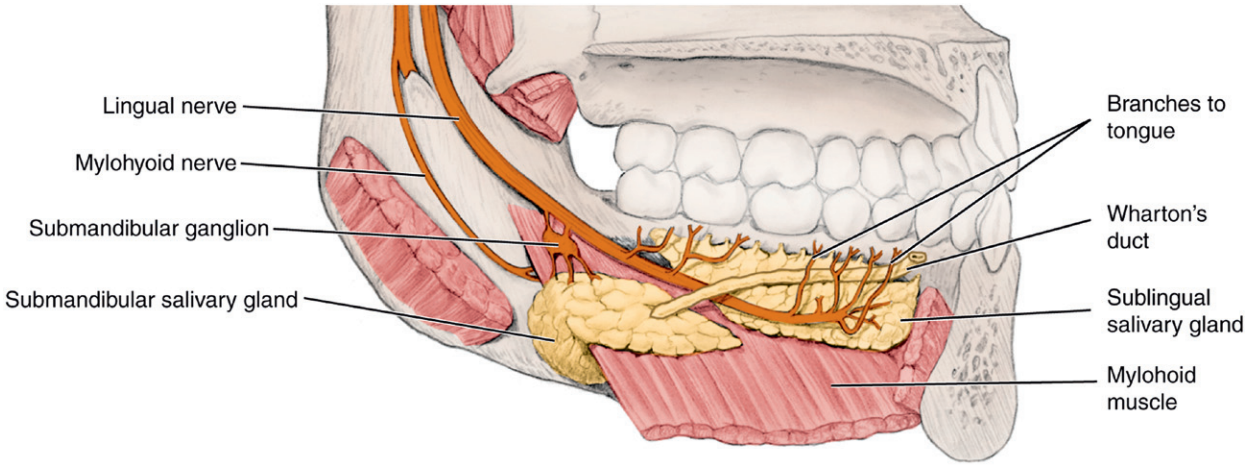


FIGURE 10-49 Floor of the mouth. (From Hupp J, Ellis E, Tucker M: *Contemporary oral and maxillofacial surgery*, ed 5, St. Louis, 2008, Mosby.)

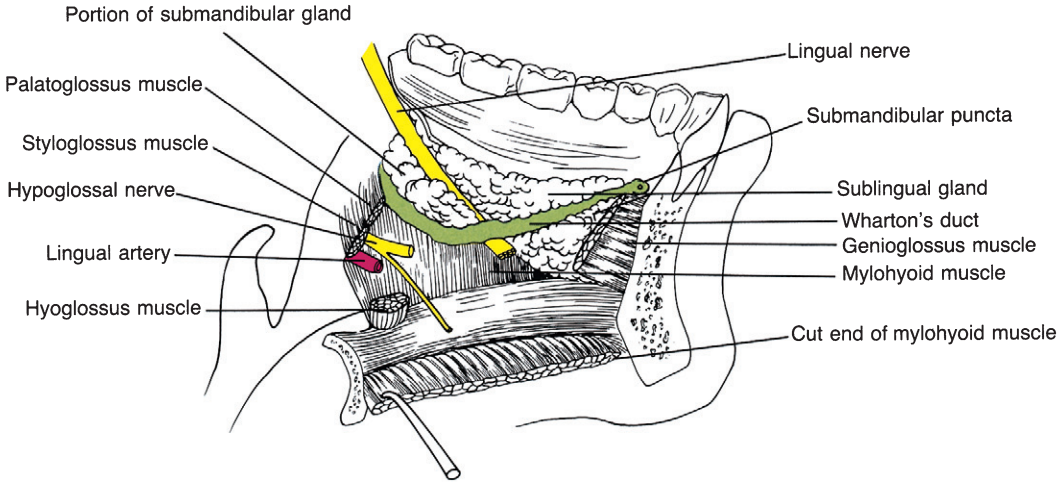


FIGURE 10-50 Sublingual anatomy.

gland may come into view. If the fragments are notably displaced, the fascia may be disrupted and may require repair.

FLOOR OF THE MOUTH

Key to the surgeon in repairing soft tissue trauma to the floor of the mouth (Figs. 10-49 and 10-50)[3,6,18] is an understanding of the anatomic position of various important structures, such as Wharton's duct and the lingual nerve, sublingual artery, sublingual gland, hypoglossal nerve, and submandibular gland itself. Each of these structures has been described, but here it is important to visualize the relationship of these structures to one another (see Fig. 10-49).

The submandibular, or Wharton's, duct exits the superior aspect of the gland, coursing above the posterior free edge of the mylohyoid muscle between the inner surface of the mandible and lateral surfaces of the hyoglossus and genioglossus muscles. The duct lies laterally to the hypoglossal nerve; it begins below the lingual nerve, which descends laterally to the duct. At this point, both the duct and lingual nerve pass around the lower border of the sublingual gland and then are positioned medially. The lingual nerve continues to run below the duct and curls medially on the genioglossus muscle. This arrangement forms almost a complete loop. As Wharton's duct passes on the medial side of the sublingual gland, it may receive the major sublingual duct, or Bartholin's duct. The last few millimeters of the anterior portion of the submandibular duct lie immediately below the oral mucosa, where the duct terminates by emptying from the sublingual papilla.

The sublingual artery passes along the side of the genioglossus muscle between the muscle and the sublingual gland, where it supplies the gland and muscles of the tongue. The deep lingual artery runs more medially below the mucous membranes on the inferior surface of the tongue. Bleeding from either of these vessels may be brisk in superficial lacerations of the mouth floor, or the vessels may be inadvertently transected during exploration, requiring ligation.

ACKNOWLEDGMENT

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CHAPTER 11

Early Assessment and Treatment Planning of the Maxillofacial Trauma Patient

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OUTLINE

Airway (With Cervical Spine Protection)
Breathing and Ventilation
Circulation
Scalp
Nose
Oral Cavity
Disability
Exposure
History of Present Illness
Review of Systems
Clinical Examination
Soft-tissue and Scalp
Forehead
Naso-orbital-ethmoid complex
Nose
Orbit
Malar Area
Ear
Maxilla, Mandible, and Dentoalveolar Structures
Neck
Cranial Nerves
Radiographic Evaluation
Postoperative Considerations

Advanced Trauma Life Support (ATLS) has been recognized as the gold standard for the initial management of multiple injured patients. Although the priority of maxillofacial injuries is usually subordinated to more critical, life-threatening injuries, the role of the maxillofacial surgeon in the primary and secondary surveys of patients with maxillofacial injuries should be emphasized.

The treatment of patients with maxillofacial injuries can be managed by a maxillofacial trauma team if the injuries are isolated to the maxillofacial area or by a designated hospital trauma team. Although initial diagnosis and emergency treatment (e.g., controlling hemorrhage) are intuitive even to the novice, the experienced surgeon will remember that significant injuries can be missed after initial management of the trauma patient. The incidence of missed injuries after trauma has been reported to range from 8% to 65%.[1] Missed injuries are especially likely when the mechanism of injury has caused substantial internal damage, as can be the case with decelerating injuries, for example. All members of the treatment team should assume the responsibility of constant patient reassessment.

Facial injuries are classified into four categories according to the urgency of necessary treatment:

- Immediate, resuscitative, or emergent treatment required: Facial injuries that are life-threatening (causing airway obstruction or severe hemorrhage) or sight-threatening (causing increases in intraocular pressure) and that require immediate interventions aimed at securing the airway, stopping the hemorrhage, or relieving the intraocular pressure via cantholysis.
- Treatment required within a few hours: Facial injuries that are extremely contaminated in a patient who is hemodynamically stable.
- Treatment required within 24 hours: Some facial fractures and lacerations.
- Treatment can be delayed for more than 24 hours if necessary: Most other facial fractures.

Details of the ATLS primary survey are discussed elsewhere in this text. The goal of this section is to highlight the role of the maxillofacial surgeon in the ABCDEs. This role is often crucial and includes life-preserving and sight-preserving procedures. A few examples are detailed here.

AIRWAY (WITH CERVICAL SPINE PROTECTION)

If a patient has an unfavorable bilateral mandibular fracture causing airway obstruction, the maxillofacial surgeon can assist by simply stabilizing the fracture with a bridle wire. By restoring spontaneous breathing, this maneuver may eliminate the need for endotracheal intubation.

BREATHING AND VENTILATION

If the patient's Glasgow Coma Scale score is lower than 15, performing a procedure to stabilize a bilateral mandibular fracture will permit the patient to breathe

spontaneously and will reduce the likelihood of airway obstruction if the patient's level of consciousness decreases. This procedure will allow the patient to breathe and ventilate without the aid of an advanced airway; in turn, the absence of endotracheal intubation allows neurologic assessment of the patient at regular intervals.

CIRCULATION

The face and neck are heavily vascularized regions of the body and this large blood supply facilitates a level of healing that is unmatched elsewhere in the body. On the other hand, however, this robust vascularity means that injuries to this region of the body can produce copious bleeding that requires immediate attention. A few key examples follow.

SCALP

The connective tissue layer of the scalp contains a rich, subcutaneous vascular supply. A laceration to the scalp can cause the loss of a large amount of blood and result in hypovolemic or hemorrhagic shock. Occasionally, the scalp is an occult source of hemorrhage. When a patient is in shock and the blood pressure is low, bleeding from a scalp wound is not obvious. Once resuscitation has been performed and the blood pressure has increased, however, bleeding will begin again and will become difficult to control. Thus it is necessary for scalp lacerations to be stabilized before any interhospital transfers or lengthy diagnostic procedures are performed.[2] The deleterious events that may follow a scalp hemorrhage should not be underestimated.

Scalp lacerations can be most rapidly stabilized with Raney clips, which should be available in emergency trauma bays and in operating rooms. Other potentially useful temporary stabilizing measures are staples and sutures.

NOSE

Epistaxis is a serious problem that may lead to airway obstruction, aspiration, shock, and exsanguination if not recognized and managed early. Many cases of unrecognized and untreated fatal epistaxis have been reported.[3-5] The most frequent cause of massive hemorrhage among patients with facial trauma is midface fractures, which typically cause epistaxis with bleeding through the oral cavity.[6] Control of a massive and a potentially lethal epistaxis consists of the following:

- Protecting the airway with endotracheal intubation or tracheotomy, if necessary
- Ruling out coagulopathy
- Packing the posterior and anterior nasal cavities

Posterior nasal packing is best performed with a 14-, 16-, or 18-Fr Foley catheter with a 10-mL balloon. The tip of the catheter is inserted through the nostril and is advanced until it is seen through the nasopharynx. This visualization is a crucial step, because the balloon of the catheter should not be inflated at the base of the skull. The catheter is then tugged forward to tamponade bleeding in the posterior nasal cavity. Bilateral posterior packs may be necessary. The catheter should be sutured or tied anteriorly so that posterior advancement and aspiration can be prevented.

The anterior nasal packing material is standard half-inch gauze moistened with bacitracin (or petrolatum gauze) placed in an overlapping fashion, beginning at the floor of the nose and extending superiorly.

Two ready-made commercial packing systems can control bleeding more rapidly than traditional packing, the Nasostat epistaxis balloon (Sparta, Pleasanton, Calif) and the Storz epistaxis catheter (Storz, St. Louis).

Packs should remain in place for no more than 24 hours; antibiotic coverage is recommended so that serious infections can be avoided. If packs are needed for more than 24 hours, they should be changed daily.

Although the posterior nasal packing procedure is safe and effective, it should be performed with caution if the patient has a fracture of the base of the skull.

Other control measures include the following:

- Reducing facial fractures
- Performing additional procedures for uncontrolled bleeding, if necessary

Interventional radiology and angiography can detect bleeding vessels and perform embolization.

If interventional radiology is unavailable, surgical exploration and ligation of the affected vessels may be necessary—transantral ligation of the maxillary artery or its terminal branches; ligation of the anterior or posterior ethmoid arteries through the medial orbital wall

Although historically the ligation of the external carotid artery has been described, the collateral circulation is very rich, and ligation of the external carotid artery will not always stop the hemorrhage.

ORAL CAVITY

Bleeding from the oral cavity can typically be managed temporarily with packing and pressure. Severe hemorrhage can indicate that the inferior alveolar vascular bundle has been severed. Applying a bridle wire or an arch bar (with or without maxillomandibular fixation) will stop the bleeding or substantially reduce it.

DISABILITY

Ocular examination is a routine part of the primary survey and includes pupil size, reactivity to light, and symmetry. In addition, the examiner should rule out a relative afferent pupillary defect (RAPD, or Marcus Gunn pupil) and palpate the globe (hard or soft).[7] These elements of the eye examination can be performed quickly and easily and yield a large amount of information.

EXPOSURE

In the maxillofacial region, any dentures or other removable appliances that impede a clear view of the site of potential bleeding should be removed.

HISTORY OF PRESENT ILLNESS

It can be difficult to obtain a history from the trauma patient; often, information must be gathered from the

prehospital personnel and the patient's family members. The following points should be addressed when the history is obtained (AMPLE is a familiar mnemonic)[7]:

Allergies
Medications
Pregnancy, previous illnesses
Last meal
Events or Environment related to and leading to the trauma

Intuitively, a knowledge of the patient's medical history, medications, and allergies helps determine whether modifications in management may be necessary. The last meal and pregnancy status of the female patient are important pieces of information, primarily as they relate to possible timing of surgery and to the evaluation and safety of the fetus.

Certain mechanisms of injury should produce a high level of suspicion for distinct types of trauma. For example, a history of a decelerating injury (restrained passenger in a motor vehicle accident [MVA]) should lead to a high suspicion of serious life-threatening consequences caused by shearing forces (e.g., lung or aortic injuries).[7] Knowledge of the mechanism of injury also assists the surgeon in identifying specific maxillofacial injuries. Blunt injuries to the midface, for example, should prompt the performance of a thorough orbital examination and appropriate radiologic imaging for a proper diagnosis.

REVIEW OF SYSTEMS

When the trauma patient is awake and oriented, a detailed review of systems (ROS) is invaluable because it directs the examiner's attention to the site and type of injury. Many physical examination textbooks provide examples of various methods for completing the ROS; every provider should develop and follow his or her own routine to avoid missing important information.

A full-body review of systems should be performed. Table 11-1 is a suggested sequence for performing a head and neck ROS. The ROS allows the inference of various levels of severity of injuries. If the patient has a history of clear discharge from the nose (rhinorrhea) or ears (otorrhea), a fracture of the base of the skull should be suspected until ruled out. If the patient has a history of loss of consciousness, amnesia, vomiting, headaches, or seizure activity after the traumatic incident, a traumatic brain injury should be suspected and a neurosurgical consultation should be requested. Loss of vision or change in visual acuity is another serious symptom that should prompt rapid intervention and consultation with ophthalmology. If the patient reports a change in the dentition or occlusion that is associated with pain and limited mouth opening, a mandibular fracture should be suspected.

CLINICAL EXAMINATION

SOFT TISSUE AND SCALP

Treatment indicated for soft tissue wounds depends on the type of injury. Abrasions and contusions are usually treated with wound cleansing or observation, whereas lacerations or avulsive injuries may need more advanced repair. Attention should be paid to all areas of the scalp; wounds to the back of the head in a patient with long hair can be easy to miss.

The degree of wound contamination and the wound contaminant should be considered during the examination of facial soft tissue injuries. Facial wounds can be classified as clean or contaminated, depending on the wounding agent. The most recent guidelines for tetanus vaccination and booster doses should be followed.[8]

Bleeding is often associated with facial soft tissue trauma. Definitive repair of these wounds should be deferred until the trauma examination has been completed and a comprehensive treatment plan has been created. Some lacerations could be used as access to facial fracture repair.

FOREHEAD

Injuries to the forehead can be easily recognized and may indicate frontal bone fractures. The soft tissue overlying the frontal bone should be examined and special attention should be paid to the closure of any lacerations involving the hairline or the eyebrows.

NASO-ORBITAL-ETHMOID COMPLEX

The naso-orbital-ethmoid (NOE) complex must be systematically inspected during the physical examination. A

TABLE 11-1 Suggested Sequence for Performing Head and Neck Review of Systems in a Maxillofacial Trauma Patient

Region	Symptom(s)
Head, CNS	Headaches, nausea, vomiting, loss of consciousness after trauma, weaknesses in limbs, numbness, dizziness
Eyes	Change in visual acuity, double vision, pain, pulsatile eye
Ears	Changes in hearing acuity, ringing in ears, history of discharge or bleeding from the ear after trauma, dizziness, pain
Nose	Discharge or bleeding from nose after trauma, pain
Oral cavity	Change in bite, pain, limited mouth opening, bleeding, teeth missing as result of trauma
Neck	Tenderness in cervical spine region, throat pain, voice change, pain on swallowing
Cranial nerves	Numbness or weakness of particular area of face

missed NOE fracture can lead to serious aesthetic consequences that cannot be easily corrected by revision surgical procedures. Because the NOE complex is rarely fractured in isolation, the examination of this area is complex.

Inspection

NOE fractures will be accompanied by ecchymosis and edema in the periorbital region and eyelids; subconjunctival hemorrhage may also be seen. If the NOE fracture occurs in combination with other facial fractures, edema and ecchymosis are generalized. NOE fractures produce a depression in the nasal bridge, causing the nose to appear short and retruded. Because telecanthus (increased intercanthal distance) is indicative of an NOE fracture, the intercanthal distance should be measured when an NOE fracture is suspected. This distance varies according to race and gender, but a rough estimate is that the intercanthal distance should typically equal 50% of the interpupillary distance.[9,10] When NOE fractures are present, the medial canthus is rounded and the palpebral fissures are shortened. The position of the globe may also be affected if NOE fractures occur in combination with orbital wall fractures; enophthalmos is a common finding when wall fractures increase the orbital volume. Because NOE fractures can also occur in combination with fractures at the base of the skull, it is important to look for any signs of cerebrospinal fluid (CSF) leakage.

Palpation

If a fracture is present, palpation of the NOE complex will demonstrate crepitus and mobility. Palpation should be performed with the index finger and thumb placed on either side of the NOE complex, specifically on the medial orbital wall adjacent to the area of the medial canthal tendon (MCT). A Kelly clamp can be placed intranasally; its tip is placed deep to the attachment of the MCT. This positioning places the MCT and the attached bone between the Kelly clamp and the examiner's finger. The examiner can then easily assess the mobility of the complex (Fig. 11-1*A*).

The bowstring test is performed by gently holding the NOE complex (in a manner similar to that described earlier) and pulling the lateral canthal tendon laterally. When a fracture is present, the examiner will be able to detect movement of the MCT and any detached bone fragments (see Fig. 11-1*B*).

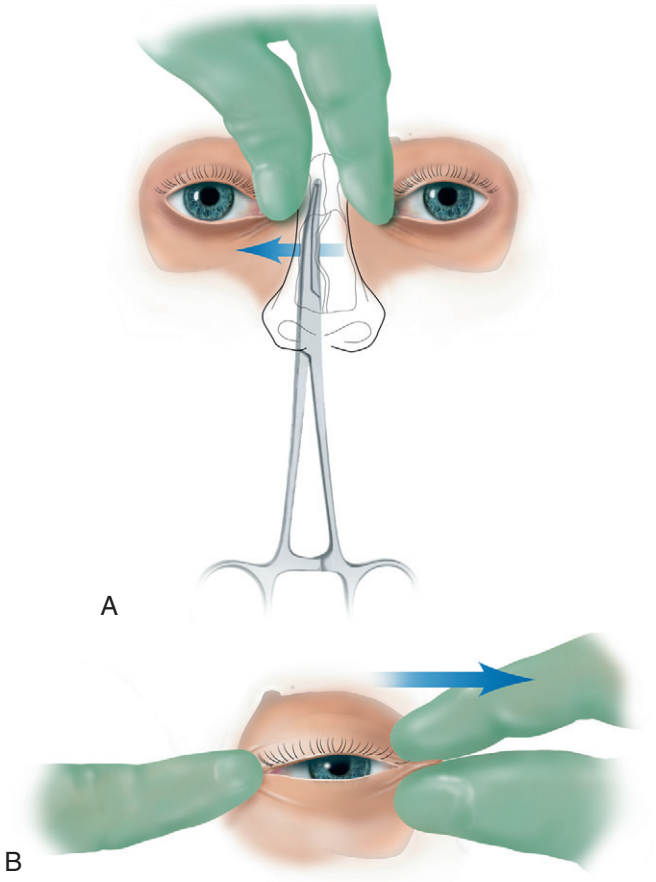


FIGURE 11-1 **A,** Bimanual palpation test. **B,** Bowstring test.

NOSE

The nasal complex should be examined with good illumination, a nasal speculum, and suction. The overlying soft tissue should be examined first; wounds should be thoroughly inspected, and any involvement of cartilage should be noted. The nasal bones should be evaluated for deformities; the examiner can perform this evaluation by looking at the nose from above (bird's-eye view) or by facing the patient. Nasal bones should then be palpated and checked for mobility. Intranasally, the examiner should look for signs of bleeding or CSF leakage. The nasal septum should be examined for perforation, deviation, or septal hematoma.

A nasal septal hematoma detaches the supplying mucoperichondrium from the cartilage, thereby reducing the blood supply to the cartilage. The late recognition of a septal hematoma may result in deforming aesthetic consequences, such as a saddle nose deformity or a retruded nasal bridge. Septal hematomas should be evacuated; needle aspiration with or without nasal packing is usually effective for this purpose, but a more formal incision and evacuation may be necessary if the hematoma has solidified.

ORBIT

Untoward sequelae of orbital fractures and periorbital injuries include serious injuries to the optic nerve, the globe, or both. These sequelae can result in partial or total loss of vision. Even orbital and periorbital injuries that appear to be trivial should be evaluated carefully so that any vision-threatening injuries can be ruled out. A thorough and systematic examination of the orbit should be performed when the patient has sustained an injury to the maxillofacial area. Details regarding the orbital anatomy, detailed examination of the orbit, and management of orbital injuries are presented elsewhere in this text. The goals of this section are to list the elements of

the clinical examination and to discuss the early findings that require immediate intervention.

The orbital examination of a patient with a maxillofacial injury is challenging. The timing of the examination is especially problematic when the patient is comatose or sedated. Early identification of the signs and symptoms of injuries to the globe and the optic nerve will lead to prompt emergency interventions that can preserve vision. For any injury to the globe, consultation with an ophthalmologist is prudent as soon as the patient's immediately life-threatening problems have been addressed.

Injuries to the orbit and the periorbital region include the following: fractures of one or more of the orbital walls (medial, floor, lateral, or roof), associated retrobulbar hemorrhage or hematoma, injuries to the cranial nerves in the apex of the orbital cavity, foreign bodies in the globe or the orbital cavity, injuries to the globe, and injuries affecting the soft tissues that surround and support the orbit (lacrimal apparatus, canthal tendons, eyelids) (Fig. 11-2, Table 11-2).

Even if the patient exhibits no initial signs of injury, a compartment syndrome could develop as the result of continued hemorrhage, inflammation, increased intraocular pressure, or other interventions or procedures that are performed. Therefore, patients with suspected ocular injury should be reassessed at regular intervals, especially if they are unconscious and unable to communicate the fact that their symptoms are worsening.

MALAR AREA

Depression of the malar eminence, crepitus, facial asymmetry, and periorbital ecchymosis are all signs of fractures of the zygomaticomaxillary complex (ZMC) (Fig. 11-3). Malar depression is best seen from a bird's-eye view (Fig. 11-4). This finding should be documented with high-quality pictures for many reasons, including intraoperative guidance. The malar area should be examined with care because edema in the area may mask the malar depression.

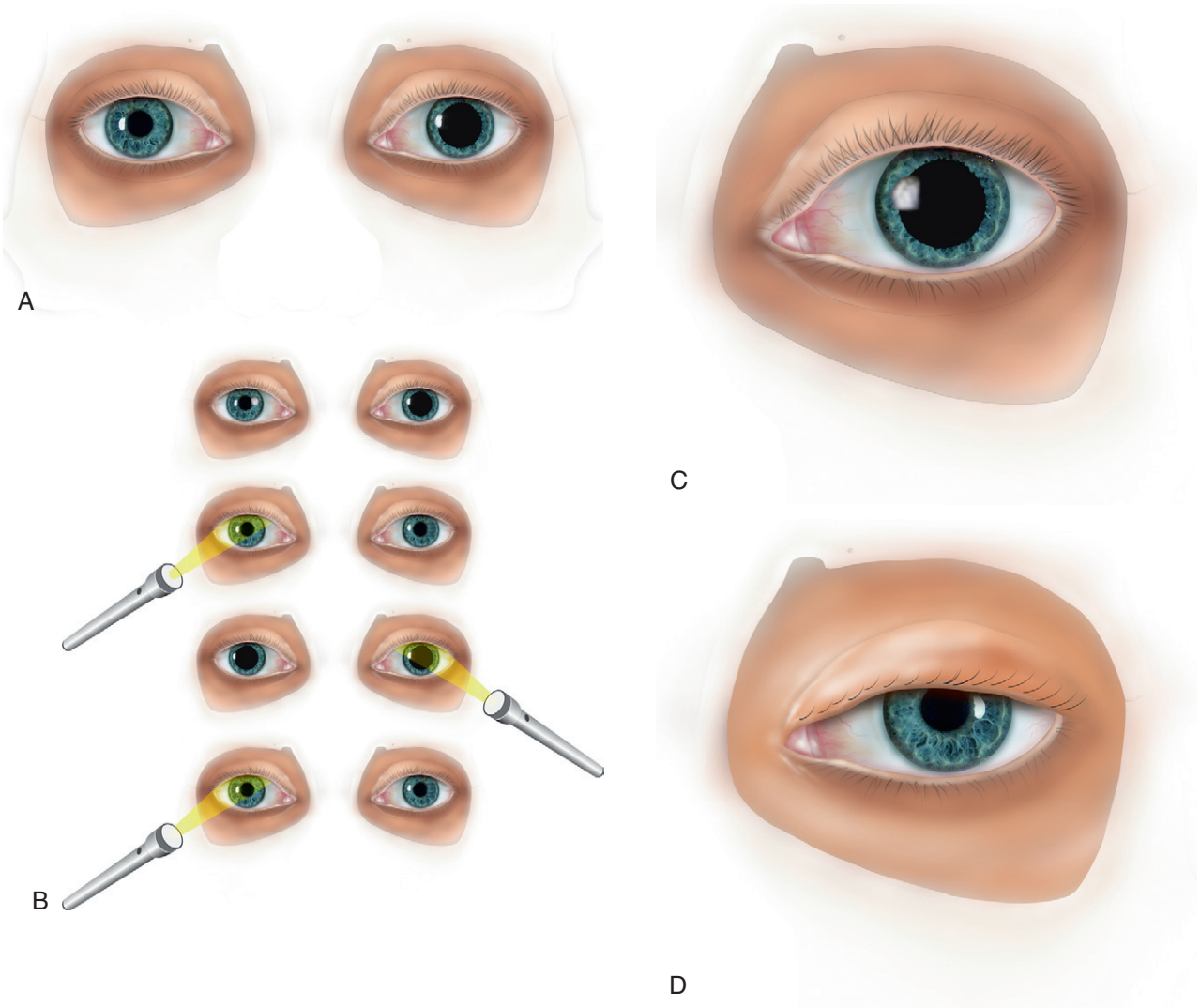
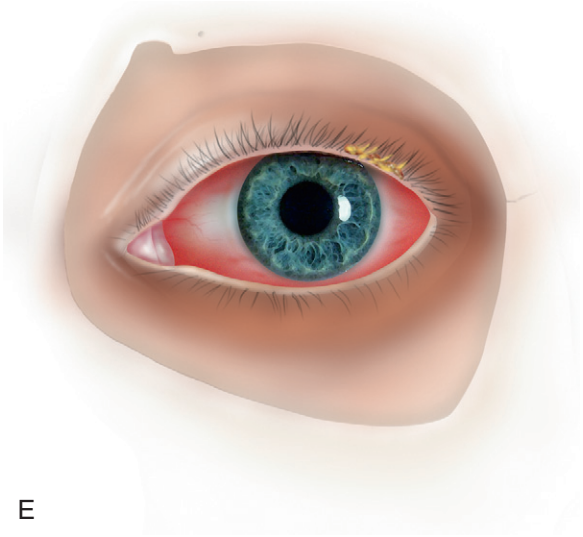
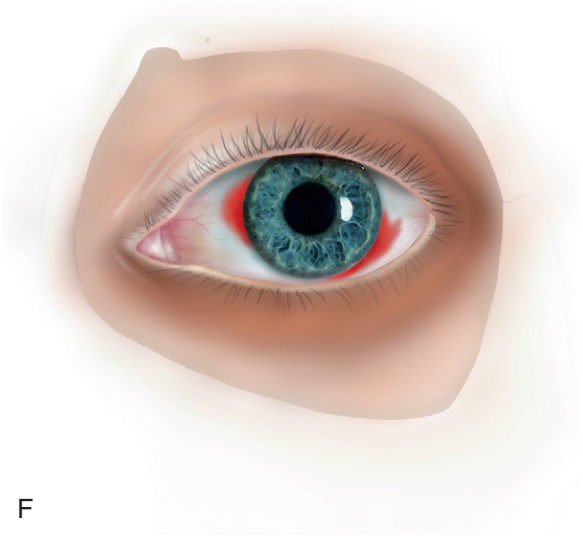


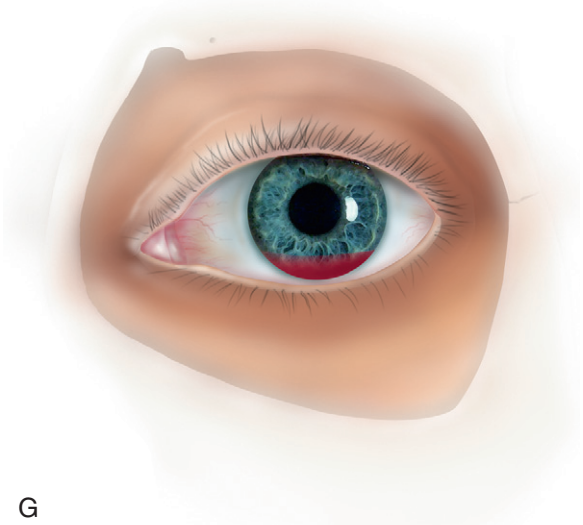
FIGURE 11-2 **A,** Anisocoria. **B,** RAPD—Marcus Gunn pupil. **C,** Fixed dilated pupil. **D,** Ptosis.



E



F



G



H

FIGURE 11-2, cont'd **E,** Conjunctival injection. **F,** Subconjunctival hemorrhage. **G,** Hyphema. **H,** Restriction in the upward gaze of the left eye caused by muscle entrapment will result in binocular diplopia.

TABLE 11-2 Clinical Findings Associated With Eye Examination

Condition or Finding	Definition, Clinical Significance, and Methods of Identification
PUPILS	
Anisocoria	Inequality in size of the pupils. The pupils should be examined with the patient in a dimly lit room. Head injury and injury to cranial nerve III (oculomotor) should be suspected if anisocoria is present (see Fig. 11-2*A*).
RAPD—Marcus Gunn pupil	Asymmetry in the response of the pupils to light. RAPD indicates injury to cranial nerve II (optic nerve). Directing a light at the normal (uninjured) eye will cause both pupils to constrict. Moving the light to the injured eye will result in dilation of both pupils because of poor or no light perception. Injury to the optic nerve may be caused by many factors, including compression caused by retrobulbar hematoma or hemorrhage, traumatic optic neuropathy, and direct injuries (see Fig. 11-2*B*).
Irregularly shaped pupils	If the pupil is irregularly shaped, globe injury should be highly suspected. If the pupil is tear-shaped, the point of the teardrop will usually be pointing to the area of the globe rupture, especially if eyelid lacerations are present.
Fixed and dilated pupil	If the pupil is fixed and dilated, head injuries should be ruled out before other causes are considered (see Fig. 11-2*C*).
EYELIDS	
Ptosis	Drooping of the eyelid. Ptosis can be the result of paresis of the oculomotor nerve. Horner's syndrome also causes this symptom; the syndrome includes ptosis, miosis, and anhydrosis and is related to interruption of sympathetic nerve stimulation (see Fig. 11-2*D*).
Lagophthalmos	Inability to close the eyelids completely. This condition is most likely caused by damage to cranial nerve VII (facial nerve).

TABLE 11-2 Clinical Findings Associated With Eye Examination—cont'd

Condition or Finding	Definition, Clinical Significance, and Methods of Identification
CONJUNCTIVA	
Conjunctival injection	Dilation of the conjunctival vessels. This condition, which causes a reddish conjunctiva, is *not* the same as subconjunctival hemorrhage (see later; also see Fig. 11-2*E*).
Subconjunctival hemorrhage	Accumulation of blood beneath the conjunctiva. The hemorrhage usually resolves without intervention (see Fig. 11-2*F*).
Chemosis	Edema of the conjunctiva; usually resolves without intervention.
ANTERIOR CHAMBER	
Hyphema	Blood in the anterior chamber. Examination should be performed with the patient sitting up at a 30-degree angle. Blood can be seen in the anterior chamber. Various degrees of blood accumulation can be seen; infrequently, the entire anterior chamber is filled with blood and vision is impaired (see Fig. 11-2*G*).
Corneal abrasions and lacerations	These painful injuries often require examination with fluorescein dye and a Wood's lamp. They are usually treated with ophthalmic ointment and resolve spontaneously.
POSTERIOR CHAMBER	
Vitreous bleeding, retinal detachment	These findings will be seen during fundoscopic examination. They result in impairment of vision.
VISUAL DISTURBANCES	
Reduced visual acuity (VA)	Decrease in the ability to see. A Snellen card should be used, if possible, but having the patient read any fine print is often substituted. Patients who wear glasses or contact lenses or use reading glasses should do so during the test, if possible. Reduced VA, particularly the inability to see light, is a serious finding. VA as determined during the initial encounter with the patient is a strong indicator of the final visual outcome.
DIPLOPIA	
Binocular Diplopia	Double vision that occurs when the patient looks at objects with both eyes. Testing for diplopia at the extremes of visual fields frequently yields positive results. Such diplopia is often related to swelling but can also be the result of entrapment of the extraocular muscles, which results in an asymmetrical movement of the globe, or of injury to cranial nerve III, IV, or VI. Positive results from a forced duction test indicate that the cause of diplopia is entrapment, not nerve injury (see Fig. 11-2*H*).
Monocular diplopia	Defined as double vision in one eye. Possible causes are lens dislocation or bleeding in the anterior chamber, or any other disturbances in the clear media along the visual access.
INTRAOCULAR PRESSURE	
Increased intraocular pressure (IOP)	IOP should be measured with a tonometry device. Elevated IOP could be related to glaucoma (unrelated to the injury). Alternatively, it could be the result of an elevated intraorbital pressure, which in turn increases the pressure exerted on the globe by the periorbita and leads to elevated IOP. A unilateral increase in the IOP should prompt a search for retrobulbar hematoma and a lateral canthotomy should be considered.
Decreased intraocular pressure	A soft globe indicates that the globe has ruptured. In such cases, the affected globe should be exenterated urgently so that the good eye can be protected from sympathetic ophthalmitis, a condition that results from the formation of antibodies caused by uveal prolapse.
PERIORBITAL SOFT TISSUE	
Telecanthus	Increased intercanthal distance. See the discussion of the NOE complex.
Epiphora	Excessive tearing. This finding may be caused by blockage of the nasolacrimal duct, disruption of the canalicular system, or presence of foreign bodies causing corneal irritation. If a disruption of the canalicular system is suspected, a thorough evaluation should be performed and a dacryocystorhinostomy should be considered.
GLOBE POSITION	
Vertical dystopia	Unequal vertical positioning of the globes.
Hypoglobus	The affected globe is positioned inferior to the normal globe. This finding is usually the result of superior mass effect or trauma.
Hyperglobus	The affected globe is positioned superior to the normal globe. This finding is usually caused by an inferior mass defect or trauma. Vertical dystopia can be visualized by using a straight edge (e.g., ruler) to align both medial canthi and comparing the globes.
Exophthalmos, proptosis	Anterior displacement of the globe (>2 mm). This finding can be related to increased IOP, blow-in fracture, or systemic disease.
Enophthalmos	Posterior displacement of the globe (>2 mm). Enophthalmos and exophthalmos can be measured by an exophthalmometer. It can also be roughly established by looking at the patient from above (bird's-eye view) or from below (worm's-eye view).



FIGURE 11-3 Periorbital ecchymosis associated with a left-sided ZMC fracture.



FIGURE 11-4 Bird's-eye view evaluating the patient for malar depression.

EAR

The ear is a complex structure with many details that must be carefully examined. Battle signs (ecchymosis behind the ear), laceration in the ear canal, and blood or CSF in the ear canal may indicate a fracture of the skull base or of the glenoid fossa. The tympanic membrane should be examined for integrity.

MAXILLA, MANDIBLE, AND DENTOALVEOLAR STRUCTURES

The maxilla, mandible, and the dentoalveolar structures should be carefully examined. Missing teeth should be noted and accounted for. Radiographs (e.g., anteroposterior [AP], lateral chest films, and/or an abdominal film) may be necessary to rule out aspiration or swallowing of teeth; it is also possible that these teeth could be lodged in associated soft tissue wounds. Ecchymosis or lacerations in the maxillary or mandibular buccal vestibules or in the floor of the mouth indicate underlying fractures (Fig. 11-5). If the patient is responsive, the maximal incisal opening and any deviation of the mandible upon opening should be evaluated; abnormal findings will indicate the location of mandibular fractures. The patient's occlusion should be evaluated next; for example, an anterior open bite indicates a condylar or subcondylar fracture (Fig. 11-6).

Palpation should be used to detect step deformities and associated segment mobility. Testing for the presence of a LeFort fracture requires that the nasal complex be held gently with one hand and the premaxilla with the other. The examiner should attempt to move the maxilla while assessing the nasal complex for mobility (Fig. 11-7). Any lacerations should be thoroughly explored, especially if injuries to nerves, vessels, and salivary glands or ducts are suspected.

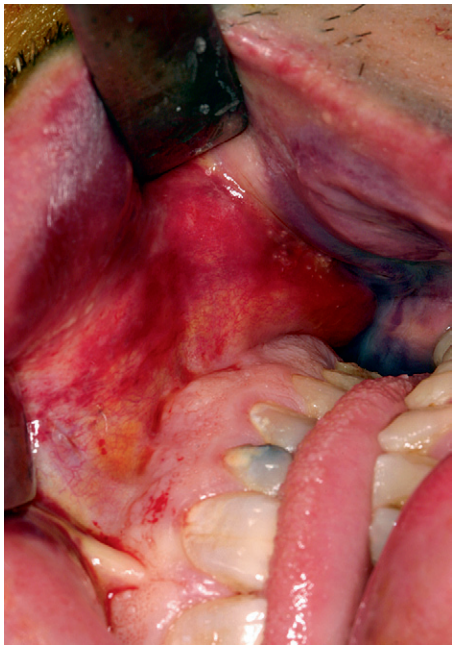


FIGURE 11-5 Hematoma in the maxillary vestibule.



FIGURE 11-6 Occlusal disharmony. Note the premature occlusion on the left posterior segment causing an anterior open bite and marked dental midline discrepancy.

NECK

Anatomically, the neck is one of the most condensed areas of the body. It contains major blood vessels, nerves, nerve plexuses, a portion of the spinal column and cord, endocrine organs, the trachea, esophagus, hypopharynx, and oropharynx, and many lymphatics and the thoracic duct (Table 11-3).

The neck has been typically divided into three zones—I, II, and III.[11] Management of penetrating neck injuries has been historically influenced by the zone of injury. Although the methods of managing these types of injuries have changed, an understanding of these zones is



FIGURE 11-7 Examination of the maxilla. The examiner is holding the nasal complex gently while attempting to move the maxilla. Mobility felt in the nasal complex indicates a LeFort fracture.

TABLE 11-3 Structures of the Neck

Structure	Features
Arteries	Common carotid, internal carotid, external carotid (and cervical branches), vertebral artery, innominate artery, subclavian artery
Veins	Internal jugular vein, external jugular vein, vertebral vein, subclavian vein
Nerves and nerve plexuses	Vagus nerve, phrenic nerve, hypoglossal nerve, spinal accessory nerve, lingual nerve, branches of the third division of the trigeminal nerve, facial nerve, sympathetic chain, cervical plexus, brachial plexus, spinal cord
Digestive system organs	Esophagus, hypopharynx, oropharynx
Lymphatic system	Thoracic duct, multiple lymph nodes
Respiratory system	Lung apices, trachea, larynx
Musculoskeletal system	Vertebral column, clavicle, neck musculature
Endocrine organs	Thyroid gland, parathyroid glands

still beneficial and assists in the decision making process (Fig. 11-8).

Zone I: From the level of the clavicles to the cricoid cartilage

Zone II: From the level of the cricoid cartilage to the level of the angle of the mandible

Zone III: From the level of the angle of the mandible to the base of the skull

The approach to penetrating neck injuries has changed in recent years. Previous recommendations included angiography of all penetrating injuries to zones I and III and elective exploration of all zone II injuries deeper than the platysma muscle, with minimal emphasis on the findings of physical examination. One study has shown that detailed physical examination has a higher yield and better sensitivity and specificity than those

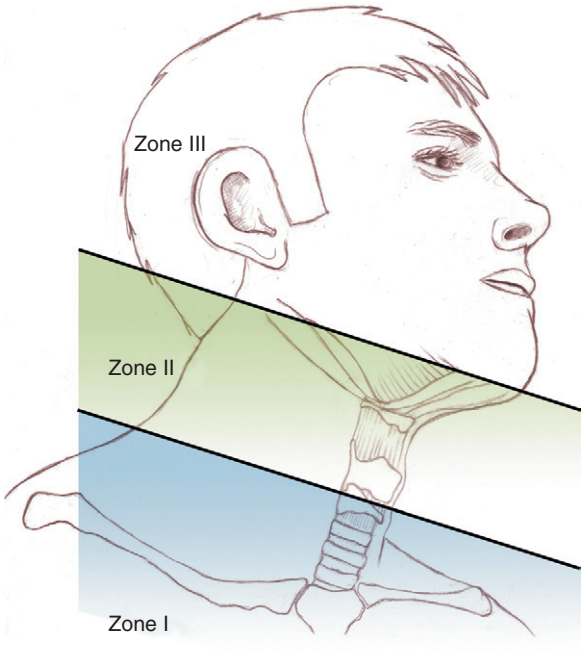


FIGURE 11-8 Zones of the neck.

historically provided by exploration and angiography. Additionally, physical examination is more time- efficient and, unlike surgery and angiography, is not associated with complications.[12] Positive findings that warrant interventions will be discussed in this section.

The following hard signs of vascular injuries indicate the need for immediate intervention[11-13]:

- Expanding hematoma
- Exsanguinating hemorrhage
- Shock unresponsive to fluid resuscitation
- Airway compromise
- Massive subcutaneous emphysema
- Absent carotid pulse
- Carotid bruit or thrill
- Diminished radial pulses
- Differential peripheral blood pressure reading between the right and left arms
- Clavicle fracture

The following are signs of injuries to the aerodigestive tract:

- Dysphagia
- Hoarseness
- Air bubbling from a wound
- Crepitus
- Dyspnea
- Subcutaneous emphysema

The patient should also be examined for potential neurologic defects. This examination should include evaluation of the sensory and motor functions of the cranial nerves, cervical plexus, and brachial plexus.[14] If the examination yields any positive findings, further treatment is warranted. Because no universal algorithm exists to guide the management of neck injuries, the examiner should follow the institution's protocol. In general, a hard sign of vascular injury in zone II warrants emergent surgical exploration and a hard sign of



FIGURE 11-9 **A,** The patient sustained multiple stab wounds to the face. **B, C,** Note the weakness of the muscles of facial expression during animation. (Courtesy Dr. Daniel Plank.)

vascular injury in zones I or II warrant an emergency angiogram. Signs and symptoms of laryngeal, tracheal, or esophageal injury should lead to rapid surgical intervention. Injuries involving the thoracic duct or the thyroid or parathyroid glands are not as pressing as are the injuries detailed here, but they do require urgent and thorough treatment.

CRANIAL NERVES

A thorough head to toe clinical examination can rule out injury to most of the cranial nerves. Of interest to the maxillofacial surgeon are the fifth (trigeminal) and seventh (facial) cranial nerves. Any motor or sensory defects should be noted, and a high suspicion of nerve injury should prompt the surgeon to explore any wounds so that early nerve repair can be performed if necessary. The second (optic), third (oculomotor), fourth (trochlear), and sixth (abducent) cranial nerves are examined as part of the orbital examination. An objective examination of the first (olfactory) cranial nerve is difficult, especially if the patient is traumatized or uncooperative; however, a gross abnormality in the sense of smell is indicative of injury to this nerve. Fractures of the cribriform plate or anterior base of the skull should be suspected if this symptom is reported.

Facial nerve injury can result from blunt or penetrating injuries to the face. Clinical examination and recording of the baseline function of the facial nerve are essential but are extremely difficult if the patient is unresponsive; however, a fingernail pinch or sternal rub can produce a grimace and facial animation. A responsive patient should be asked to animate all the facial muscles; the results should be recorded by using a facial nerve grading system such as the House-Brackmann scale.[15] If the patient exhibits loss of motor function in the upper and lower face, injury to the main branch of the facial nerve should be suspected. Injury confined to one branch of the facial nerve will produce a localized loss of function associated with the affected muscles. Any lacerations in the face, especially those proximal to an imaginary vertical line dropped from the lateral canthus, should be explored for facial nerve injuries. Delayed diagnosis leads to serious aesthetic sequelae[16] (Fig. 11-9).

The examination of facial sensation is important in determining the presence of injuries to the trigeminal nerve. Sensory changes in the lower lip and chin are frequently seen with fractures of the mandibular body because of involvement of the inferior alveolar nerve, part of the third division of the trigeminal nerve. Paresthesia, hypoesthesia, or anesthesia in the upper lip region in a patient with evidence of malar flattening indicates damage to the second division of the trigeminal nerve. The same findings on the forehead indicate an injury to the first branch of the trigeminal nerve. This specific finding should alert the surgeon to a possible orbital apex or superior orbital fissure syndrome.

Neck injuries should prompt examination of the spinal accessory, vagus, glossopharyngeal, and hypoglossal nerves. The sympathetic cervical chain can also be affected. Hearing disturbances should alert the examiner to injuries of the vestibulocochlear nerve (cranial nerve VIII).

RADIOGRAPHIC EVALUATION

In today's level I trauma centers, imaging technology is advanced, and diagnostic abilities are extremely high. It may be tempting for the craniofacial trauma surgeon to rely heavily on reports from emergency room physicians and radiologists. However, it is in the best interest of all involved (patient, surgeon, health care payers) to order scans that are dictated by the findings of the clinical examination and mechanism of injury. For a young

healthy patient who has sustained an isolated fracture of the mandible as the result of a low-energy event (e.g., a punch to the face), plain radiographs of the mandible in three spatial planes are sufficient. Conversely, the same patient who has sustained a mandible fracture as the result of ejection from a motor vehicle will require a much more extensive evaluation, including neurologic and maxillofacial computed tomography (CT) scans, cervical spine evaluation, possibly with plain films, CT scans, and magnetic resonance imaging (MRI), plain films of the chest, abdominal ultrasound and CT, and other indicated images of the thorax and extremities.

When the findings of physical examination indicate the possibility of fractures of the midface, frontal bone, or skull, CT scanning is the most commonly used technique. Head CT scans are routinely ordered for patients who have lost consciousness or have exhibited changes in the level of consciousness. CT imaging of the face is most effective diagnostically when axial, coronal, and sagittal views are obtained. When orbital injuries are suspected, coronal images will most definitively diagnose orbital floor fractures and muscle entrapment (e.g., entrapment of the inferior rectus of the globe). In our opinion, plain radiographs are indicated for the diagnosis of midface trauma only when a CT scanner is unavailable.

Plain radiographs are more commonly ordered to establish fractures of the mandible. The mandible must be viewed in three spatial planes. Panographic radiographs offer a two-dimensional representation of the lower jaw; posteroanterior (PA) and Towne's views of the mandible identify the mediolateral position of condylar fractures and indicate the amount of mediolateral displacement of horizontal mandible fractures. CT scans of the mandible offer an occlusal view that is useful when bilateral condylar and symphyseal fractures are present. Coronal CT scans also help localize the position and size of condylar fracture segments. Occlusal and periapical radiographs are invaluable for the further diagnosis of dentoalveolar fractures, avulsed and fractured teeth, and foreign bodies. When maxillofacial CT scans have been obtained because of suspected midface fractures and the mandible is included, plain films of the mandible are unnecessary. A traditional dental radiographic survey can contribute to treatment planning by highlighting the patient's oral health before the accident. Treatment planning for a mandibular fracture that is amenable to closed reduction may be modified if the remaining dentition is mobile and unhealthy.

POSTOPERATIVE CONSIDERATIONS

Multiple goals should be considered when formulating the operative plan for the stable maxillofacial trauma patient. Intuitively, the immediate goal is restoring the maxillofacial skeleton and soft tissue drape to their pretraumatic condition, both functionally and aesthetically. The intermediate goals also address the patient's postoperative rehabilitation, including nutrition and airway considerations (see later). These considerations often require operative modifications, such as a short-term tracheostomy. The long-term goals are as important as the immediate reduction of the facial bones; these include social and psychological support to ensure the patient's return to the community with minimal delay and easy integration.

Each patient presents unique challenges associated with the planning of an airway in the immediate postoperative period. The patient could arrive in the operating room awake and alert and breathing, without need of a protected airway, or an airway device associated with a resuscitation procedure may still be in place. Often, the situation is further complicated by the presence or suspicion of a cervical spine injury. When patients with maxillofacial injuries exhibit airway compromise or cognitive impairment, securing the airway is an emergency procedure.[17]

There are many options for securing the airway, including orotracheal or nasotracheal intubation, cricothyroidotomy, and tracheotomy. When possible, postoperative airway issues should be considered prior to any operative intervention.

Nutrition is usually affected in patients with facial trauma. Postoperative pain frequently limits the oral intake of these patients. Dietary suggestions, consultation with nutritional experts while the patient is hospitalized, and postoperative pain management should be part of the planning for the postoperative course. Maxillomandibular fixation (MMF) presents a special consideration for postoperative management. When patients are awake and responsive, teaching them to feed themselves is essential. Generally, they should be instructed to maintain a high-protein, high-calorie diet while MMF is in place.

For patients who are unresponsive or unable to maintain an oral diet, planning should include enteral feeding with a nasogastric feeding tube, such as a Dobhoff tube or percutaneous endoscopic gastrotomy (PEG) tube. Because enteral feeding has many advantages (e.g., low cost, maintenance of an intact gastrointestinal mucosal lining, reduction of the risk of gastric bleeding, ease of delivery of a large amount of good-quality nutrition, improved wound healing), it should be planned whenever possible. It has also been linked to shorter hospital stays.[18] If enteral feeding is impossible, parenteral feeding should be started without delay.

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CHAPTER

12

Radiographic Evaluation of Facial Injuries

Maria B. Papageorge | Daniel Oreadi

OUTLINE

Maxillofacial Skeleton and Significance of Osseous Structures
Causes and Classification of Facial Injuries
Diagnostic Imaging of Maxillofacial Injuries
Imaging Modalities
Radiographic Evaluation
Facial Fractures
Upper Face Fractures
Midface Fractures
Lower Face Fractures

Maxillofacial trauma is becoming an increasingly prevalent part of the multiple trauma victim and can be extremely complex in nature. Of all trauma patients, 25% sustain a facial injury. Although facial trauma alone is rarely life-threatening, associated injuries can be devastating to the precarious patient if not diagnosed early. Therefore, the treating clinical team must constantly be aware of the overall stability of the trauma patient and quickly diagnose and treat any threatening conditions. One must also always maintain a high index of suspicion based on the mechanism of injury.

Accurate diagnosis is essential for the proper treatment of facial injuries and minimizing postoperative morbidity for the patient. Knowledge of the bony and soft tissue anatomy of the craniofacial region and proper clinical and radiographic evaluation are paramount. Although the basic treatment principles for the management of maxillofacial trauma remains unchanged, there have been significant advances in diagnostic techniques, thus improving postoperative clinical results and patient comfort and reducing morbidity.

Until the 1980s, diagnostic imaging of facial injuries consisted almost exclusively of standard facial and panoramic radiographs and, if available, tomographic studies. Although standard radiographs are still useful for assessing these injuries and their repair, especially during the intraoperative and early postoperative periods, computed tomography (CT) is widely and routinely used as initial or supplemental diagnostic imaging of facial trauma. This chapter will review the role of diagnostic imaging in the evaluation of the patient with maxillofacial trauma, radiographic maxillofacial anatomy, and various imaging techniques.[1]

MAXILLOFACIAL SKELETON AND SIGNIFICANCE OF OSSEOUS STRUCTURES

In the presence of trauma, there will be alteration of the normal anatomy and symmetry; therefore, a basic understanding and knowledge of the osseous structures of the maxillofacial region and of the basic biomechanical strengths and weaknesses of the facial skeleton are necessary. In addition, focusing on certain anatomic landmarks will facilitate radiographic evaluation.

There are a number of inherent structural strengths and weaknesses in the facial skeleton. The strengths include the maxillofacial buttresses, which are important in the structural support of the facial skeletal complex. Their function is the transmission of forces to the cranium. The weaknesses in the facial skeleton include the lack of complete incongruity between the base of the facial skeleton and cranium and the presence of numerous air-filled sinuses and passages with thin membranous walls. In addition, the attachments between the maxilla, zygoma, and cranium are through sutures that can readily separate.

There are three vertical buttresses of the midface that provide the primary support in the vertical and anteroposterior (AP) directions. These are the nasomaxillary, zygomatic, and pterygomaxillary buttresses[2] (Fig. 12-1*A*). In the AP direction. the structures that support the facial projection are the frontal bone, zygomatic arch and zygoma complex, maxillary alveolus, palate, and basal segment of the mandible from one angle to the other.[3] The main objective of the vertical buttresses is to dissipate forces and transmit them along a vertically oriented vector. In addition, the buttresses also maintain the spatial position of the maxilla in relation to the cranium above and the mandible below.

There are also three horizontal buttresses of the maxillofacial region—the superior, middle, and inferior buttresses (see Fig. 12-1*B*). The superior horizontal buttress is composed of the orbital plate of the frontal bone and cribriform plate of the ethmoid. The middle horizontal buttress consists of the zygomatic process of the temporal bone, body and temporal process of the zygoma, infraorbital process, orbital surface of the maxilla, and segments of the frontal process of the maxilla. This buttress provides lateral stability to the facial skeleton and protects the central facial skeleton from horizontally directed forces. The inferior horizontal buttress consists of the

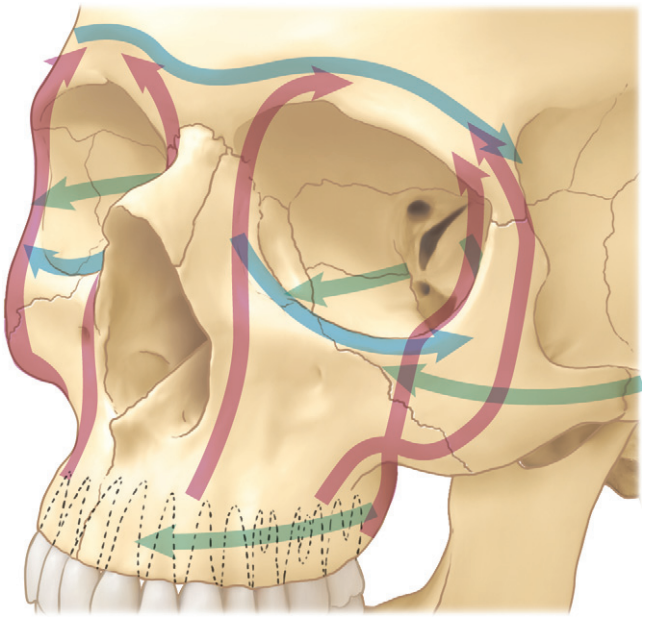


FIGURE 12-1 Vertical buttresses (red lines) and horizontal butresses (green and blue lines).

alveolar ridge and hard palate.[4] Because of their functions, realignment and proper reconstruction of the buttresses in three dimensions during repair of maxillofacial fractures is an important step for preserving function and appearance.[5]

CAUSES AND CLASSIFICATION OF FACIAL INJURIES

The most common causes of facial injuries include motor and other vehicular accidents, altercations, falls, and sports- and work-related injuries.[4] The degree of force delivered by the impact to the skeletal tissue plays a role in the severity or complexity of the resultant injury. Low-energy forces may cause little comminution or displacement, moderate-energy injuries have an increased chance of fracture displacement, and high-energy injuries could result in highly comminuted fractures, accompanied by dramatic instability and marked alteration in facial skeletal architecture.[1] Therefore, the cause of the injury is an important component of the patient's evaluation and assessment. Knowing the mechanism of the trauma may lead to more specificity in the clinical examination and acquisition of radiographic images and their assessment. Classification of facial injuries is also important to define the treatment better and develop a successful plan. In addition to a thorough clinical examination of the patient, radiographic analysis is extremely helpful in classifying facial injuries and plays a central role in providing essential information for the initial diagnosis and treatment. Maxillofacial injuries can range from isolated fractures involving only one or two osseous structures of the facial skeleton to complex facial injuries involving the entire osseous facial skeleton, with different degrees of displacement. Important clinical and radiographic findings that need to be adequately defined include severity of injury, comminution, bone loss, occlusal disharmony, loss of teeth, alveolar fractures, presence of condylar fracture, displacement of segments, and entrapment of vital structures.

The facial radiographic examination should also depend somewhat on the clinical assessment and suspected involvement based on signs and symptoms. The purpose of the radiograph is to confirm the suspected clinical diagnosis, rule out the presence of fractures or foreign bodies, and obtain information that may not be clear from the clinical examination to determine the diagnosis and extent of injury more accurately. The radiographic examination should also document fractures from different angles or perspectives to classify them and determine the best treatment for the patient.

DIAGNOSTIC IMAGING OF MAXILLOFACIAL INJURIES

IMAGING MODALITIES

Plain Films

Radiographic evaluation of severe facial trauma requires some essential radiographic projections and, although CT with three-dimensional reformatting has become the imaging modality of choice in complex facial trauma, plain films are still widely used in the initial evaluation of the trauma patient. The facial bone series consists of three to five projections, including the lateral cephalic, Caldwell's lateral oblique, and Waters' views. The submentovertex (SMV) and Towne's view can also be obtained to help delineate fractures not seen on the other views (Fig. 12-2).

Towne's view is the most useful for assessing subcondylar fractures because it is the only plain radiographic study that optimally demonstrates lateral or medial angulation and/or displacement in these fracture patterns (Fig. 12-3). It is also useful for the evaluation of the orbits because it provides optimal demonstration of the inferior orbital fissure. Towne's view also provides additional exposure of the maxillary sinuses and inferior orbital rims, aiding in the postoperative evaluation after fracture repair. When Towne's view is obtained from a posteroanterior (PA) angulation it is useful to evaluate areas such as the petrous ridges and mastoid air cells, as well as the foramen magnum, dorsum sellae, and occipital bone.[6] In the reverse Towne's projection, which is a reverse of the half-axial or Towne's view, the same fracture patterns seen on the PA Towne's view can be observed.[7]

Lateral cephalic views can provide information for evaluation of the airway, retropharyngeal soft tissue, anterior and posterior maxillary antral walls, and anterior alveolar ridge, as well as fractures involving the midface, such as LeFort I, II, and III fractures and nasal fractures (Fig. 12-4). PA films are also useful for evaluating midface and mandibular fractures (Fig. 12-5). The Caldwell view is used for evaluation of the midface and paranasal sinuses and provides the best view of the orbits and posterior facial structures. This view is particularly useful for evaluating the nasofrontal and vertical segments of the zygomatic buttresses, nasal fossa, and mandible. Lateral oblique views can be used for evaluation of the



FIGURE 12-2 Facial series. **A,** Waters' view. **B,** Lateral cephalic view. **C,** Lateral oblique view. **D,** SMV view.

mandibular angle and condyles. Waters' view is useful for evaluating the midface and delineating fractures of the orbital rims, zygomatic arches, and anterior facial structures. The SMV view provides a good view for zygomatic arch and midface fractures.

As supplemental films, the panoramic radiograph is extremely useful when evaluating for mandibular fractures, including fractures of the condyle. In addition, occlusal films can be used to evaluate dentoalveolar injuries.

Cervical Spine Films

In cases of facial trauma, cervical spine (C-spine) injuries should be ruled out with a complete cervical spine series, which includes lateral view (cross table), odontoid (open mouth), and oblique views before any manipulation of the neck. Undiagnosed cervical spine injury can occur in 15% to 30% of patients and lateral views have a false-negative rate of 26% to 40%.[8] In equivocal clinical cases, CT is recommended because it identifies more fractures and is more accurate in locating the position of bone fragments. CT scans are also recommended for all unconscious patients with suspected neck trauma and all alert conscious patients who complain of neck pain and spasms after high-velocity injuries. CT, however, is not adequate for identifying ligamentous injuries in the cervical spine and, although they can be identified in flexion-extension lateral views, this is a potentially hazardous procedure. It must be done only for alert and cooperative patients with minimal spasm who have no evidence of a potentially unstable fracture on plain film or CT imaging. MRI, on the other hand, can identify ligamentous injuries and should be considered if the previous investigations remain unhelpful or cannot be undertaken.



FIGURE 12-3 Towne's view showing clear appreciation of the subcondylar region.



FIGURE 12-5 PA view showing evidence of mandibular fracture.

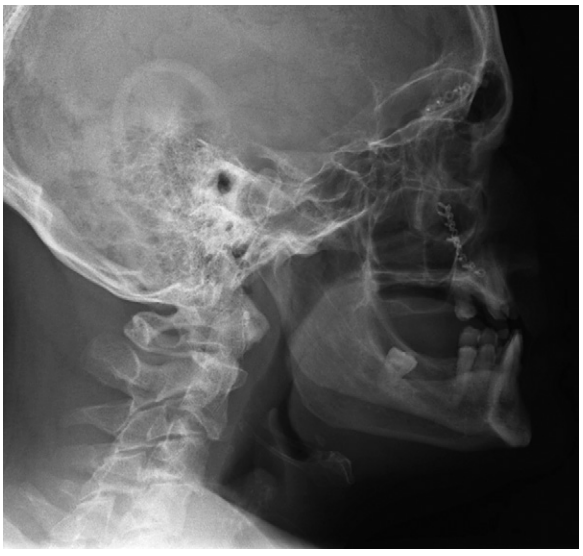


FIGURE 12-4 Lateral cephalic view showing repaired LeFort I fracture.

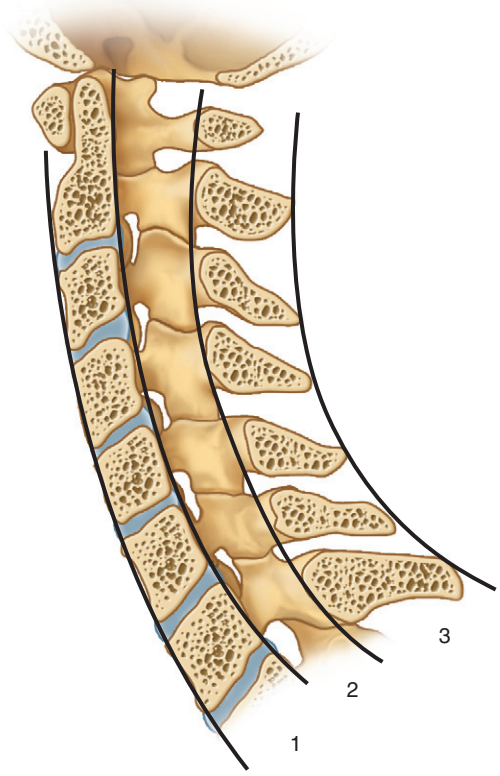


FIGURE 12-6 Denis classification—columns dividing the C-spine in anterior, middle, and posterior segments.

One of the primary purposes of C-spine imaging is to identify potentially unstable injuries; one of the most helpful classifications of instability has been proposed by Denis.[9] This classification divides the spine into three columns (Fig. 12-6):

1. Anterior: This is comprised of the anterior longitudinal ligament, anterior disc, and anterior vertebral body.
2. Middle: This is comprised of the posterior longitudinal ligament, posterior disc, and posterior vertebral body.
3. Posterior: This is comprised of the posterior ligamentous complex, pedicles, lamina, and spinous processes.

An injury is considered unstable if two or three columns are disrupted. An alert asymptomatic patient without a distracting injury or neurologic deficit, and who can complete a functional range of motion

evaluation, may safely be cleared from cervical spine immobilization, even without radiographic evaluation.[10]

Computed Tomography

Although plain films will often provide adequate information, CT scans will often yield additional information or can be used when C-spine precautions or other injuries do not permit standard facial films. There is wide agreement that the exact anatomic identification and quantification of facial fractures, recognition of the true extent of bone displacements, and precise assessment of major bone and soft tissue complications can be effectively and accurately imaged with high-resolution CT.[11]

Since the introduction of CT in the late 1970s and early 1980s, there have been major advancements in the diagnostic imaging of maxillofacial injuries. CT scanning allows for the visualization of injuries of each of the osseous components of the facial skeleton in the axial and coronal planes and allows for the evaluation of various soft tissue injuries. Continued improvements in software have enabled multiplanar reconstruction of image slices in various planes using the digitized data obtained in the initial axial evaluation. These advances in computer technology enable automatic reconstruction of surface models using digitized contour lines and three-dimensional representation on a graphic terminal (Fig. 12-7). Three-dimensional reconstruction of facial bones from two-dimensional images can help guide treatment of facial injuries; in addition to trauma, it is helpful for the evaluation of congenital malformations and pathology.

Although two-dimensional CT scans can be helpful when evaluating facial trauma, one of the difficulties is obtaining coronal sections because they require significant movement of the patient's neck. Thus, the examination is restricted to axial sections and the impossibility of obtaining sagittal sections requires the additional use of conventional tomography.[12] Interpretation of three-dimensional reconstruction allows for easier evaluation and the use of three-dimensional images for the assessment of facial injuries ensures a high degree of reliability in morphologic diagnosis.

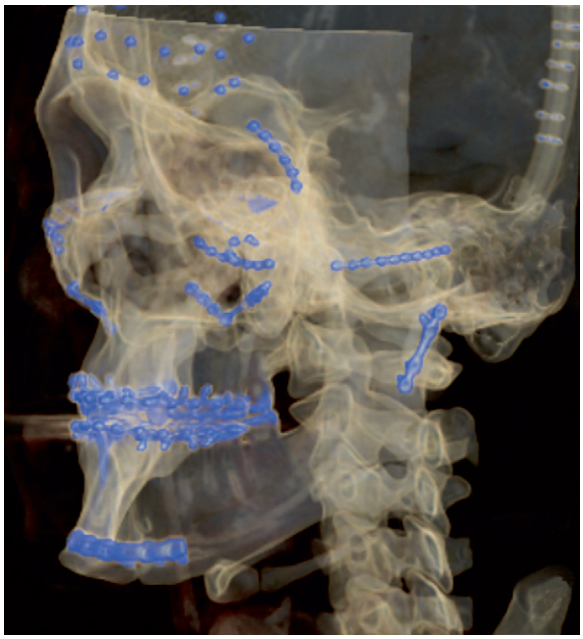


FIGURE 12-7 Three-dimensional reconstruction from CT scan.

CT can also be useful to rule out neurologic injury in the maxillofacial patient and thus can be used to supplement the facial injury evaluation. This imaging is also the most useful in acute situations because of the rapid acquisition times, excellent detail of bone, and ability to examine anatomy in multiple planes. CT scans should also be considered in patients in whom plain films are difficult to assess. For example, midface fractures are difficult to evaluate with plain radiographs because of the overlap of anatomic structures. In these cases, CT scans in several spatial planes of space—axial and coronal and possibly three-dimensional reconstruction—should be considered.

With the increasing role of CT for the assessment of trauma patients, most clinicians advocate its use. It is considered the gold standard for radiographic evaluation of the facial trauma patient. In the past, the use of traditional or single-slice acquisition CT produced images by data collected from detectors after a 360-degree rotation. After each tomographic image, the patient table was moved and another image obtained. A time delay of 10 to 15 seconds between each slice was necessary, which made image acquisition a slower process. The development of spiral or helical scanning has allowed for faster image acquisition.[13,14] Spiral CT involves the simultaneous movement of the patient table and x-ray tube, which results in a volume acquisition of data from which individual tomographic images can be reconstructed. Because a volume data set is acquired, excellent multiplanar reformations are possible when using thin image slices (≤3 mm).[9] Thus, spiral CT scanners can rapidly scan acutely traumatized patients in less than 1 minute and can generate direct images in the scan plane and three-dimensional images in a matter of seconds.

Multidetector CT is another improvement over spiral CT; whereas spiral CT uses a single row of detectors, multidetector CT uses a matrix of detectors that allows for the acquisition of multiple tomographic images per revolution, which greatly increases the speed of imaging. Also, fracture detection has been shown to be significantly higher with thin multiplanar reformations.[15] These are currently considered state of the art imaging for the patient with severe maxillofacial injuries.

Although CT scans of maxillofacial injuries have proven to be invaluable for the diagnosis and treatment of these fractures, one must remember that a treatment plan is based on other variables, including the patient's age, physical status, and preexisting conditions. CT scans should be used as an adjunct in the development of the treatment plan.

Computed Tomography Angiography

CT angiography (CTA) is an important tool in the maxillofacial field for a variety of indications, ranging from the management of traumatic injuries to the treatment of pathologic conditions such as vascular malformations or vessel aneurysms. CTA in combination with MR

angiography (MRA) is highly efficient for the diagnosis of most arterial and venous traumatic lesions in the acute setting and when patients develop delayed symptoms. Conventional angiography is mainly recommended for therapeutic purposes or when the diagnosis remains unclear after performing cross-sectional imaging.[16] Taking into consideration that approximately 25% of penetrating injuries to the neck result in vascular injury, in addition to an 80% chance of carotid artery injury and a 43% chance of vertebral artery injury in the trauma patient, the development of vascular imaging techniques has been beneficial for the diagnosis of these injuries.

Additional diagnostic modalities include dacrocystography, ultrasound, and sialography, which become useful in the delayed management of lacrimal/salivary duct injury.[17] Currently, multidetector row CT provides isotropic data acquisition, allowing imaging reformats with high resolution. This provides an excellent noninvasive evaluation of the major vascular structures of the head and neck regions. Studies have reported 100% sensitivity and 98% specificity in diagnosing vascular occlusion, pseudoaneurysms, fistulas, and partial thrombosis.[7]

Magnetic Resonance Imaging

MRI has become the preferred diagnostic tool for examination of the soft tissue structures of the extracranial head and neck. In the early 1980s, the first MRI scan became available after the independent and simultaneous work of Felix Bloch and Edward Purcell in the 1940s, which led to their being awarded the Nobel Prize for Physics in 1956.[18]

Magnetic resonance is a dynamic and flexible technology that allows one to tailor the imaging study to the anatomic part of interest and disease process being studied. With its dependence on the more biologically variable parameters of proton density, longitudinal relaxation time (T1), and transverse relaxation time (T2), variable image contrast can be achieved by using different pulse sequences and by changing the imaging parameters. Signal intensities on T1, T2, and proton density–weighted images relate to specific tissue characteristics. For example, the changing chemistry and physical structure of hematomas over time directly affects the signal intensity on MRI scans, providing information about the age of the hemorrhage. Moreover, with MRI multiplanar capability, the imaging plane can be optimized for the anatomic area being studied and the relationship of lesions to eloquent areas of the brain can be defined more accurately. Flow-sensitive pulse sequences and MRA yield data about blood flow and can display the vascular anatomy. Even brain function can be investigated by having a subject perform specific mental tasks and noting changes in regional cerebral blood flow and oxygenation. MR spectroscopy has enormous potential for providing information about the biochemistry and metabolism of tissues.[18]

The images obtained by MRI are made when the hydrogen nuclei or protons in the body align along the direction of the main magnetic field. A short radiofrequency (RF) pulse at the proper frequency and duration is then transmitted into the body. The protons absorb RF energy and flip over into a plane that is at an angle with the direction of the main magnetic field. On precession back to their original alignment in the magnetic field, the protons reemit some of the absorbed energy, which induces an electric current in an especially designed RF receiver coil. The induced current (magnetic resonance signal) is then transformed into an image by computerized mathematical methods. Several parameters affect the signal intensity—the density (concentration) of the hydrogen nuclei, characteristics of the nuclei as determined by two different relaxation time constants (T1 and T2), and bulk flow of protons in tissues. Maximal tissue contrast, therefore, can be obtained by properly selecting the data acquisition parameters. Varying the pulse sequence imaging variable, such as pulse repetition time or echo delay time, allows for the discrimination of different tissue types (e.g., fat, blood, bone, muscle).[19]

This technique does not depend on x-rays, which is an advantage over CT. A disadvantage is that the demonstration of fine bone detail in the maxillofacial region is inferior to that of CT. Furthermore, ferromagnetic objects and materials on or inside the patient, such as orthodontic appliances, cardiac pacemakers, neurotransmitters, electronic cochlear implants, and some intracranial aneurysm clips may move, with disastrous consequences. Also, these objects could produce artifacts, which would degrade the resultant image. In addition, poor access and difficulty with some forms of monitoring equipment make MRI less attractive than CT for examining acute maxillofacial trauma. However, MRI has growing applications in a number of specific areas, such as evaluation of the temporomandibular joint, detection of cerebrospinal fluid (CSF) leaks and intraocular injuries, and evaluation of the optic nerve sheath complex. Technologic advancements have also allowed for the evaluation of vascular lesions and structures with the advent of MRA, making this technique extremely valuable in the delayed treatment of traumatic injuries.

When reviewing an MR image, the easiest way to determine which pulse sequence was used, or the weighting of the image, is to look at the CSF. If the CSF is bright (high signal), it is a T2-weighted image. If the CSF is dark, it is a T1-weighted image. Then one should look at the signal intensity of the brain structures. On MRI scans of the brain, the primary determinants of signal intensity and contrast are the T1 and T2 relaxation times. The contrast is distinctly different on T1- and T2-weighted images. Also, brain pathologies have some common signal characteristics. Pathologic lesions can be separated into five major groups by their specific signal characteristics on the three basic images—T2-weighted, proton density–weighted (PD) and fluid-attenuated inversion recovery (FLAIR), and T1-weighted. As an imaging technology, MRI has advanced considerably over the past 10 years, but it continues to evolve, and new appplications will likely be developed.[20]

RADIOGRAPHIC EVALUATION

Evaluation of radiographs requires an organized and systemic approach to identify the injury accurately and minimize the possibility of overlooking the extent of the trauma or underdiagnosing the patient. The skeletal anatomy of the face is the most complex in the body. A

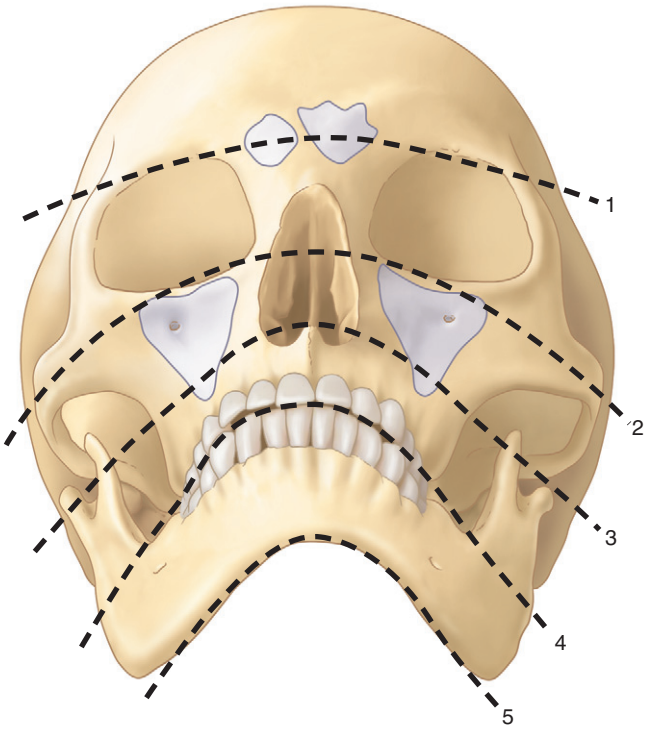


FIGURE 12-8 Trapnell lines facilitate radiographic examination of those parts of the facial skeleton where fractures and other signs are likely to be found.

number of approaches to image assessment in the context of facial injury have been suggested.

McGrigor and Campbell[21] have described a search pattern of four lines that the eye should follow when examining the frontal views. These lines, in addition to a fifth one described by Trapnell,[15] allow one to examine all parts of the facial skeleton where fractures and other signs are likely to be found, thereby reducing chances of misdiagnosis (Fig. 12-8). Dolan et al[22] have described three lines for evaluation of the occipitomental projections that can be used as an adjunct. These lines, known collectively as Dolan's lines are described as the orbital line, zygomatic line, and maxillary line.

Another systemic approach to evaluating radiographs has been presented by Delbalso et al.[19] They described a useful tool as a reminder of what we should be looking for during the evaluation of radiographs. This approach to evaluation involves the four Ss—*s*ymmetry, *s*harpness, *s*inus, and *s*oft tissue.

Symmetry. Each radiographic image should be examined for symmetry or loss of symmetry between respective contralateral structures. Loss of symmetry will result following facial trauma with displacement of facial structures. This is particularly helpful in the evaluation of prominent structures such as the zygoma, nose, maxillary sinus, and orbits (Fig. 12-9).

Sharpness. This refers to the accentuated sharpness of margins or bone fragments in a fracture. Any displacement of the fractured segments can result in the fragment being tangential to the x-ray beam, thus leading to accentuation on the radiographic image. This principle has led to the description of the trap door, bright light,

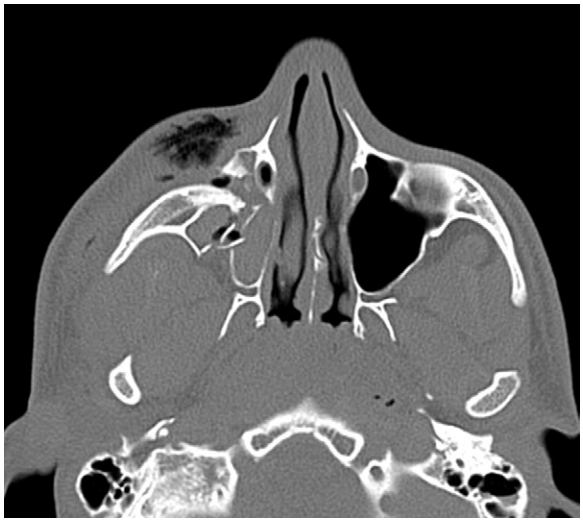


FIGURE 12-9 Approach to evaluation based on the four Ss. Note the marked asymmetry secondary to the ZMC fracture on the right side in this axial cut of the CT scan.

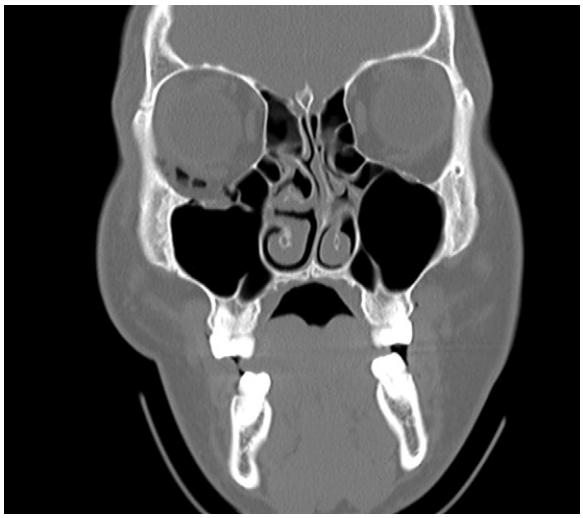


FIGURE 12-10 Trap door sign. A bone fragment is partially avulsed from the orbital floor and hangs into the right maxillary sinus in this coronal cut of the CT scan.

and railroad track radiographic signs. The trap door sign is described in a blowout fracture involving the orbital floor, in which a bone fragment is partially avulsed from the orbital floor and hangs into the maxillary sinus (Fig. 12-10). The bright light sign refers to a displaced free bone fragment in the maxillary sinus (Fig. 12-11). The railroad track sign refers to an additional oblique line in the lateral orbit following a tripod fracture (Fig. 12-12). This results from rotation of the zygoma and its frontal process. When this occurs, the x-ray beam hits the fractured edge of the frontal process in a tangential plane, resulting in an accentuated linear density parallel to the linea innominata (innominate line). This is a radiodense oblique line normally observed in the lateral orbit on the Waters' and Caldwell views formed by the greater wing of the sphenoid.

Sinus. Because all major bone structures of the maxillofacial region border at least one paranasal sinus, trauma



FIGURE 12-11 Bright light sign. A displaced free bone fragment is seen in the maxillary sinus.



FIGURE 12-12 Railroad track sign. This is evident as an oblique line in the left lateral orbit.

in these regions will often result in radiographic changes of these sinuses. These changes can include haziness caused by mucosal edema, opacification, an air-fluid level caused by bleeding secondary to a mucosal tear, and a well-defined mass caused by hematomas. Although haziness and opacification of the sinus can have other causes, such as acute and chronic sinusitis, these changes in the trauma patient must be highly suspicious for fractures (Fig. 12-13).

Soft Tissue. Evaluation of the soft tissue for abnormalities is also important when assessing maxillofacial injury. Soft tissue abnormalities such as edema, signs of penetrating injury with a foreign body, soft tissue emphysema, and intraorbital air could all be signs of osseous injury. Thus, along with radiodense foreign body fragments, the presence of these signs should lead one to examine the

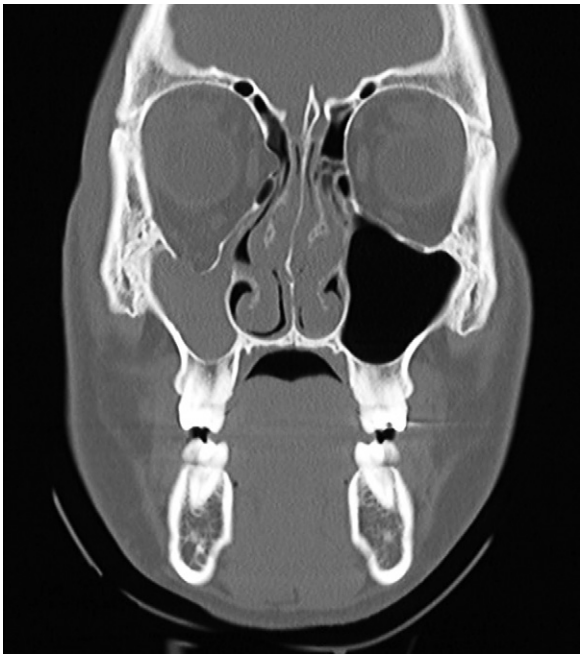


FIGURE 12-13 Complete opacification of the right maxillary antrum secondary to blowout fracture of the orbit. Note the difference when compared with the uninjured left side.

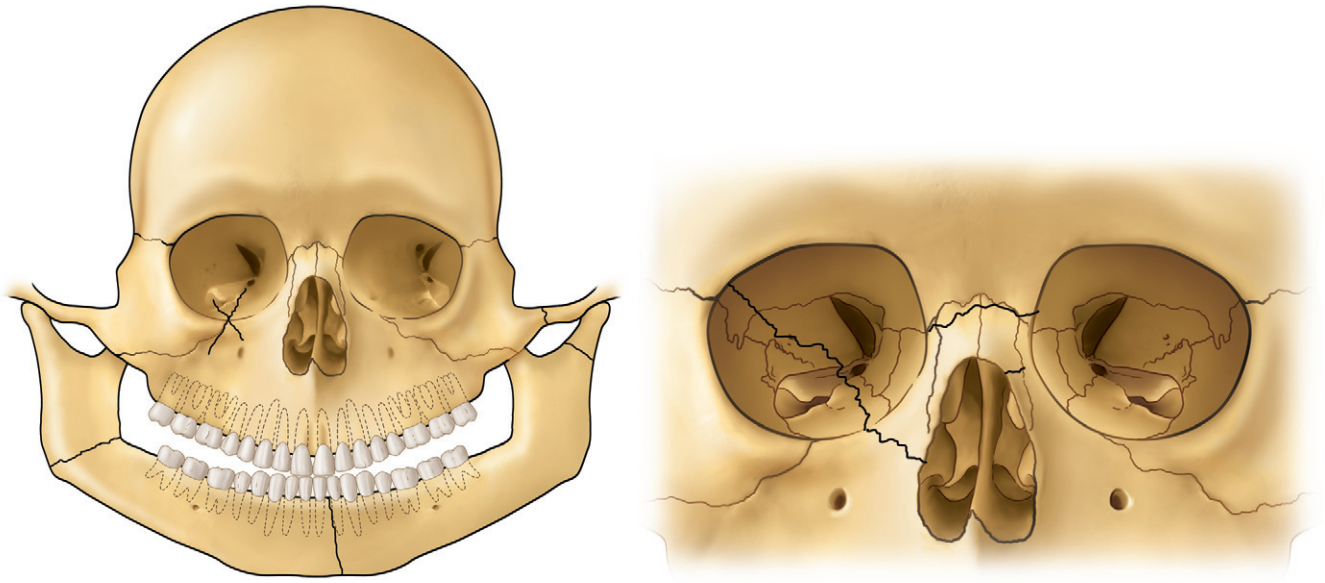
surrounding bony structures more closely. The presence of air emphysema could also signal air introduced by a penetrating injury or a sinus fracture, or could be the result of tracheal or esophageal rupture, with dissection of air superiorly.

In addition to the approaches just described, one should also evaluate systematically and precisely certain key structures of the craniofacial skeleton, including the cranium, nasal structures, paranasal sinuses, and mandible individually. We recommend the use of diagrams (Fig. 12-14) to aid in the systematic and structural radiographic evaluation of the maxillofacial skeleton by identifying each fracture as the CT images are loaded or as the films are viewed. This could be done after the search patterns described earlier are followed, therefore minimizing the possibility of overlooking a fracture. This modality will also allow the surgeon to develop a treatment plan as the diagnoses are made.

Not all fractures are evident radiographically by plain films or CT scanning. Some skeletal injuries are only visible microscopically. Hairline fractures may become visible radiologically after approximately 7 to 10 days, when decalcification at the fracture site edges occurs. Greenstick fractures, in which there is a fracture through one side of the cortex and buckling on the other, and pure buckle fractures are common in children, particularly in the condylar neck. In addition, separation and movement may occur at suture lines and be indiscernible radiologically hence the importance of clinical correlation and knowledge of the mechanism of injury.

FACIAL FRACTURES

These can occur in the upper face, midface, or lower face.



A B

FIGURE 12-14 Skeletal diagrams for fracture identification during imaging review. **A,** Maxillofacial skeleton. **B,** Midface for orbital and NOE fractures.

UPPER FACE FRACTURES

Frontal Bone Fractures

Poor outcomes after the treatment of frontal sinus fractures typically result from misdiagnosis and inadequate planning. In the past, it was mainly Waters' and reverse Towne's projections, lateral skull films, and laminar tomograms that were used to visualize midface and upper face fractures. These films often did not show a complete picture of these fractures, leading to misdiagnosis and thus poor outcomes; they are now secondary to CT scans, which are currently the gold standard for imaging these types of fractures. The CT scan plane of choice for frontal sinus imaging is the axial view, preferably with a slice thickness of 1.0 or 1.5 mm (Fig. 12-15). Using two and three-dimensional CT scans, proper identification of the nasolacrimal duct is important in order to asses for patency and determining the appropriate treatment of choice (Fig. 12-16). Also, in the case of naso-orbital-ethmoid (NOE) involvement, CT scans provide the most information about the medial orbital wall, medial maxillary buttress, and piriform aperture.

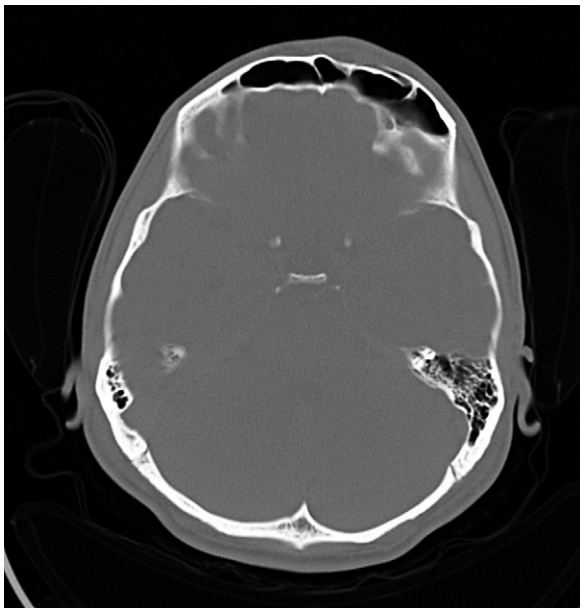


FIGURE 12-15 Frontal bone fracture seen in axial view of the CT scan.

Naso-Orbital-Ethmoid Complex Fractures

NOE complex fractures can sometimes be difficult to interpret and classify radiographically because of the complex anatomy of the region. The NOE region of the face has a large number of air spaces within it and the walls of the septa dividing them are relatively thin, thus making them particularly susceptible to trauma.

Fractures in this region can be isolated fractures confined to one segment of the complex or multiple fractures resulting in massive depression of the entire midface. In light of this anatomic complexity, findings on plain films are also highly variable and often do not demonstrate the full extent of the injury, making CT scans essential for their evaluation.

A simple approach in identifying these fractures from a radiographic standpoint is to determine the predominant area of injury. NOE fractures resulting from a point of impact below the nasofrontal suture result in injury primarily to the ethmoid complex, nose, medial orbit, and upper maxillary sinus (Fig. 12-17). These fractures are often termed *naso-orbital-ethmoid maxillary fracture complexes.*

NOE fractures occur when the point of impact is above the nasofrontal suture, producing an involvement of the adjacent frontal sinus. These fractures can have



FIGURE 12-16 Evidence of involvement of the nasofrontal duct is seen in axial cuts of the CT scan.



FIGURE 12-17 NOE fractures from a point of impact below the nasofrontal suture result in injury primarily to the ethmoid complex, nose, medial orbit, and maxillary sinus.

notable intracranial extension and result in complications such as damage to the nasolacrimal ducts, intracranial air, dural tears, CSF rhinorrhea, and injury to the anterior ethmoid artery. Severe NOE injuries can result in internal telescoping, with a wide range of radiographic findings. These include fractures of the cribriform plate of the ethmoid, fracture of the clivus, and orbital injuries, including disruption of the trochlea of the superior oblique muscle.[23,24]

Because of the anatomic proximity of the NOE complex to the cranium, it is also important for these patients to undergo a thorough examination of the intracranial content, because these injuries are often associated with significant intracranial injuries.

Radiographic findings associated with nasoethmoidal fractures include the following:

- Midface depression
- Fractures of the lamina papyracea
- Opacification of the frontal and ethmoidal sinuses
- Fractures of the inferior, medial, and supraorbital rims
- Fracture of the frontal sinus walls
- Fracture of the superior orbital walls
- Fracture of the sphenoid bones
- Presence of infraorbital and intracranial air
- Fracture of the nasal bones and nasofrontal process of the maxilla
- Fracture of the clivus and base of skull

The high degree of detail required for evaluating NOE fractures necessitates CT axial and coronal views, with slice thicknesses of 1.0 or 1.5 mm. It has been shown that for severe fractures of the NOE region, CT scans provide the most information about the medial orbital wall, medial maxillary buttress, and piriform aperture.

MIDFACE FRACTURES

Orbital Fractures

Orbital fractures can occur isolated or in combination with other injuries, usually zygomaticomaxillary complex (ZMC), NOE, and LeFort II fractures. When isolated, the mechanism of injury is likely caused by an increase in intraocular pressure (IOP), resulting in what is known as blowout or blow-in fractures, referring to floor and medial wall fractures, respectively. It is important to identify these fractures properly because any changes in orbital volume can negatively affect the patient's vision, as well as the involvement of orbital muscles affecting extraocular movements.

The accuracy of plain radiography in diagnosing orbital fractures is poor, with obvious signs being seen only in 10% of cases. Rarely will a fracture or displaced fragment be directly identified. More often, there may be indirect suggestive signs, such as opacification of the ethmoid air cells or orbital emphysema on the affected side, seen in slightly more than 50% of cases. Medial wall fractures rarely cause diplopia but may contribute to enophthalmos. They may be seen in 20% to 40% of cases with blowout fractures.[21] Both CT and MRI may be used to address the questions posed at the beginning of this section. CT has the advantage of delineating the skeletal tissues more effectively than MRI and is frequently indicated for the evaluation of possible concomitant injuries. MRI, on the other hand, is more sensitive in assessing soft tissue components, including orbital fat, muscle, and their possible displacement. In the setting of trauma, fat density (<–10 Hounsfield units [HU]) in the maxillary or ethmoid air cells is related to fracture until proven otherwise. The same rule applies to the presence of intraorbital emphysema.

CT imaging is best reviewed in coronal reformats because it displays the floor of the orbits better than the axial cuts, allowing a clearer assessment (see Fig. 12-11). The sagittal view on the CT scan will provide optimal

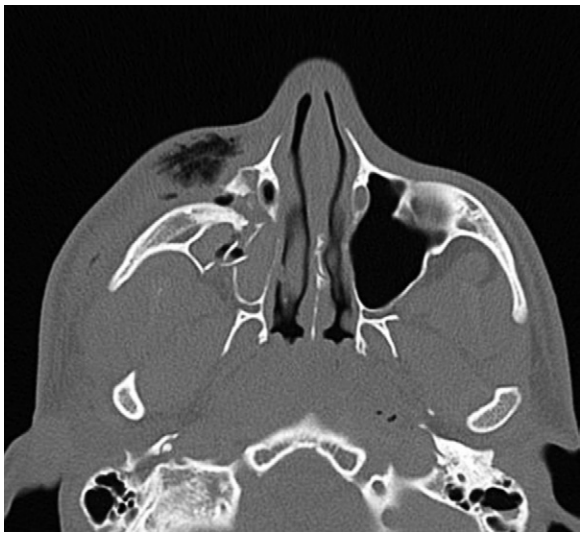


FIGURE 12-18 Fracture involving the zygomaticomaxillary articulations at the level of the infraorbital foramen.



FIGURE 12-19 Water's view showing evidence of repaired ZMC fracture.

demonstration of the AP aspect of the orbital floor, allowing for better appreciation of soft tissue displacement and the anterior and posterior limits of the fracture.

Zygomaticomaxillary Complex Fractures

ZMC fractures, also known as tripod fractures, represent the most common isolated midfacial fracture. They are usually caused by a direct blow to the zygomatic bone, resulting in disruption of its three major attachments. The disruptions occur in the region of the zygomaticomaxillary, zygomaticotemporal, and zygomaticofrontal articulations. The fractures involving the zygomaticomaxillary articulations usually occur medially to the zygomaticomaxillary suture at the level of the infraorbital foramen (Fig. 12-18). It extends circumferentially around the anterior and posterolateral walls of the maxillary sinus and may involve the orbital floor and lateral aspect of the inferior orbital fissure, which can be visualized by Towne's view. The zygomaticotemporal component of a ZMC fracture consists of a separation of the suture line or fracture of the zygomatic arch, at or near the zygomaticotemporal suture. Finally, to complete the tridimensional description of a tripod fracture, we must include the fracture and/or diastasis extending inferomedially from the zygomaticosphenoid articulation to the inferior orbital foramen. These disruptions result in the mobilization of the zygomatic bone, allowing for varying degrees of rotation and/or displacement. Isolated zygomatic fractures result from direct trauma to the arch, causing its fracture in two or more places, and may also involve the coronoid process, depending on the force of the trauma.

Plain film evaluation of the patient suspected to have a ZMC fracture includes Waters', Caldwell's, and SMV views (Fig. 12-19). Waters' view is the most useful view to evaluate for zygomatic injuries because it defines the injury involving the arch, lateral wall of the maxillary sinus, inferior orbital rim, and floor of the orbit. The Caldwell view provides an undistorted view of the frontal process of the zygoma as well as the lateral orbit, and best defines fractures and diastases involving the zygomaticofrontal suture and lateral orbit. The SMV view is essential for demonstrating the status of the zygomatic arch, posterior displacement, and lateral or medial rotation on the vertical axis. Towne's view is useful for evaluating these fractures because it provides an additional demonstration of displacement of the zygoma, depression of the zygomatic arch, and disruption of the inferior orbital rim, as well as the relationship of an orbital floor component.

CT, however, is the accepted imaging modality of choice for use in the evaluation of ZMC fractures. CT with three-dimensional reformatting provides an excellent demonstration of the injury to the osseous orbit and may demonstrate additional osseous injuries that may not be readily apparent on plain film (Fig. 12-20). These include fractures to the orbital apex, greater and lesser wings of the sphenoid bones, and anterior or posterior cortex involvement of the frontal bones. CT evaluation of the orbit is especially important in trauma because these injuries may be occasionally associated with soft tissue entrapment involving the ocular muscles.

Zygomatic Arch

The dense composition of the zygoma makes it structurally functional as a unit, allowing forces to dissipate along its body, with fractures typically occurring at the suture level. The zygomatic arch is susceptible to fractures when direct impact is sustained, making it the most common isolated fracture of the zygoma, comprising 10% to 16% of zygomatic fractures.[22] Fractures involving the proximal end of the zygomatic process of the temporal bone may extend posteriorly to involve the adjacent glenoid fossa of the temporomandibular joint, a finding mostly noted on axial CT scan section examination only. If sufficient medial displacement of the zygomatic arch occurs, impingement of the coronoid process (Fig. 12-21) may cause limitations in mandibular motion.

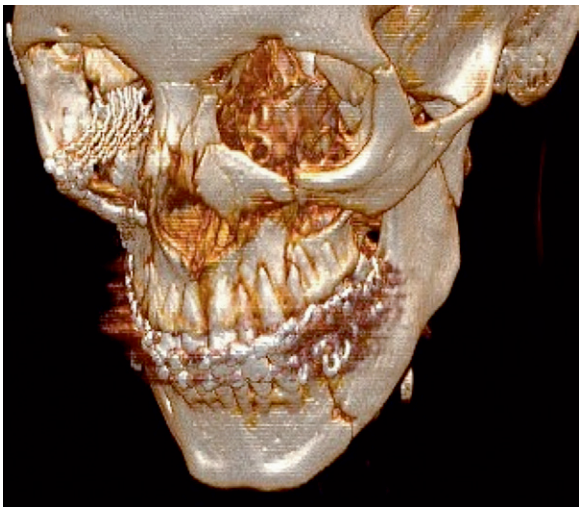


FIGURE 12-20 Three-dimensional reconstruction clearly demonstrating zygomaticomaxillary complex involvement and degree of fracture displacement.



FIGURE 12-21 Fracture of the zygomatic arch causing impingement of tissues surrounding coronoid process.

Some of the radiographic signs of zygomatic arch fracture seen on plain films include the following:

1. Displacement of the arch seen on Water's and Caldwell's views
2. Superimposed radiopaque band over the body of the zygoma on Water's and Caldwell's views
3. Diastasis of the zygomaticotemporal suture seen on Water's and SMV views
4. Depression of the temporal process of the zygoma and zygomatic process of the temporal bone, resulting in a so-called W deformity better visualized on a SMV view (Fig. 12-22)

The vertical radiopaque band superimposed over the body of the zygoma occurs in fracture depression of the zygomatic arch in more than 80% of fractures involving the arch. This is caused by the anterior end of the fractured fragment being tangential to the x-ray beam.



FIGURE 12-22 Submentovertex view showing evidence of zygomatic arch fracture. This is easily visualized when compared with the contralateral uninvolved side.

For accurate evaluation of the zygoma, both axial and coronal images of CT scan are needed. The lateral orbital wall is often interrupted or angulated on axial images. A common confounder is confusing the normal suture for a fracture and, occasionally, a slight widening of the suture (diastasis) is the only clue. Comparison with the opposing site is valuable, as well as evaluation of the orbital floor, which is difficult on axial images. This is why obtaining coronal and sagittal reformats is of extreme importance when evaluating this fracture. The curvilinear orbital floor should appear smooth and interrupted only by the inferior orbital fissure.

LeFort Fractures

A simplified approach to understanding the LeFort classification system of midfacial fractures is to consider the human head as being composed of four principal components—the oval-shaped cranium, the pyramid-shaped facial skeleton, and the paired three-legged zygoma.[12]

The LeFort I fracture usually results from a direct horizontal blow to the lower face, resulting in fractures involving all three walls of the maxillary sinus and pterygoid processes. Radiographically, one may see opacification of the maxillary sinuses and fractures involving the sinus walls on Water's and Caldwell's views. CT scans demonstrate LeFort fractures with more detail, even when there is involvement of the hard palate and alveolar ridges (Fig. 12-23). In contrast to the LeFort II and III fracture complexes, the LeFort I fracture does not involve the glabella or zygoma. The LeFort II fracture results from horizontal trauma to the midface. The fracture lines start in the region of the nasion and extend in an oblique fashion through the medial aspect of the orbits and inferior orbital rims. It then continues posteriorly in a horizontal fashion above the hard palate to involve the



FIGURE 12-23 Coronal view of noncontrast CT scan showing LeFort fracture pattern.



FIGURE 12-24 Axial view of noncontrast CT scan of patient with panfacial fractures.

pterygomaxillary buttresses, resulting in a disarticulation of the pyramid-shaped facial skeleton from the rest of the skull. In contrast to the LeFort III fracture complex, the zygoma remains attached to the cranium.

Radiographic signs of LeFort II fractures include disruption of the nasion and nasal bones and bilateral fractures of the inferomedial orbital rims on frontal projections, depression of the midface and disruption of the nasion and pterygoid plates on lateral projections. Similar findings are noted on axial and coronal CT scan sections.

The LeFort III fracture is the most severe of the LeFort fractures. It represents a complete disarticulation of the pyramidal facial skeleton, along with the zygomas from the ovoid-shaped cranium. The fracture lines extend through the superior aspect of the nasofrontal buttress and then posteriorly through the ethmoid bone along the attachment between the facial skeleton and cranial base to involve the pterygomaxillary buttresses. Bilateral fractures or diastases also occur in the region of the zygomaticofrontal and zygomaticomaxillary sutures. In contrast to the LeFort II fracture complex, the zygomas are avulsed from the cranium, along with the maxilla. The classic imaging findings in the LeFort III fracture include the folllowing:

1. Fracture through the nasion, posterior orbital wall and vertical and horizontal segments of the zygoma
2. Fractures or diastases at or near the zygomaticofrontal or zygomaticotemporal sutures
3. Disruption of the nasion and pterygoid plates and separation between the facial skeleton and calvarium as seen on the lateral view

LeFort fractures may be further classified as being pure, complete, incomplete, and unilateral or hemi-LeFort.[22] A complete LeFort fracture represents a pure LeFort fracture in addition to a fracture(s) in other parts of the maxillary supporting buttresses. An incomplete LeFort fracture complex represents a fracture along one of the three major lines of weakness in the facial skeleton that do not fully involve the entire fracture line. A unilateral or hemi-LeFort fracture represents a transverse fracture of the maxilla that extends to the pterygoid plates and is limited to one side of the face. The pure LeFort fracture is an unusual occurrence because patients with severe midfacial trauma usually have multiple fracture complexes (Fig. 12-24), which led to the introduction of these descriptive terms. If elements of two LeFort fractures are present on the same side, the higher LeFort category is applied when naming the fracture. One should always describe the injury in terms of location, displacement at the fracture site(s), and involvement of adjacent structures.

LOWER FACE FRACTURES

Mandibular and Dentoalveolar Fractures

Mandibular fractures are prevalent in facial trauma, typically with four anatomic sites being mostly affected—angle (32%), condyle (23.3%), body (17.7%), and parasymphysis (15.6%).[12] They can occur as single or multiple fractures involving multiple locations, including the contralateral side, dentoalveolar complex, condyles, and dentition. Mandible fractures can also be classified as simple, comminuted, compound, or complex. They can occur as isolated injuries or in association with other facial fractures. Ellis et al have reported that 50% of patients with maxillofacial injuries have at least one associated mandibular fracture and 33% of midfacial injuries occur in conjunction with mandibular fractures.[20]

There are inherent weaknesses in the mandibular anatomy that predispose it to be such a common fracture. These are the condylar process, mandibular angle, mental foramen, and areas containing impacted teeth or pathologic entities such as cysts, neoplasms, or vascular lesions. The thinnest portion of the mandible is the condylar neck; thus, it is often associated with fractures in other parts of the mandible, such as the angle and parasymphysis areas (Fig. 12-25). This is caused by transmission of the forces sustained by the trauma superiorly



FIGURE 12-25 Panoramic xray showing subcondylar fracture in conjunction with parasynphyseal fracture.



FIGURE 12-26 Panoramic xray showing evidence of symphyseal fracture compound to no. 26.

and posteriorly along the entire mandible from the point of impact. The alveolar complex can also be a point of weakness, depending on the presence or absence of teeth. If associated dentoalveolar injuries are present, all teeth must be accounted for. If it is suspected that teeth are missing, the patient should undergo chest radiography to rule out inhaled foreign bodies.

As with all facial trauma patients, the radiographic examination of the mandibular trauma patient will depend on a number of factors, including the clinical examination, patient symptoms, and type of force sustained. The panoramic radiograph and intraoral films, including occlusal radiographs, can provide a good initial evaluation of mandibular trauma. The panoramic radiograph allows visibility of the entire mandible, including the condyles, dentoalveolar complex, and dentition. It is especially useful in observing the anterior mandible, which is often not well observed in other plain films (Fig. 12-26). Panoramic radiographs are generally easily obtained, as long as the patient's clinical condition allows. The panoramic is a tomographic film that images the mandible in a two-dimensional plane and thus carries some limitations in accurately assessing the displacement and angulation of fractured segments, including the condyles. A recent study comparing the sensitivity and specificity of panoramic radiographs with those of coronal CT scans in the diagnosis of mandibular condylar fractures in the pediatric population has concluded that CT scans provide consistently greater accuracy of diagnosis, with a higher sensitivity and specificity than panoramic radiographs in the assessment of children suspected of having condylar fractures. In view of the high rate of false-negative and false-positive results associated with panoramic radiographs, coronal CT scans should be considered routine investigation in patients whom this type of injury is suspected.[25] Therefore, additional views to the panoramic radiograph should be considered in patients in whom there is a clinical indication of fracture not evident on a panoramic image or to assess the angulation and/or displacement of the fractures better.

The mandibular series, which includes lateral oblique, PA or AP, Towne's, and lateral views can provide further information on the displacement, degree, and/or complexity of the fractures. The PA view, if obtainable, is preferred to the AP view because it provides less distortion of anatomic structures. The lateral oblique projection of the mandible provides views of the condylar and subcondylar regions, coronoid process, mandibular ramus, and angle and proximal portion of the mandibular body of the side closest to the film (see Fig. 12-2). This view does not provide adequate visualization of the contralateral mandible and only poor visualization of the mandibular symphysis because of the overlap of osseous structures. The PA projection provides an excellent view of the entire mandible, except for the condyles, which are superimposed by the temporal bone. Also, nondisplaced symphyseal fractures may be partially obscured by the superimposed cervical spine. This projection allows

visualization of the ramus and coronoid process fractures and can aid in determining the degree of mediolateral displacement of fractures in these regions and in the mandibular body. Towne's projection allows for optimal visualization of the condyle and subcondylar regions, including the degree of mediolateral displacement or dislocation of fractures. Although not an optimal projection, the lateral view can add information to the other views of the mandibular series and is often taken for evaluation of the facial bones and skull. If taken for evaluation of mandibular trauma, it should be obtained with the traumatized side close to the film. As with all films, the side closest to the film will appear sharper and nonmagnified. A true lateral projection would show complete superimposition of the right and left mandible, which is rarely the case. The lateral view usually varies from a true lateral and thus can show both sides of the mandible, especially the rami and posterior borders. The superimposition of the two sides of the mandible makes this a limited film for evaluation of the mandibular body and anterior mandible. The lateral projection, however, is an excellent view for the evaluation of fractures of the posterior borders of the condylar necks, ramus, angle, inferior body of the mandible, and anterior dentoalveolar fractures, including AP displacement of the condyle. Because of the clear visualization of the airway, it is a good projection for viewing foreign bodies in the airway.

On occasion, routine plain films may not provide adequate information and can be supplemented with intraoral radiographs, periapical and especially occlusal films. An occlusal view of the mandible can be taken in the posterior or anterior regions, but usually is indicated to supplement information in the anterior region. Occlusal views of the anterior mandible will show the area between the canines and a clear view of the inferior border of the mandible, thus making it easier to identify a symphysis fracture.

Plain films will often provide adequate information for assessing mandibular trauma. However, when plain films fail to diagnose adequately or the patient's clinical status prevents their acquisition, CT scans and three-dimensional reformatted images can be obtained. These will offer the most accurate view of the fractures because they demonstrate the actual fracture complex in a spatial context in relationship to the surrounding anatomy, to which the clinician can easily relate.

SUMMARY

Maxillofacial trauma can be complex and proper treatment requires accurate evaluation and assessment of the patient. Recognition and proper management are crucial because of the important functions of the craniofacial complex and the vital structures that can be involved. Severe facial injuries can be life-threatening and the psychological impact of facial disfigurement can be devastating. Thorough assessment of the facial trauma patient includes history, clinical examination, and radiographic evaluation. Currently, advances in radiographic techniques allow the surgeon to explore the traumatized anatomy in detail. Radiographic examination may include basic facial films, especially when CT is not available. However, CT, in particular facial multidetector CT, if available, is the most accurate imaging technique and is considered the standard for identification of facial fractures, as well as associated injuries. Three-dimensional reformatting allows better conceptualization and better presurgical planning. CT scanning, along with MRI and MRA, will provide the surgeon with the necessary tools to assess the facial trauma patient fully and provide better treatment outcomes.

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CHAPTER 13

Diagnosis and Management of Dentoalveolar Injuries

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OUTLINE

Examination and Diagnosis
Classification
Injuries to Hard Dental Tissue and Pulp
Injuries to the Periodontal Tissue
Injuries to the Supporting Bone
Injuries to the Gingiva or Oral Mucosa
Treatment
Injuries to Hard Dental Tissue and Pulp
Injuries to the Periodontal Tissue
Injuries to the Supporting Bone
Splinting Techniques
Restoration
Mouth Protectors
Types of Mouth Protectors

Traumatic injuries to the teeth and supporting structures are commonly seen in the injured patient. Orofacial trauma is involved in approximately 15% of all emergency room visits, and 2% of these cases involve isolated dentoalveolar trauma.[1] These injuries can be isolated, as in childhood falls (the major cause of all dental injuries),[2] or can occur in association with other injuries, as seen in motor vehicle accident (MVA) victims. Children commonly sustain dentoalveolar injury usually between the ages of 8 through 12 years.[3] Dentoalveolar injuries pose a major threat to dental health that may even surpass that of caries and periodontal disease.[4]

Dentoalveolar trauma ordinarily results from falls, playground accidents, abuse and domestic violence, bicycle accidents, MVAs, assaults, altercations, and athletic injuries. The dentoalveolar structures can be injured from direct trauma to the teeth or from indirect trauma, usually from forced occlusion, as the mandibular dentition is forcibly closed against the maxillary dentition.[5-7] Direct trauma usually causes injury to the maxillary central incisors because of their relatively exposed position.[8-13] Predisposing factors include abnormal occlusions, overjet exceeding 4 mm, labially inclined incisors, lip incompetence, a short upper lip, and mouth breathing (Fig. 13-1*A*).[14] These conditions can be seen in individuals with class II division I malocclusions or oral habits, such as thumb sucking. Dental injuries are approximately twice as frequent among children with protruding incisors as among children with normal occlusions.[15,16] Therefore, early orthodontic treatment in predisposed children may be an effective prevention strategy.[17] Dental injuries are also common in patients who have been identified as accident prone (see Fig. 13-1*B* and *C*). Indirect trauma to the teeth and supporting structures usually results from a blow to the chin or from a forceful whiplash to the head and neck. If the teeth are out of occlusion or not protected by a mouthguard, they are forced into occlusion, resulting in damage to the posterior teeth, anterior soft tissue, or both (Figs. 13-2 and 13-3).[7] The extent of the dental injury can be characterized by the energy of the impact. A low-velocity blow usually causes damage to the supporting dentoalveolar structures and a high-velocity impact usually results in crown fractures. Objects that are cushioned on impact typically result in alveolar damage and a reduced incidence of crown fractures. Sharp objects usually favor clean crown fractures as opposed to blunt objects, resulting in luxation, or root fractures. Variations in the direction of the impacting force usually result in multiple fracture sites.[11,18]

Injuries to the dentoalveolar structures increase in frequency substantially as a toddler begins to attempt to walk and run, at approximately 1 year of age.[7,10,19,20] The incidence of injury to the dentoalveolar structures in school-age children is reported to be approximately 5%, usually resulting from falls on playgrounds and from bicycle accidents.[13,21-25] Lacerations to the chin and vermilion border of the lip and crown fractures are commonly seen in children.[26] Lacerations of the lip account for most injuries to oral structures.[27] Participation in contact sports, such as hockey, soccer, football, basketball, boxing, and wrestling, can result in oral trauma. The use of an intraoral mouthguard during athletic practice and competition has been found to reduce intraoral injuries, notably in contact and noncontact sports.[28] Skiing and snowboarding have also been shown to increase the risk of dentoalveolar trauma. Helmets with faceguards can help prevent serious trauma to the head and maxillofacial structures.[29]

Multiple injuries of the hard and soft tissues are the result of automobile accidents, with the greatest incidence of dental injuries occurring through the ages of 18 to 23 years.[13] Dental trauma in older age groups may result from assault and domestic abuse, usually closely related to the abuse of drugs or alcohol, or both.[11,30]



A B,C

FIGURE 13-1 **A,** Exposed maxillary incisors, with incompetent upper lip coverage, are frequently involved in trauma associated with falls and facial injury. Teeth may be fractured or avulsed, and associated injury to the lower lip is commonly encountered. **B,** Predisposing factors for direct trauma include abnormal occlusions, overjet exceeding 4 mm, and labially inclined incisors. **C,** Direct trauma to the maxillary incisors is approximately twice as frequent in children with protruding incisors as in children with normal occlusions.



A B

FIGURE 13-2 **A,** Indirect trauma to the dentoalveolar structure is usually the result of falls and blows to the chin. Abrasions and lacerations of the soft tissue of the chin should be investigated further for damage to the posterior teeth or anterior vestibule of the mandible. **B,** Low-velocity trauma associated with falls may result in abrasions to the facial soft tissue and damage to the underlying dentoalveolar structures, especially tooth avulsion in children 7 to 10 years of age. (**B,** Courtesy Dr. Johnson, Iowa City, Iowa.)

Mucosal or gingival lacerations and mobile incisors are seen in oral injuries as a result of child abuse.[31,32] In 1996, 4.3% of children younger than 18 years in the United States were reported to be victims of maltreatment.[33] More than 3 million cases of child abuse are reported each year, with 1 million cases later being substantiated.[34] Approximately 50% to 75% of the physical abuse cases had trauma to the facial and oral regions, with injuries to the upper lip and maxillary labial area.[11,14,35,36] Abuse should always be considered if the child's injuries show a marked discrepancy between the clinical evaluation and the history reported by the supervising adult, or if there appears to be a considerable period between the time of injury and when treatment is sought (Fig. 13-4).[6]

Many dentoalveolar injuries are associated with the management of the comatose patient[37] or the patient undergoing general anesthesia.[14,38-48] Lockhart et al[44] have surveyed 133 directors of training programs in anesthesiology and found that an average of 1 in 1000 tracheal intubations resulted in dental trauma. They also reported that 90% of the dental complications might have been prevented with a screening dental examination of the patient and the use of a mouth protector (Fig. 13-5).[45,46] It is important to remember that a mouth protector can be useful in protecting the anterior teeth during intubation, but it will not protect the teeth from poor technique. A retrospective analysis of 14 years of general anesthetic cases from a university hospital showed a 0.02% incidence in dentoalveolar trauma related to



FIGURE 13-3 Indirect trauma from traumatic occlusion can result in crown fractures of the posterior teeth, lacerations to the anterior vestibule and gingiva of the mandible, and alveolar fractures or displacement of the anterior teeth of the maxillary or mandibular arch.

general anesthesia. Only 50% of these incidents occurred during intubation or extubation procedures, and 80% of all incidents were deemed by anesthesiologists to be unavoidable. Despite adequate prescreening, damage to dentoalveolar structures can occur. Care must be taken to avoid trauma to the dentoalveolar structures at all times during general anesthesia, not just during endotracheal intubation.[47]

Dental professionals are often asked to evaluate these injuries. Good documentation of the injury is essential because these cases may become involved in litigation. In the United States, damaged teeth constitute the most frequent anesthetic-related claim.[48] An increased number of dentoalveolar injuries have also been seen in patients with mental retardation, epilepsy, drug abuse, and congenital abnormalities, such as familial dysautonomia.[11,27]

Dental trauma (single and multitooth) of the primary and permanent dentition occurs primarily in boys,



FIGURE 13-4 Abuse should be considered if the patient's injuries do not appear to correlate with history reported by the supervising individuals and the mechanism of trauma. **A,** Falls commonly result in lacerations to the chin rather than to the side of the cheek, which are more commonly seen with blows. **B,** Domestic physical abuse cases commonly involve trauma to the orofacial region of the body, especially the perioral structures. (**A,** Courtesy Dr. J. Berg, Houston.)

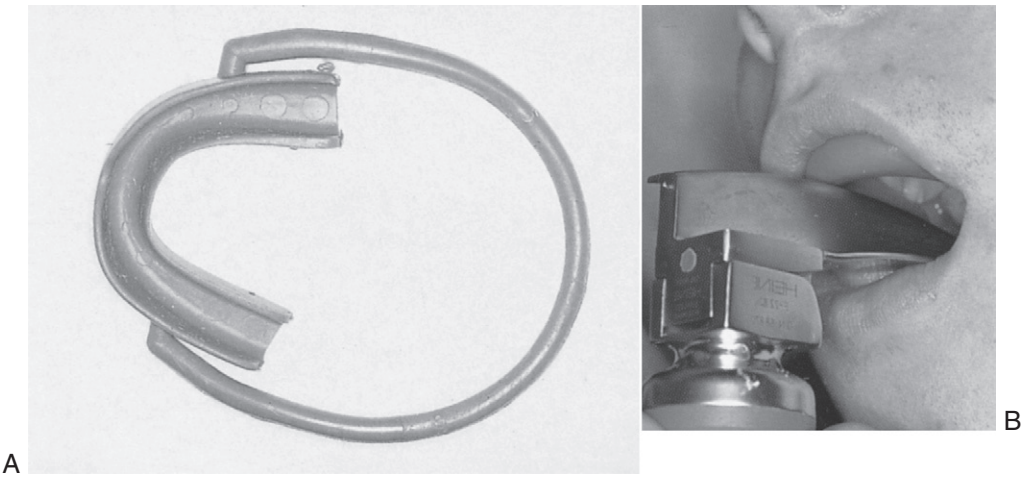


FIGURE 13-5 Trauma to the maxillary central incisors may be associated with endotracheal intubation from a fulcrum effect on the teeth with elevation of the tongue. Many operating rooms have mouthguards, which can be used to protect the teeth during oral intubation. An effective preanesthetic dentoalveolar evaluation and consultation by a dentist are recommended for all patients before oral intubation to minimize the risk of dentoalveolar trauma and injury. (From Andreasen JO, Andreasen FM: *Textbook and color atlas of traumatic injuries to the teeth*, ed 3, Copenhagen, 1994, Munksgaard.)

originally related to participation in contact sports. The incidence of dentoalveolar injury in girls has increased notably as more competitive athletic opportunities have become available. A 2003 study reported that 9% of young adults aged 18 to 19 years who participated regularly in at least one sport had experienced dental injuries during sports participation at some point in their lifetimes.[49] Several studies have shown a proportional relationship between the frequency of dental injuries and the summer months. Injuries to the deciduous teeth involve the supporting bone, because of its resilient nature, whereas injuries in the permanent dentition usually result in a higher percentage of crown and crown-root fractures. Therefore, impact absorption injuries are preponderant among younger children and sharp impact injuries are preponderant among older children.[11-13,24,25,50]

EXAMINATION AND DIAGNOSIS

Injuries to the teeth and supporting structures should be considered an emergency situation because successful management of the injury requires proper diagnosis and treatment within a limited time, especially in the case of avulsions and alveolar fractures.[51]

The initial evaluation must include a general assessment of the patient's overall condition. The patient should be relieved of pain and displaced teeth and alveolar fractures should be reduced as quickly as possible to improve the prognosis for survival of these structures. A complete history of the mechanism and events of the injury should be obtained and a thorough clinical and radiographic examination performed quickly to ensure proper diagnosis and treatment (Fig. 13-6) because teeth have the lowest potential of any tissue for returning to a normal healthy state after injury. An incomplete examination can lead to inaccurate diagnosis and less successful treatment.[52] It is important to remember that patients with dentoalveolar trauma may have concomitant head injury and that continual evaluation must be made of the patient's neurologic status.[53,54]



FIGURE 13-6 A thorough clinical evaluation may be difficult in the pediatric trauma victim. Alterations in occlusion, lacerations of the gingiva, and mobility of teeth are suggestive of additional dentoalveolar injury. General anesthetic or IV sedation may be necessary for complete examination and treatment. (Courtesy Dr. Eric Fort, West Chester, Pa.)

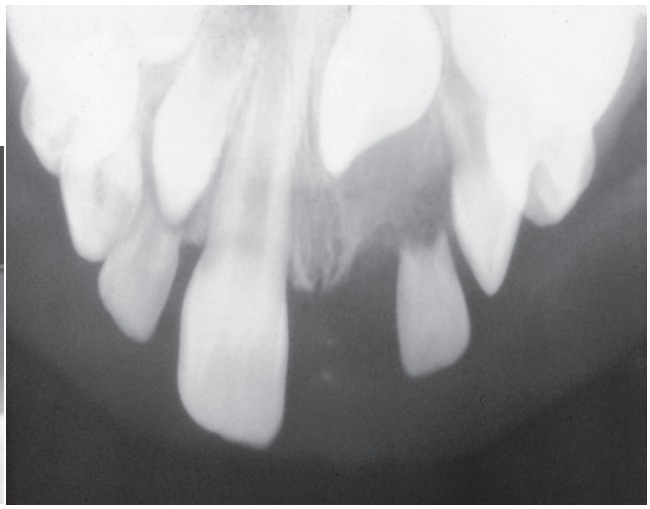
A history can provide valuable information regarding the nature of the injury and any alterations in the normal occlusion, such as open bites or crossbites that were present before the injury. The nature of the accident can provide insight into the type of injury suspected, such as injuries to the maxillary anterior teeth with falls or associated jaw fractures with blows to the chin. If the history of the injury does not correspond to the clinical presentation, abuse should be considered. The patient should be referred for a medical examination.[52,55-57] The time interval between the injury and presentation to the clinic is critical because the success of treating luxated teeth, avulsed teeth, crown fractures with and without pulp exposure, and alveolar fractures may be influenced by delayed treatment. The sooner the injury is treated, the more favorable the prognosis in most situations.[7] Many post-traumatic dentoalveolar complications may also be associated with delays in treatment.[1] Alterations in normal occlusion reported by the patient may indicate displaced teeth, dentoalveolar fractures, jaw fractures, or a combination.

It is prudent that all teeth be accounted for at the time of examination. Extraoral as well as intraoral photographs have become increasingly beneficial in trauma cases. They not only aid in diagnosis and treatment planning, but can serve as further documentation of the trauma. Because trauma can result from abuse, assault, or even medical procedures, pictures can provide further evidence for medicolegal proceedings.[58] Missing teeth or pieces of teeth that have not been left at the scene of the accident must be considered to have been aspirated, swallowed, or displaced into soft tissue of the lip, cheek, floor of the mouth, neck, nasal cavity, or maxillary sinus.[59] A radiographic examination of the head and neck, chest, and abdomen must be performed to rule out the presence of teeth or teeth fragments within these tissues or organs (Figs. 13-7 to 13-11). It is important to note that in patients with maxillofacial trauma, teeth that are unaccounted for and loose may complicate anesthetic procedures.[60] Early diagnosis and surgical removal of theses fragments could prevent undesirable foreign body reaction and scarring. In such cases, the need for taking routine facial soft tissue radiographs and chest x-ray before starting treatment is emphasized.[61] The patient or those who have who transported the patient with avulsed teeth should be questioned regarding the storage media in which the avulsed tooth was transported, as well as how long the tooth has been out of the mouth.

A clinical examination should include an inspection of soft tissue for embedded fragments of tooth or debris. Lacerations, abrasions, and contusions should be examined and evaluated for damage to vital structures, such as the parotid duct, submandibular duct, nerves, and blood vessels. Extraoral wounds may indicate underlying dentoalveolar injuries. A wound under the chin suggests injury to the premolar and molar regions, mandibular fractures, or both, which may be associated with condylar trauma. Patients with suspected condylar trauma should have a full neurologic examination to rule out deficits



A



B

FIGURE 13-7 In cases of suspected tooth avulsion injury, care must be taken to examine adjacent tissue fully. Radiographs must be used to visualize any foreign body and/or dentition that may be lodged in soft tissue and an attempt made to account for any missing teeth. **A,** Trauma to the maxillary anterior teeth from an automobile accident resulted in apparent avulsion of the maxillary left central and left lateral incisors, along with displacement of the right central incisor and gingival lacerations. **B,** This occlusal film reveals an avulsed maxillary right central incisor and possible alveolar injury to the partially resorbed primary right maxillary lateral incisor.

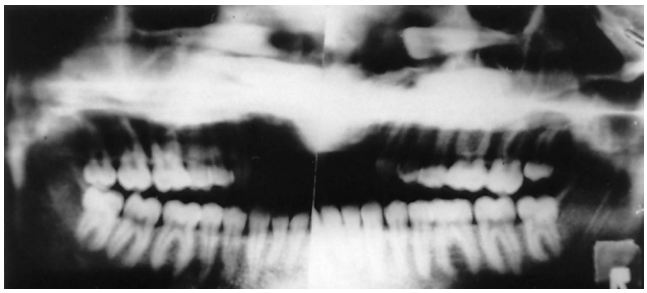


FIGURE 13-8 Panoramic radiograph revealing that teeth had intruded into the soft tissue and nasal cavity. (Courtesy Dr. G. Pedersen, Chardon, Ohio.)

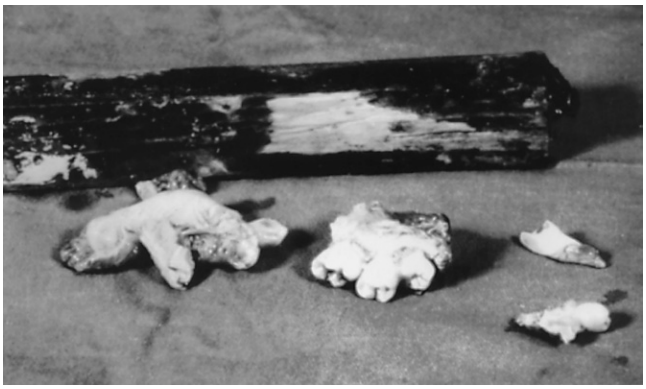


FIGURE 13-9 A young man involved in an MVA suffered multiple avulsion injuries when he was hit by a piece of wood. A thorough clinical and radiographic examination recovered all the segments, but reimplantation was not possible. Two large segments were found at the scene.

and the improbable but reported protrusion of the condyle into the middle cranial fossa.[54] Abnormalities in occlusion can indicate fractures of the jaws, dentoalveolar structures, or displacement of teeth. The direction of tooth displacement should be noted. In the primary dentition, the dislocation of the apex of the displaced primary tooth can possibly damage the succedaneous tooth. The direction of the dislocation and extent should be recorded (in millimeters). Fracture of the alveolar socket is common in displaced teeth. All teeth should be tested for abnormal mobility, horizontally and axially, which would suggest displacement of teeth or alveolar or jaw fractures. Disruption of the vascular supply to the pulp should be expected when axial mobility is present.[7] Tongue blades can be found in emergency rooms to assess tooth mobility. Erupting teeth may exhibit some mobility that is unrelated to the injury. Movement of several teeth en bloc during the evaluation of a single tooth suggests fracture of the alveolar process. Uneven contours of the alveolar process may indicate a bony fracture.[52,56]

All the crowns of the teeth should be cleansed of blood and debris before evaluation for fractures of the crown and exposure of the pulp (Fig. 13-12*A*). Bleeding may be from lacerated mucosal tissue and nonlacerated marginal gingiva with damage to the periodontal ligament. Blood covering the alveolar process must be removed and the teeth and tissue evaluated for possible fracture, damaged teeth, and damaged periodontal tissue. The color of the traumatized tooth should be noted with the use of digital photographs or transillumination techniques because color changes may occur over time, indicating alterations in tooth vitality and representing gradual pulp necrosis.[60-62] The involved teeth should be tapped or percussed with the handle of a mouth mirror or with a calibrated percussion instrument (e.g., Periotest, Medizintechnik Gulden, Modautal,

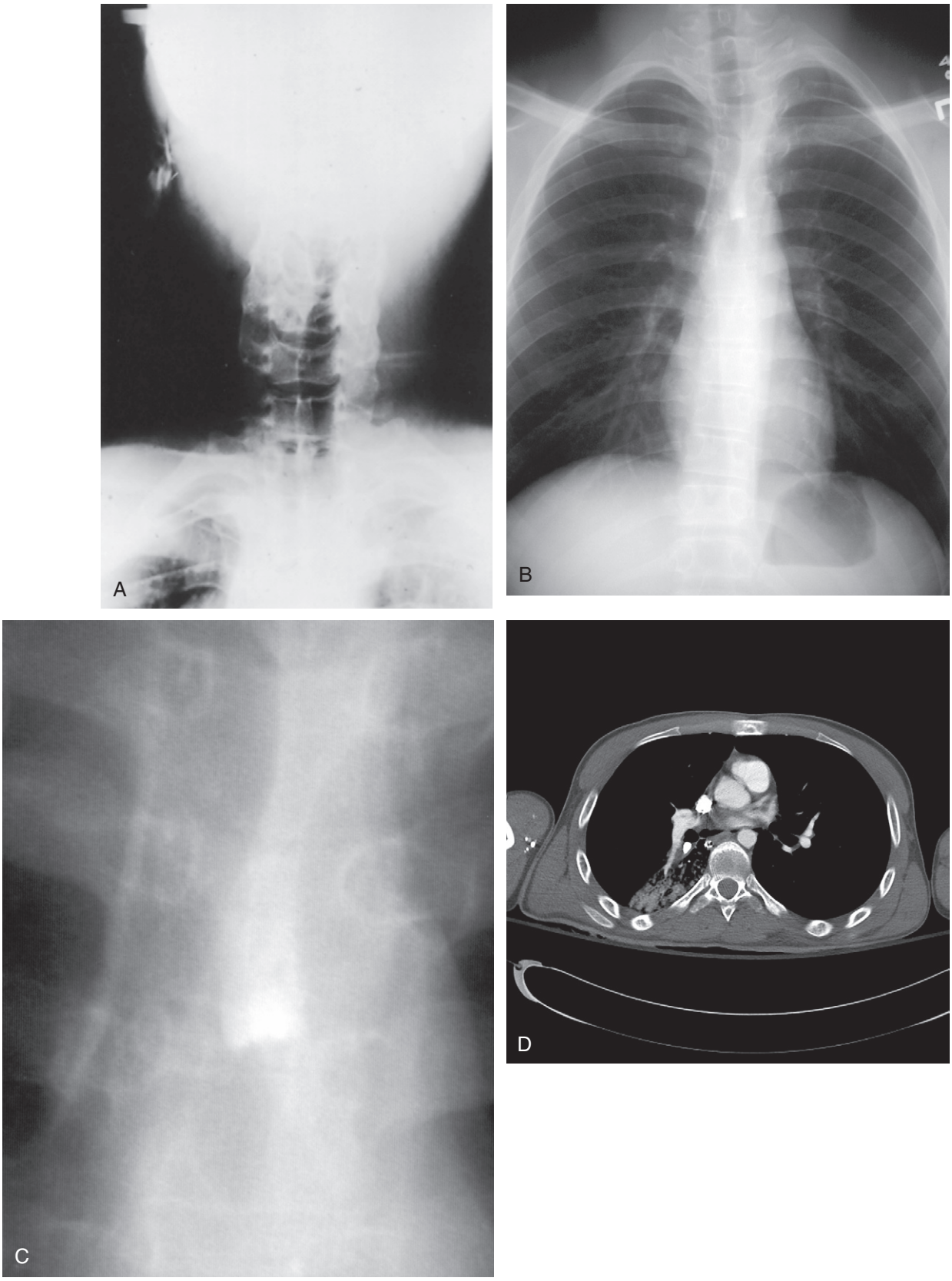


FIGURE 13-10 Radiographs taken after a traumatic incident to locate missing teeth. **A,** An anteroposterior (AP) head and neck radiograph located a canine tooth that was pushed across the oral cavity and into the soft tissue of the lateral neck. **B,** A molar can be seen in the midline within the esophagus in this AP chest radiograph. The patient evidently swallowed the tooth following a traumatic avulsion of the tooth in an automobile accident. **C,** The patient is clinically followed without any invasive intervention to remove the tooth because the radiograph demonstrates that the lost molar was within the esophagus and should pass without difficulty through the gastrointestinal structures. **D,** Axial CT image of an avulsed tooth in right lower lobe of the lung after a traumatic incident.

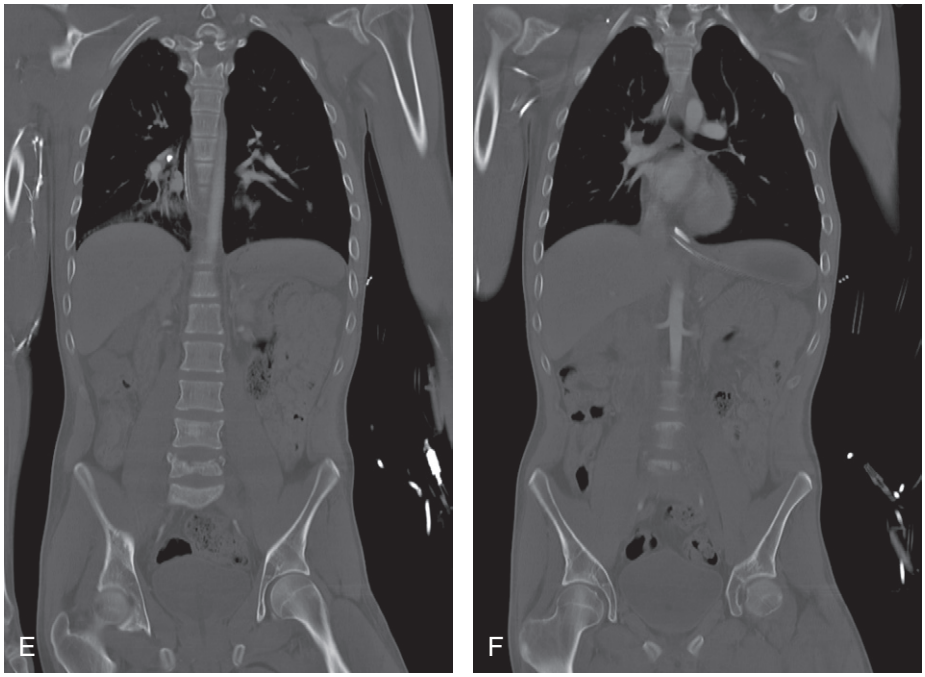


FIGURE 13-10, cont'd **E,** Coronal CT image of the tooth in the right lower lobe. **F,** Coronal CT image of the same patient with a tooth also in the distal esophagus. (**A-C,** Courtesy Dr. B. Zargari, New York.)

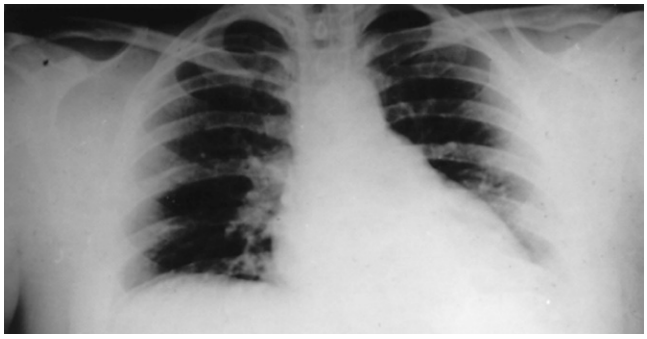


FIGURE 13-11 The premolar in this patient was located on a chest radiograph in the right mainstem bronchus. Teeth are frequently aspirated during the traumatic episode and are usually located in this position. The tooth was removed by bronchoscopy before repair of the facial injury.

Germany) to evaluate damage to the periodontal ligament. Pain elicited with percussion is suggestive of injury to the periodontal ligament (see Fig. 13-12*B*). Teeth that have undergone lateral luxation and intrusion usually do not demonstrate tenderness to percussion because these teeth are normally locked into their displaced position. The sound elicited by percussion is also of diagnostic value. A sound resembling a hard metallic ring is elicited with teeth that are locked into bone, whereas a dull sound indicates a subluxated tooth.[52]

Testing pulp vitality in acutely traumatized teeth is of questionable value because these procedures require the cooperation of a relaxed patient to prevent false-negative reactions. Mechanical stimulation and electric pulp testing have been advocated for examining the vitality and sensory perception of the dental pulp. Thermal tests, such as with heated gutta-percha, ice, ethyl chloride, carbon dioxide, and dichlorodifluoromethane, are usually of little value until several months have passed. Also, the reliability and the value of these tests have been questioned because noninjured teeth may also fail to respond. Although the functional repair of pulp nerve fibers has been shown to be reestablished approximately 35 days after reimplantation, the electric pulp tester is of little or no value in teeth with incompletely formed apices.[63] The interpretation of vitality tests performed immediately after traumatic injuries is complicated by the fact that sensitivity responses are temporarily decreased, especially after luxation injuries. Repeated vitality tests show that normal reactions can return after a few weeks or months.[7,62] Teeth that have been loosened can elicit pain responses merely from pressure of the pulp testing instrument; therefore, it is important to immobilize these teeth before testing. Teeth at different stages of eruption may sometimes show no reaction to stimulation. A 2007 study has shown that recently traumatized teeth yield positive responsiveness in thermal or electric pulp tests, which increased from no teeth showing responsiveness on day 0 (day of trauma) to 29.4% teeth on day 28, 82.35% of teeth at 2 months, and 94.11% teeth at 3 months.[63] However, vitality readings with a pulse oximeter have yielded positive vitality readings that remained constant over the study period, from day 0 to 6 months in all patients. This is because blood flow in the tooth was being measured rather than thermal or electrical response in the tooth, which indicates the functioning of A delta nerve fibers.[64,65]

Another new method that has been developed to measure pulp vitality by measuring blood flow in the pulp is laser Doppler flowmetry (LDF). This noninvasive technique measures blood flow in the pulp and has been

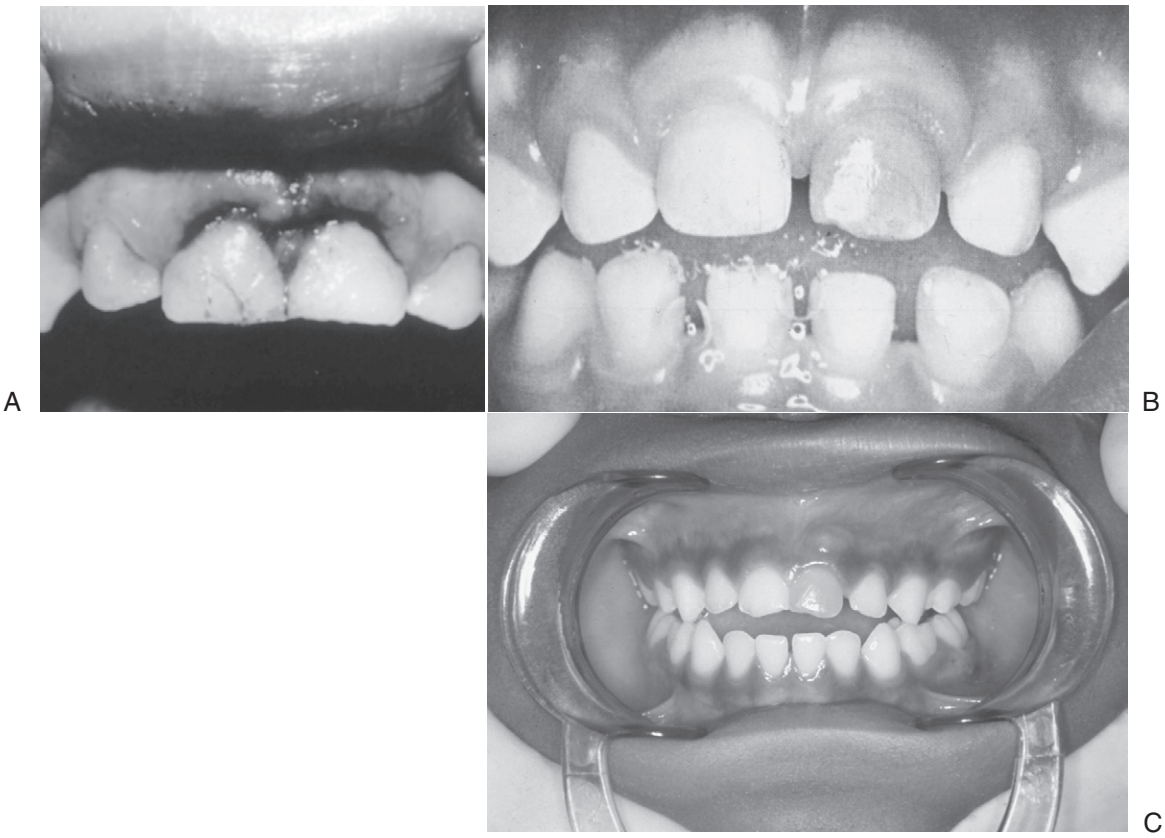


FIGURE 13-12 **A,** The crowns of the teeth should be cleansed of blood and debris for evaluation of crown fractures and possible pulp exposures. **B,** This central maxillary incisor suffered direct trauma, with no obvious injury noted immediately after the fall from a bicycle. The tooth crown color darkened over the next 24 hours as a result of bleeding from the pulp chamber into the open dentinal tubules. The gingival tissue became swollen and erythematous as a result of secondary damage to the periodontal ligament with the fall. **C,** The discoloration of the crown of the maxillary left central incisor developed several months following trauma to a primary tooth. The discoloration of the primary tooth did not require treatment and, because there had been no damage to the underlying permanent tooth or alveolar bone, the tooth was retained until it was replaced by the eruption of the permanent central incisor. (**B** and **C,** Courtesy Dr. Seth Canion, Case Western Reserve University, School of Dental Medicine, Department of Pediatric Dentistry, Cleveland.)

used in studying traumatized and luxated teeth to differentiate between those that are vital and those that are nonvital. Because this test produces no noxious stimuli, apprehensive or distressed patients accept it more readily than current methods of pulp vitality assessment.[66,67] There has been some promise with this technique, especially in the case of deferred treatment. Studies have shown that vitality of a traumatized tooth can be ascertained in the first month with LDF and other modalities—ethylene chloride, CO_2, or ice—do not elicit a clinical response until the 6-month follow-up.[62,68] This may prevent the need for early invasive techniques that would otherwise be carried out if LDF were not used. However, this technique has been limited in its diagnostic value because measurements are difficult to evaluate through the crown of a tooth discolored by blood pigments and LDF relies on measurement of light transmission to function. The high cost of an LDF unit is also a limiting factor in its use.[60,69]

Radiographic evaluation of dentoalveolar injuries should include at a minimum a panoramic radiograph and periapical radiographs of the involved teeth. Computed tomography (CT) and cone beam CT (CBCT) provide more detail than panoramic and periapical radiographs and are helpful in diagnosing fractures of the teeth, roots, and supporting bone. Patients who are evaluated in the hospital setting will often have CT performed if there is evidence of trauma to the head and maxillofacial structures. These images can be helpful in diagnosis and treatment planning for further reconstruction after dentoalveolar injury. Use of CBCT in the oral and maxillofacial surgery office is becoming more prevalent and should be considered for dentoalveolar trauma patients. Valuable information for implant therapy can be obtained by CT and CBCT images regarding bone height and width and location of anatomic structures.[70-72]

The radiographic examination reveals the stage of root formation and discloses injuries affecting the root portion of the tooth and periodontal structures. Multiple periapical radiographs taken at different angles are useful to demonstrate root fractures that are minimally displaced. Ideally, three different angles should be obtained for each traumatized tooth.[52] The radiographic examination should provide information concerning the following[73]:

- Presence of root fractures
- Degree of extrusion or intrusion
- Presence of preexisting periodontal disease



FIGURE 13-13 **A,** A fracture of the mandible was noted on this periapical radiograph through the second premolar. Note the widened periodontal ligament (PDL) associated with the second premolar. **B,** Two fractures of the mandible, a right parasymphyseal fracture associated with the right mandibular first premolar and a left mandibular angle fracture associated with the impacted mandibular third molar, are demonstrated in this panoramic radiograph. There is PDL involvement with tooth 17 and a noticeable change in the occlusal plane involving the right mandibular premolars, as shown in the clinical picture. **C,** With the mandibular parasymphyseal fracture, an obvious step in the occlusion is noted between the mandibular premolars and extravasation of blood into the adjacent tissue. **D,** The lateral oblique film of the left mandibular angle fracture further demonstrates the fracture of the mandible through the angle, with a communication involving an impacted mandibular third molar.

- Extent of root development
- Size of the pulp chamber and root canal
- Presence of jaw fractures
- Tooth fragments and foreign bodies lodged in soft tissue
- Presence of dental caries

The radiographic appearance of teeth following luxation injuries is important in assessing pulp survival or necrosis. Teeth that have been displaced laterally or extruded radiographically exhibit a widening of the periodontal ligament space or displacement of the lamina dura, whereas intruded teeth often demonstrate an absence of the periodontal ligament space. Fracture of the alveolar process may appear radiographically as a small radiolucent line, which in some cases is blurred on the panoramic radiograph but detectable on periapical radiographs (Fig. 13-13).

Foreign bodies within the soft tissue of the floor of the mouth, lips, or cheeks can be viewed radiographically by placing the film between the soft tissue and alveolus. The floor of the mouth can best be visualized by an occlusal film, with the beam directed from a submental approach. The exposure time for radiographs of soft tissue should be reduced by approximately 25% to 50% of the normal exposure time; the use of low kilovoltage is advocated for these exposures.

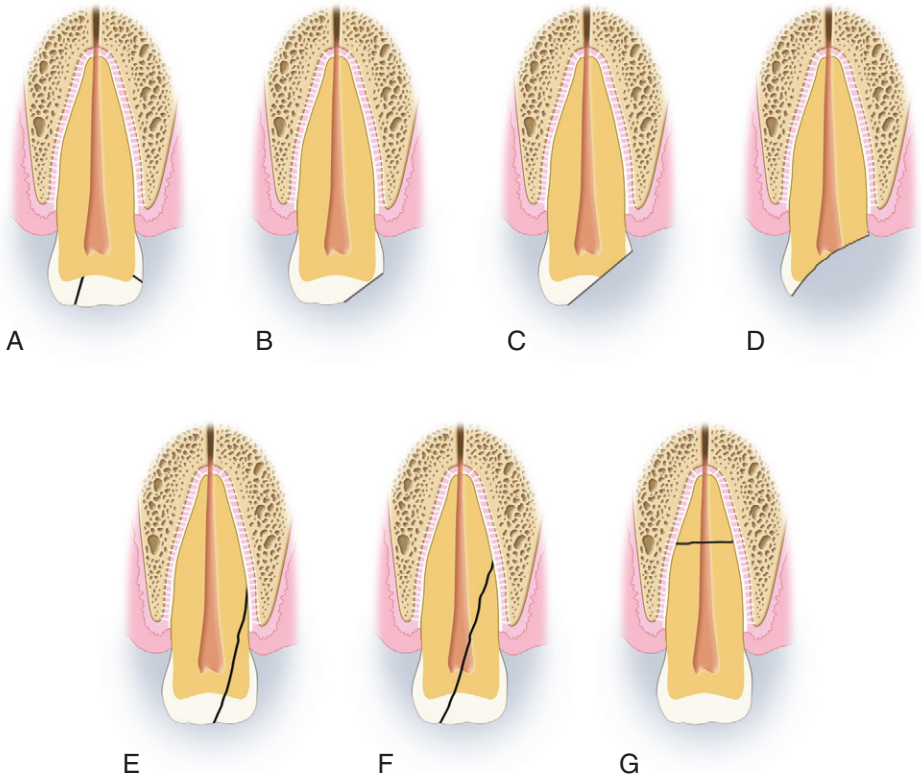


FIGURE 13-14 Injuries to the hard dental tissue and pulp tissue. **A,** Crown infraction. **B**, **C,** Uncomplicated crown fracture. **D,** Complicated crown fracture. **E,** Uncomplicated crown-root fracture. **F,** Complicated crown-root fracture. **G,** Root fracture.

CLASSIFICATION

Many systems have been developed to classify various traumatic injuries to the teeth and supporting structures.[21,74-78] The present classification is based on a system presented by the World Health Organization[26] and modified by Andreasen.[7] It includes injuries to the teeth, supporting structures, gingiva, and oral mucosa based on anatomic, therapeutic, and prognostic considerations.

INJURIES TO HARD DENTAL TISSUE AND PULP

Crown Infraction. A crown infraction (Fig. 13-14*A*) is an incomplete fracture or crack of the enamel without loss of tooth substance.

Uncomplicated Crown Fracture. An uncomplicated crown fracture (see Fig. 13-14*B* and *C*) is a fracture confined to the enamel or involving the enamel and dentin without exposing the pulp.

Complicated Crown Fracture. A complicated crown fracture (see Fig. 13-14*D*) involves enamel and dentin with exposure of the pulp.

Uncomplicated Crown-Root Fracture. An uncomplicated crown-root fracture (see Fig. 13-14*E*) involves enamel, dentin, and cementum without exposure of the pulp.

Complicated Crown-Root Fracture. A complicated crown-root fracture (see Fig. 13-14*F*) is a fracture involving enamel, dentin, and cementum with exposure of the pulp.

Root Fracture. A root fracture (see Fig. 13-14*G*) involves dentin, cementum, and the pulp.

INJURIES TO THE PERIODONTAL TISSUE

Concussion. A concussion (Fig. 13-15*A*) is an injury to the tooth-supporting structures without abnormal loosening or displacement of the tooth but with marked reaction to percussion.

Subluxation (Loosening). Subluxation (see Fig. 13-15*B*) is an injury to the tooth-supporting structures with abnormal loosening but without displacement of the tooth.

Intrusive Luxation (Central Dislocation). Intrusive luxation (see Fig. 13-15*C*) is displacement of the tooth into the alveolar bone with comminution or fracture of the alveolar socket.

Extrusive Luxation (Peripheral Dislocation, Partial Avulsion). An extrusive luxation (see Fig. 13-15*D*) is partial displacement of the tooth out of the alveolar socket.

Lateral Luxation. A lateral luxation (see Fig. 13-15*E* and *F*) is displacement of the tooth in a direction other than axially, accompanied by a comminution or fracture of the alveolar socket.

Retained Root Fracture. A retained root fracture (see Fig. 13-15*G*) is a fracture with retention of the root segment but loss of the crown segment out of the socket.

Exarticulation (Complete Avulsion). An exarticulation (see Fig. 13-15*H*) is a complete displacement of a tooth out of the alveolar socket.

INJURIES TO THE SUPPORTING BONE

Comminution of the Alveolar Socket. Crushing and comminution of the alveolar socket (Fig. 13-16*A*) can occur together with intrusive and lateral luxation.

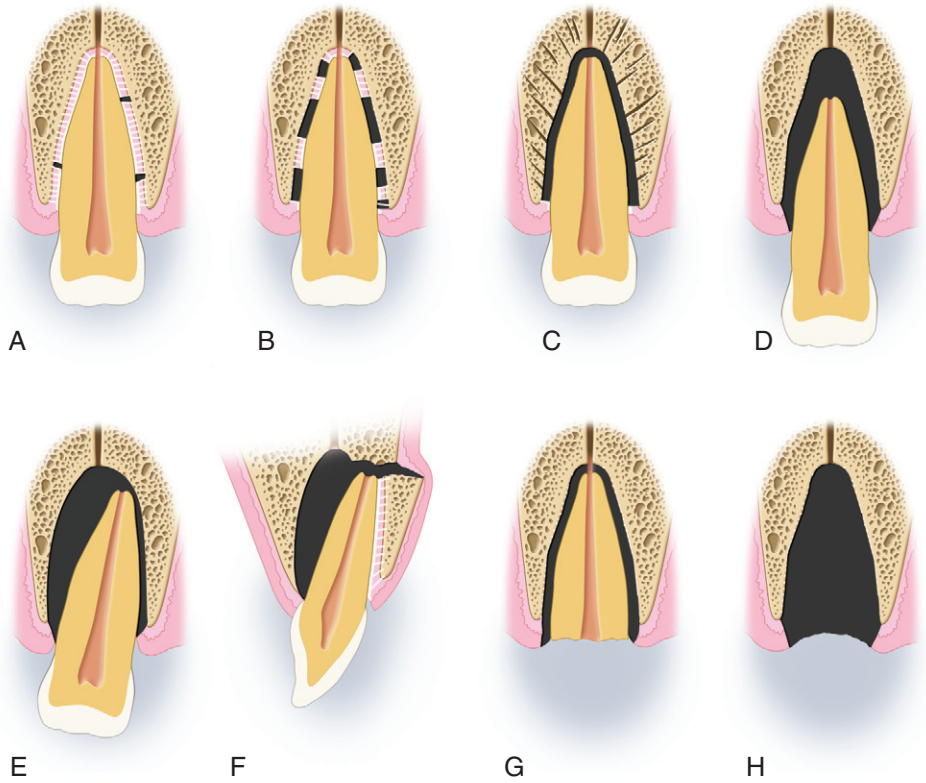


FIGURE 13-15 Injuries to the periodontal tissue. **A,** Concussion. **B,** Subluxation. **C,** Intrusive luxation. **D,** Extrusive luxation. **E**, **F,** Lateral luxation. **G,** Retained root-crown fracture. **H,** Exarticulation.

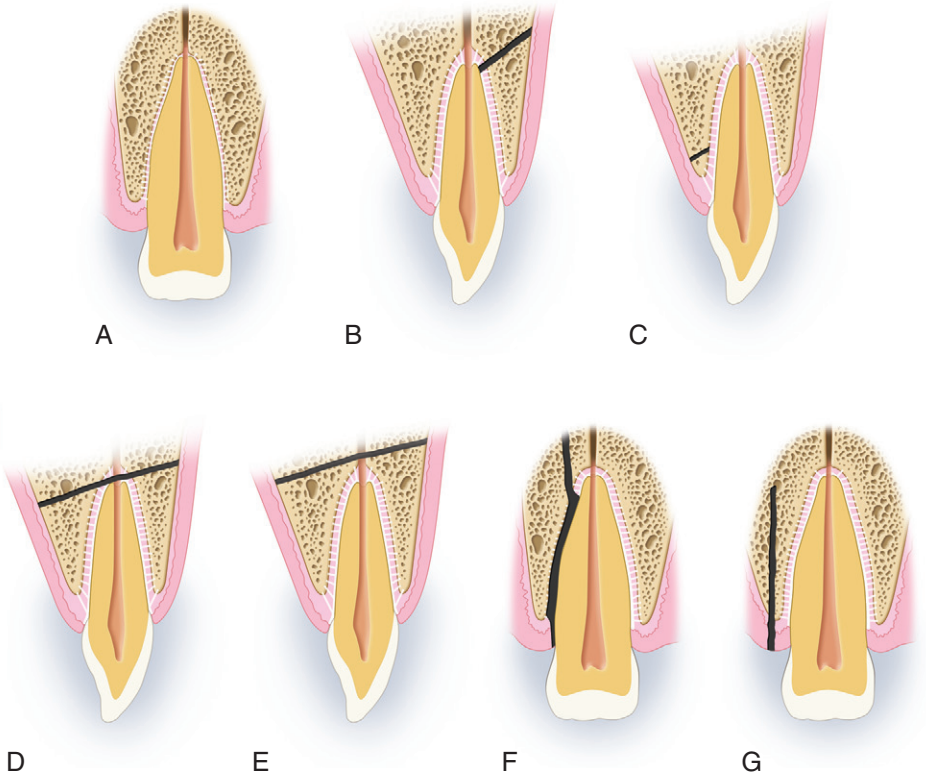


FIGURE 13-16 Injuries to the supporting bone. **A,** Comminution of the alveolar socket. **B**, **C,** Fracture of the alveolar socket wall. **D, E,** Fracture of the alveolar process. **F, G,** Fracture of the mandible and maxilla.

Fracture of the Alveolar Socket Wall. A fracture of the alveolar socket (see Fig. 13-16*B* and *C*) is confined to the facial or lingual socket wall.

Fracture of the Alveolar Process. A fracture of the alveolar process (see Fig. 13-16*D* and *E*) may or may not involve the alveolar socket.

Fractures of the Mandible or Maxilla. A fracture involving the base of the mandible or maxilla (see Fig. 13-16*F* and *G*), and often the alveolar process, may or may not involve the alveolar socket.

INJURIES TO THE GINGIVA OR ORAL MUCOSA

Laceration of Gingiva or Oral Mucosa. A shallow or deep wound in the mucosa results from a tear and is usually produced by a sharp object.

Contusion of Gingiva or Mucosa. A bruise is usually produced by impact from a blunt object and results in submucosal hemorrhage without a break in the mucosa.

Abrasion of Gingiva or Oral Mucosa. A superficial wound produced by rubbing or scraping of the mucosa, leaving a raw, bleeding surface, constitutes an abrasion of the gingiva or oral mucosa.

TREATMENT

After obtaining a thorough history and performing a clinical and radiographic examination, several factors should be considered in the definitive treatment of the dentoalveolar injury:

1. Age of the patient
2. His or her cooperation
3. Injury to the primary or permanent dentition and stage of root development
4. Location and extent of the injury—horizontal and proximal superficial (corner) fractures demonstrate a low frequency of pulp necrosis, whereas deep proximal fractures show an increased risk of eventual pulp necrosis.
5. Residual bone support
6. Periodontal health of the remaining teeth
7. Whether or not there has been a fracture of supporting bone
8. Vitality of the teeth
9. Whether apical foramina are wide or narrow
10. Injury to soft tissue
11. Extent of any concomitant head, chest, or abdominal injuries may affect the treatment of the dentoalveolar injury and length of time between trauma and treatment.[11,79]

Also, the practitioner should determine the amount of time that has passed since the injury occurred and what storage medium, if any, was used.[80,81] Lack of cooperation on the part of the patient, especially young children and mentally challenged individuals, may require the use of a general anesthetic or intravenous sedation.

The periodontal health of the involved and adjoining teeth must be evaluated for osseous support of the traumatized teeth and to determine whether the adjoining teeth can be used for splinting, if indicated. The prognosis for traumatized teeth is generally better in younger patients and in vital teeth with wide apical foramina, teeth with intact soft tissue, teeth with no root fractures, and teeth with maximal bone support.[82] Any blow to a tooth, even in the absence of obvious dental injury, can endanger pulp vitality by severing the apical vessels or through secondary pulp hyperemia and congestion, resulting in ischemia and possibly leading to necrosis. Therefore, long-term dental follow-up is necessary. The typical follow-up schedule should include a clinical visit within 24 to 48 hours after the initial clinical evaluation and treatment, followed by a clinical visit within 2 weeks for removal of the splint. Monthly clinical visits are planned for the first 6 months and should be followed up by annual visits for 5 years.[3,83,84] The patient should be informed that future endodontic therapy may be necessary, regardless of the severity of injury to the teeth and supporting structures (Fig. 13-17).[7,85-89]

The treatment of traumatized primary incisors is complicated by the size of the pulp cavity, susceptibility of the developing permanent tooth, and cooperation of the child. It is also important to note than any type of traumatic injury to a primary tooth could result in potential sequelae to the permanent tooth. Concerning the permanent dentition, the most common developmental disturbances are discoloration of enamel and/or enamel hypoplasia (46.08%) and eruption disturbances (17.97%) caused by the traumatic injury in their predecessors. It has not been possible to find an association between the type of injury in primary teeth and sequelae in their successors.[90] Heroic methods designed to maintain the primary incisors after trauma should be discouraged. The loss of primary incisors does not require space maintenance as growth occurs, regardless of which primary incisors are involved, but loss of primary molars may require space maintenance to prevent mesial drift of the permanent first molar.[7,9,85,91] If the primary incisor is lost before the roots begin to be absorbed (between the ages of 3 and 4 years), eruption of the succedaneous permanent tooth is often delayed. However, this delay usually does not create additional orthodontic problems. If the primary tooth is lost after 25% of the permanent incisor root has formed (between the ages of 4 and 5 years), eruption of the permanent successor is often accelerated.

INJURIES TO HARD DENTAL TISSUE AND PULP

In the permanent dentition, crown fractures are associated with most dental injuries. The most common type of injury involves enamel and dentin (45%) with crown fractures. The treatment of crown fractures in the permanent dentition must initially relieve the pulp's response to the injury before the final necessary restorations are considered.

Crown Infractions. Infractions are often overlooked. It is important that direct transillumination (directing a light beam perpendicular to the long axis of the tooth from the incisal edge) be performed for adequate evaluation and treatment.[92] Infractions predominantly result from a direct impact to the enamel, frequently occurring on the labial surface of the maxillary incisors. Usually no treatment is indicated for a crown infraction or cracks in the enamel layer (see Fig. 13-14*A*). Sealing multiple infraction lines with unfilled resin and an acid etch technique may prevent stains from becoming an aesthetic

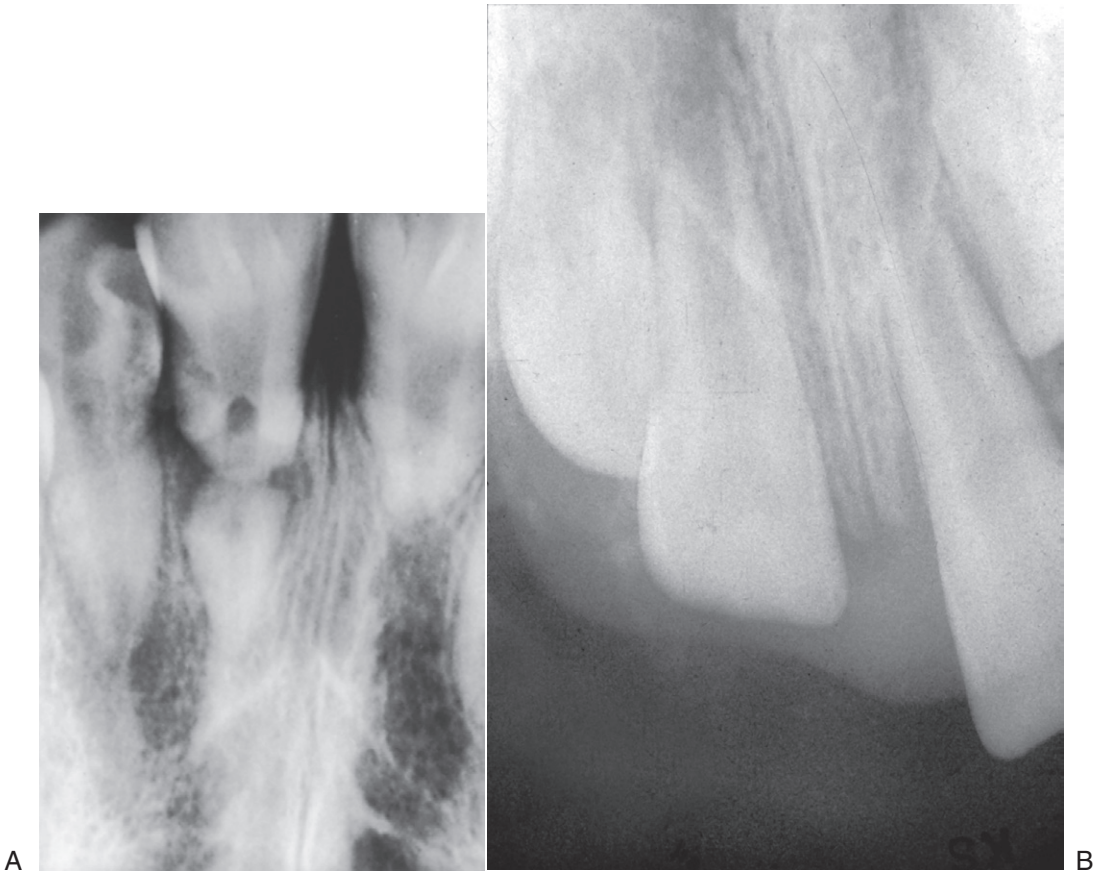


FIGURE 13-17 Root resorption is a major common complication associated with dentoalveolar trauma, which if left untreated will result in loss of the tooth. Resorption resulting from toxic byproducts and bacteria from the necrotic pulp tissue may be seen months to years after trauma to the tooth. **A,** Note the various degrees of resorption associated with the maxillary incisors—early external resorption of the lateral incisor, resorption around a middle third root fracture of the central incisor, and total resorption of the apical root in the other central incisor. **B,** Root resorption of a maxillary central incisor is demonstrated in this occlusal radiograph with a patient who was asymptomatic, without complaints and noted during an initial screening examination. (**A,** Courtesy Dr. J.A. Wallace, Fox Chapel, Pa; **B,** courtesy Dr. Seth Canion, Case Western Reserve University, School of Dental Medicine, Department of Pediatric Dentistry, Cleveland.)

problem. The vitality of the traumatized tooth should be documented at the time of diagnosis and evaluated adequately by periodic follow-up to monitor the pulpal health of the involved tooth. If the tooth does not respond to pulp vitality testing at the time of injury, endodontic therapy is not indicated immediately, but the tooth must be kept under observation. The patient should be informed of signs and symptoms that may occur, such as pain, swelling, and tooth discoloration. If the patient experiences any of these, further evaluation and potential treatment may be necessary. The prognosis with respect to pulp necrosis is good, provided that concussion or subluxation injuries are not overlooked.[11]

Crown Fractures. For crown fractures that involve the enamel only (see Fig. 13-14*B*), treatment is limited to smoothing of sharp edges or restoration with acid etch composite (Fig. 13-18*A*). Because of aesthetic demands, midline symmetry may also be recovered with orthodontic extrusion of the tooth to restore properly balanced incisal height. The treatment objectives are to restore the crown's anatomy and occlusion, thus preventing labial protrusion, drifting, tilting, or extrusion of adjacent and opposing teeth into the fracture site (see Fig. 13-18*B* and *C*). The risk of pulp necrosis is minimal.

Fractures without exposure of the pulp from oral trauma occur more frequently than any other type of crown fracture in the primary and permanent dentitions. Exposed dentin usually is sensitive to thermal changes and mastication. If there is a notable amount of dentin exposed, measures should be taken to do the following: (1) seal the dentin tubules from microbial irritants to prevent ingress of bacteria; and (2) promote secondary dentin deposition by the pulp tissue (Fig. 13-19; also see Fig. 13-18*D*). A calcium hydroxide liner is placed over the exposed dentin and the tooth form is restored with a composite restoration. This traditional technique has been well studied, but current evidence suggests that because calcium hydroxide is soluble in water, the dentinal tubule fluid on surfaces of the exposed fractured site interferes with the calcium hydroxide material, resulting in its dissolution and loss of protective function over time. An alternative treatment uses glass ionomer cement as a liner, which has proven to be as effective.[92] Reattachment of the



FIGURE 13-18 **A,** Uncomplicated crown fracture of a maxillary central incisor that is confined to enamel only. **B**, **C,** Crown fracture limited to enamel. Treatment is limited to smoothing and a direct composite restoration. The aesthetic direct restoration is shown to demonstrate the corrected anatomy and occlusion. **D,** Uncomplicated crown fractures that involve dentin require treatment of the exposed dentin to manage the possible symptoms associated with thermal change and oral fluid exposure. A dentin-bonding agent was used before application in the restorative material.

fractured segment using dentin-bonding agents can be performed to restore the tooth and reduce the microleakage around the restoration.[11] There is no change in the bond strength with the use of the dentin-bonding agents compared with standard enamel bonding techniques.[93] No matter which technique is used, the tooth should be evaluated periodically for alterations in pulpal health. The overall risk for pulp necrosis is minimal, with associated luxation injuries, stage of root development, type of treatment, and extent of fracture increasing the risk of pulp necrosis.

If the pulp tissue is exposed (see Fig. 13-14*C*), measures must be taken quickly to attempt to preserve the vitality of the neurovascular tissue because hemorrhage from the exposed pulp is usually present. Teeth with pulp exposure require pulp capping, partial pulpotomy, or endodontic therapy. The primary aim is to preserve a vital noninflamed pulp surrounded by hard tissue. Whichever treatment option is used, careful periodic evaluation is necessary, including clinical examination, vitality testing, and periapical radiographs. If the tooth is relatively sound, a pulp-capping procedure with calcium hydroxide liner or mineral trioxide aggregate (MTA) and composite restoration should be performed. MTA is a bioactive material that can create an ideal environment for healing. From the time that MTA is placed into direct contact with human tissue, it appears that the material does the following:

1. Forms calcium hydroxide (CH) that releases calcium ions for cell attachment and proliferation
2. Creates an antibacterial environment by its alkaline pH
3. Modulates cytokine production
4. Encourages differentiation and migration of hard tissue–producing cells and
5. Forms hydroxyapatite (HA, or carbonated apatite) on the MTA surface and provides a biologic seal

To achieve the best prognosis, the pulp-capping procedure should be carried out only in teeth with small exposures and those that appear within 24 hours after injury (Fig. 13-20*A* and *B*). Pitt-Ford and Patel[65] have compared CH and MTA as pulp-capping agents on monkeys' teeth. Their results showed that most pulps capped with MTA were free of inflammation and all of them showed calcified bridge formation after 5 months. In contrast, the pulp of teeth capped with CH showed the presence of inflammation and significantly less calcified bridge formation. MTA promotes dentin bridge formation through differentiation of pulpal cells into odontoblastic-like cells.[94-96] MTA has grown in favor with clinicians because of its biocompatibility and ability to help maintain pulp vitality.



FIGURE 13-19 Presentation of a patient following a fall with an uncomplicated crown fracture of the permanent maxillary central incisor that had a considerable amount of exposed dentin. The tooth did not require root canal therapy and was treated conservatively with an acid-etched composite resin restoration and follow-up evaluations.

Pulp capping is also indicated in complicated crown fractures in immature teeth, in which maintenance of pulp vitality is desirable. Most complicated fractures eventually require partial or complete pulpotomy; the latter procedure should be considered a temporary treatment and should be followed by pulpectomy when root development is complete.[92] A cervical pulpotomy involves removing 2 to 4 mm of pulp tissue subadjacent to the exposure (i.e., all the coronal pulp), placing a CH or MTA base, and using an acid etch composite for restoration.

A cervical pulpotomy is indicated in cases involving immature teeth with incomplete closure of the apex because it is not indicated for teeth with concurrent displacement injuries. CH paste or MTA has been demonstrated to promote closure of the apex of the immature tooth.[7] There have been multiple studies confirming MTA's ability to help promote apex closure and, again, may be a superior material to CH.[94]

Completion of root canal therapy is usually indicated by closure of the apex. This closure can be confirmed by serial radiographs, usually within 3 to 6 months. Partial, or Cvek, pulpotomy implies the removal of pulp tissue to only a depth of 1 to 2 mm. Partial pulpotomy provides many advantages when compared with cervical pulpotomy because there is no necessity for subsequent endodontic therapy and the cell-rich coronal tissue is preserved (see Fig. 13-20*C*).[97]

If the teeth are mature and fully formed, with notable exposure of the pulp tissue, total pulp removal is indicated (Fig. 13-21). The canals should be completely treated endodontically and filled with gutta-percha to prevent post-traumatic complications associated with pulp necrosis. Follow-up examinations are necessary to evaluate all traumatized teeth (with pulp exposure treated conservatively for degenerative pulp necrosis) and to determine whether definitive root canal therapy is necessary. Definitive restoration of crown fractures consists of composite restorations, reattachment of original crown fragments, full crown coverage, or laminate veneers.

Crown-Root Fractures. Crown-root fractures (see Fig. 13-14*E* and *F*) account for 5% of traumatic injuries to the permanent teeth and 2% of injuries to the primary dentition. The treatment options are dependent on the amount of root remaining. Treatment goals are to preserve the remaining root fragment to support a post and crown prosthesis, which requires additional periodontal procedures, such as gingival-alveolar surgery or orthodontic measures to expose the retained root segment.

Primary teeth with any type of crown-root fracture should be extracted and conventional prosthetic-restorative treatments provided.[7,85,98] Emergency treatment should include stabilization of the coronal fragment to adjacent teeth with an acid etch resin splint.[99] In the permanent dentition, uncomplicated fractures of the enamel, dentin, and cementum depend on the location of the fractured segment, which determines the type and extent of treatment. If the fracture line is above or slightly below the cervical margin, the tooth can be restored, as with a crown fracture. Occasionally, crown-lengthening procedures are necessary to allow restoration of the margin. If the fracture continues too apically to allow adequate restoration, extraction of the tooth may be indicated. If the pulp is involved (complicated), the level of the root fracture determines the treatment. Extraction is usually indicated if the coronal segment includes more than one third of the clinical root or in cases of vertical root fractures; otherwise, the tooth may be treated endodontically and restored. When the coronal fragment compromises one third or less of the clinical root and the fracture line extends below the marginal bone level, adequate access to provide margins on sound tooth structure is necessary. This can be accomplished by extrusion, by surgical exposure (gingivectomy or osteotomy) or orthodontic elevation of the fracture surface, or by crown-lengthening procedures.[92] Surgical procedures are indicated only in teeth with completed root development and an apical fragment long enough for definitive restorative procedures.[99] The stability of orthodontic movements has been evaluated and it was concluded that the risk of relapse is always present but can be minimized by fibrotomy techniques.[100] If the tooth is mature and root formation is complete, endodontic treatment is indicated. If the apex is open, a pulpotomy should be performed with a CH or MTA base and should be observed through the use of x-rays for closure of the apex, after which definitive root canal therapy and permanent restoration can be initiated. A crown-root fracture of a tooth in a young person may require extraction and placement of a space-maintaining partial denture. Alternative treatments have been presented in the literature, suggesting the retention of the root fragment by the procedures described until the completion of the pubertal growth spurt to preserve the alveolar bone. After that time, definitive treatment can be accomplished with placement of endosseous implants.[98]

Root Fractures. Root fractures (see Fig. 13-14*G*) account for approximately 6% of cases of dental trauma.[101] Root fractures account for approximately 7.7% of the trauma associated with the permanent dentition and

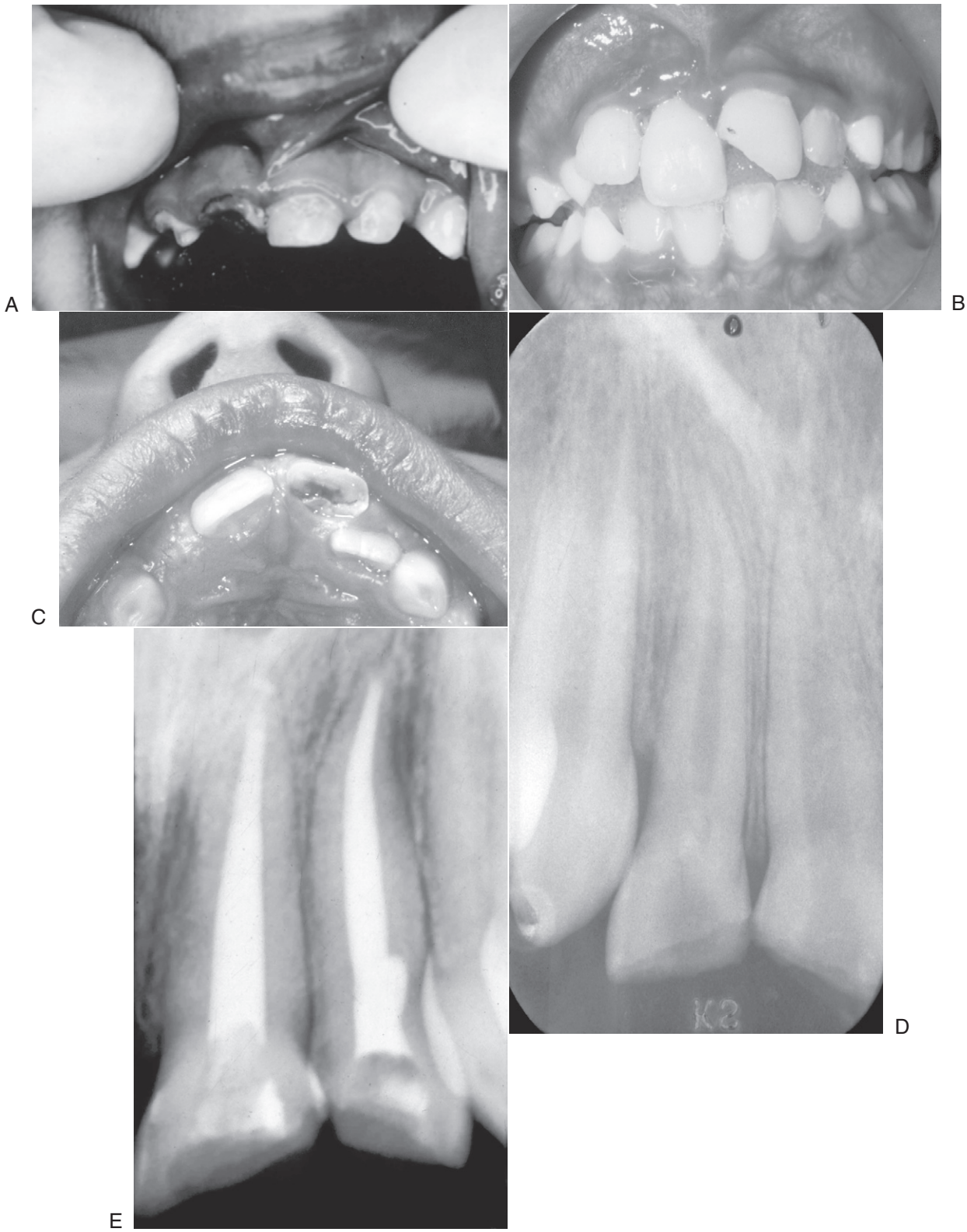


FIGURE 13-20 **A,** Significant crown fracture involving pulp tissue in primary dentition. **B,** Teeth that have exposure of pulp tissue will require pulp capping, partial pulpotomy, or endodontic therapy. Ideally, pulp-capping treatment should be initiated within an acute period of less than 24 hours. **C,** In teeth that sustain complicated fractures, treatment usually involves a complete pulpectomy. This final treatment is usually deferred until root development is complete by performing a cervical pulpotomy as a temporary measure. **D,** A periapical radiograph demonstrates a complicated crown fracture of the maxillary central and lateral incisors involving exposure of the pulpal tissue. The involved teeth will require root canal therapy and final restoration of the crowns. **E,** Once the root canal therapy has been completed, the teeth should be restored with full crowns. (**A,** Courtesy Dr. J. Berg, Houston, Tex; **D, E,** courtesy Dr. Seth Canion, Case Western Reserve University, School of Dental Medicine, Department of Pediatric Dentistry, Cleveland.)

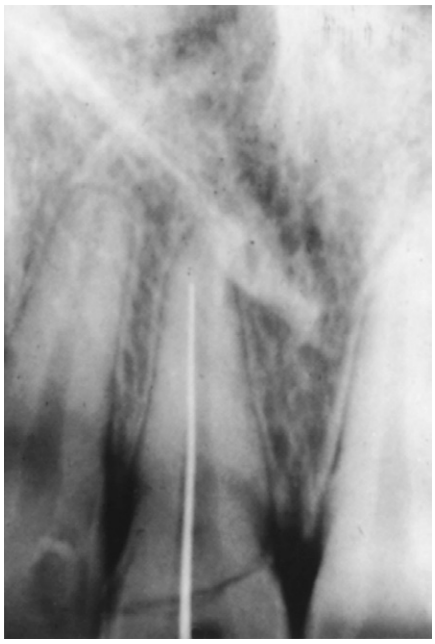


FIGURE 13-21 Endodontic treatment is indicated for mature, fully formed teeth with crown fractures that have notable pulp tissue exposure. The canals are filled with gutta-percha to prevent post-traumatic complications associated with pulp necrosis.

3.8% in the primary dentition. Careful examination of the supporting soft tissue, alveolar bone, and surrounding teeth is important in patients with suspected root fractures.

The teeth most commonly traumatized by root fractures are the maxillary incisors (75%). Concomitant fracture of the surrounding alveolar bone is associated with 40% of teeth involved with root fractures, and additional teeth are traumatized in 45% of cases initially found with root fractures.[102] Careful follow-up care is important for patients with root fractures, not only to evaluate the teeth that are directly involved but also to evaluate surrounding teeth and alveolar bone, which may demonstrate complications over time not obvious at the initial presentation and evaluation of the patient. Horizontal root fractures occur mostly in maxillary central incisors of male patients. These are fully erupted permanent teeth with closed apices and are solidly supported by bone and periodontium.[103] The most common mechanism of injury is a frontal impact on the dentoalveolar structures. The clinical examination reveals a slightly extruded tooth, usually displaced in a lingual direction and clinically difficult to distinguish because of root fracture from luxation injury. A reliable radiographic examination is necessary for a proper diagnosis. Occlusal radiographs are ideal for disclosing fractures in the apical third of roots, whereas periapical views are better for fractures in the coronal segment.[101,104]

Root fractures in primary teeth are uncommon before the completion of root development. Root fractures in primary teeth without mobility can be preserved and should exfoliate normally. If there is mobility or dislocation of the coronal segment, the tooth should be removed without attempts to remove the apical fragments, which could possibly damage the permanent tooth. Normal physiologic resorption of the apical fragment can be expected (Fig. 13-22*A*). Leaving a mobile coronal segment will more likely cause pulp necrosis to develop and removal is indicated.[104]

Root fractures of the permanent teeth are most prevalent in the maxillary central incisor region in the 11- to 20-year-old age group.[104] Studies have shown a survival rate of up to 80% in root-fractured teeth when treated appropriately.[105]

The prognosis of the involved tooth and treatment indicated are dictated by the level of the fracture along the horizontal aspect of the root (see Fig. 13-22*B* and *C*). If the fracture is in the apical third of the root and there is little or no mobility, the prognosis is good and minimal treatment is indicated.[1,3,101,103] Surgical removal of the root tip can also be considered with the retained segment sealed.[105] If possible, however, the apical segment should be left to minimize bony resorption. The coronal fragment of the tooth generally remains vital and endodontic treatment usually is not necessary. If the fracture line is closest to the gingival crevice, there is a poor chance that healing with calcified tissue will occur. The worst prognosis is associated with high root fracture where the coronal portion is mobile (see Fig. 13-22*B*) compared with apical or midroot fractures (see Fig. 13-22*C*).[104,106] The higher the fracture on the root, the greater the chance of a communication between the oral cavity and root fracture line developing, based on the degree of periodontal disease and bone loss present before the injury. It is important to evaluate the patient's oral hygiene, especially around the gingival margins, and the periodontal status around the involved tooth, which might eventually dictate treatment. If the fracture occurs near the cervical margin (see Fig. 13-22*D*) and significant mobility is present, both the coronal and apical fragments are extracted or only the coronal portion is removed, complete endodontic treatment is carried out on the retained apical portion, and restoration is completed with a crown over a post and core. Alternatively, after removal of the coronal fragment, subsequent orthodontic or surgical extrusion of the remaining apical fragments may be necessary so that the final crown is even with those of the adjacent teeth.[102] Fractures in the middle third of the root have a good prognosis, especially if there has been minimum displacement of the coronal portion of the tooth (Fig. 13-23). This type of injury is usually the most troublesome because splinting is necessary to prevent migration of the two segments. The segments will usually heal without the need for endodontic therapy. Other treatment options include replacement with endosseous implants.

The principles for treatment of permanent teeth include reduction of the displaced coronal fragments and firm immobilization.[104] The latter should be performed as soon as possible to allow optimal consolidation and repair.[101] The stable fixation of teeth with fractured roots is essential for good healing and successful treatment. Fixation is usually accomplished by splinting with an acid etch resin splint for at least 2 to 3 months to ensure sufficient hard tissue consolidation. However, no studies have established the influence of the length of

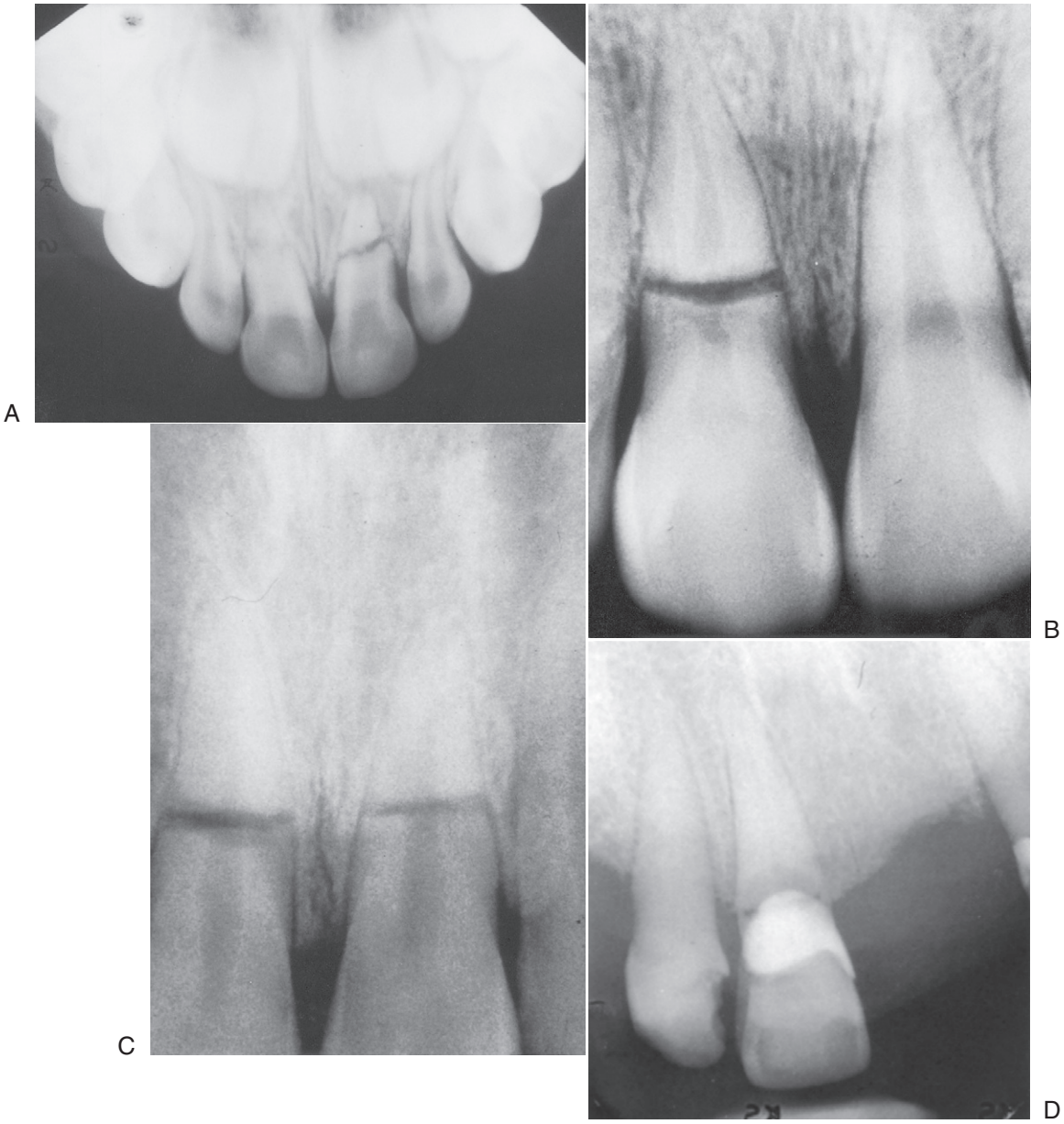


FIGURE 13-22 **A,** Occlusal radiograph reveals a root fracture of a primary central incisor. Care was taken during removal of the coronal segment and the apical segment was not removed to prevent damage to the permanent tooth. **B,** Root fracture shown in the apical third. The prognosis for this fracture is good and minimal treatment may be required. **C,** Root fracture of the coronal third of the root has a poorer prognosis because of communication with the oral cavity. **D,** Large cervical restoration that has a higher risk of root fracture. Treatment may involve removal of the coronal segment and endodontic treatment and restoration with a crown, post, and core system.

the splinting period on fracture healing. The use of rigid, orthodontic-based fixation or extensive wiring is contraindicated because it can result in additional periodontal injury and can affect healing.[103] Following stabilization, the occlusion should be examined and adjusted to avoid excessive masticatory forces on the affected tooth. Extraction is indicated for permanent and primary teeth with vertical root fractures (Figs. 13-24 and 13-25).

Follow-up examinations are important after the treatment of root fractures. Radiographic examinations should be performed at subsequent visits to evaluate pulpal and periapical healing. Radiographic findings of pulp canal obliteration and calcification, external and internal surface resorption, which occurs in approximately 60% of root fractures 1 year after injury,[107] pulp necrosis, or fracture nonhealing are possible posttraumatic complications. There is no evidence that definitively demonstrates that a delay in treatment is associated with an increased incidence of complications; however, treatment within 48 hours is recommended.[1] Pulpal necrosis has been found with displacement of the coronal fragment, forceful application of splints, nonsplinting of involved teeth, and teeth with incomplete root formation at the time of injury.

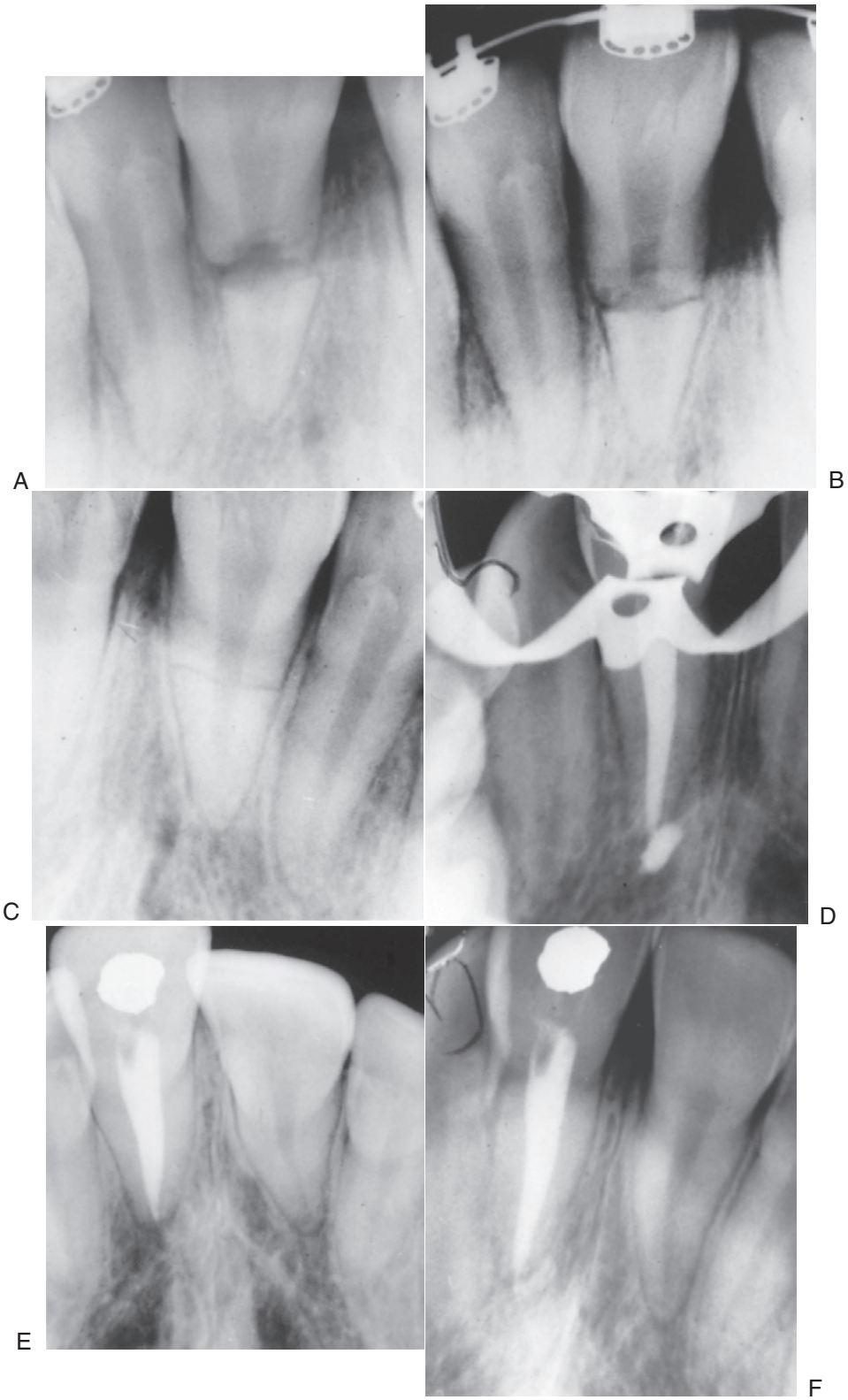
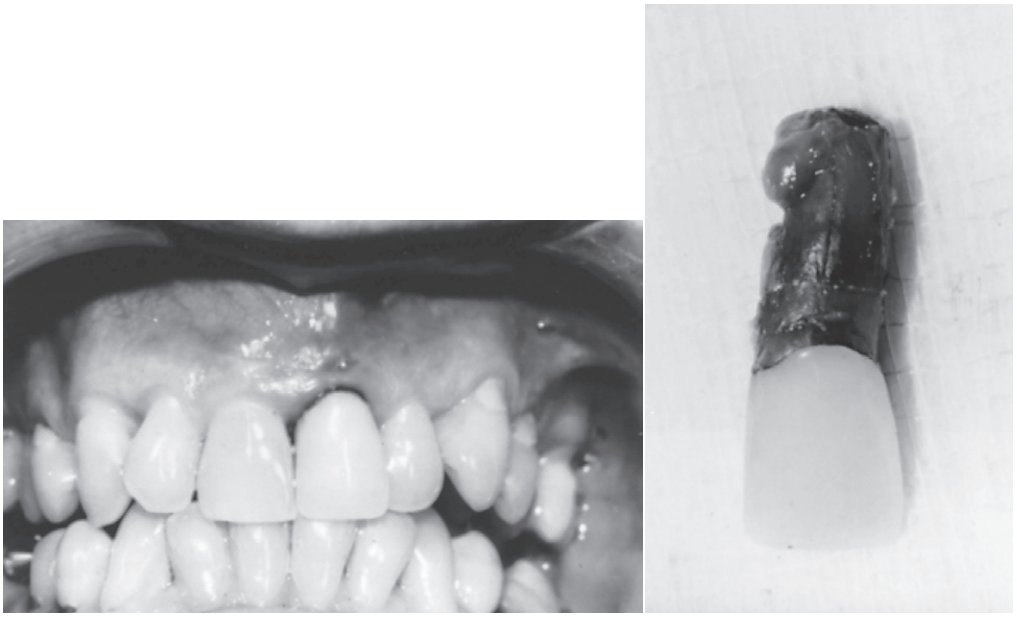


FIGURE 13-23 **A,** Fractures in the middle third of the root have a good prognosis if there has been minimal displacement of the coronal portion of the tooth. In this patient, there has been significant displacement of the coronal portion, and resorption around the fragmented segments is noted. **B,** Orthodontically, the coronal segment was slowly repositioned so that alignment could be obtained. **C,** Good alignment of the coronal and apical segments was obtained. **D,** Endodontic therapy was performed first with a calcium hydroxide paste in the canal. **E,** Endodontic treatment was completed with a gutta-percha filling material. **F,** The tooth 5 years after treatment is functional, without evidence of internal or external resorption. (Courtesy Dr. J.A. Wallace, Fox Chapel, Pa.)



A B

FIGURE 13-24 **A,** A traumatized central incisor with full crown coverage continued to be painful for a prolonged period. There was occasional purulent material that drained from a fistula in the facial mucosa. The infection was controlled with antibiotics but returned after the antibiotics were discontinued. **B,** Extraction of the involved tooth revealed a vertical root fracture. (Courtesy Dr. J.A. Wallace, Fox Chapel, Pa.)

INJURIES TO THE PERIODONTAL TISSUE

Of all dental trauma, luxation injuries most commonly occur in the permanent and primary dentition.[106] Causative factors in the permanent dentition include bicycle accidents, sports injuries, falls, and fights, whereas falls dominate in the primary dentition.[108,109] In both types of dentition, luxation is most commonly seen in the maxillary central incisor region. The type of luxation injury is dependent on the type of force and direction of impact. In the primary dentition, intrusions and extrusions comprise most injuries, probably because of the resilient nature of the alveolar bone in children of this age, whereas intrusion injuries are notably reduced in the permanent dentition. More frequently, two or more teeth are luxated simultaneously, and concomitant crown or root fractures occur. Diagnosis of the type of luxation injury is wholly dependent on clinical and radiographic examination. Common sequelae of intrusion injuries are pulp canal obliteration, pulp necrosis, internal resorption, external resorption, marginal bone loss, and transient apical breakdown, each dependent on the type of injury, maturity of the affected tooth, and subsequent treatment intervention.[110]

Concussion. In concussion injuries (see Fig. 13-15*A*), patients usually have a chief complaint of a tooth that is tender to touch. This form of injury primarily involves the supporting structures of the tooth, with no evidence of loosening or displacement. The clinical examination reveals percussion sensitivity in horizontal and vertical directions. No treatment is recommended for concussion injuries other than palliative therapy. If symptoms develop in the patient, delay of treatment of more than 1 week does not seem to have any adverse effects on outcome in this group.[1] At a minimum, occlusal grinding of the opposing tooth may be indicated to relieve excessive masticatory forces on the injured tooth. Again, with traumatic injury to the teeth, a periodic follow-up evaluation of pulpal health is necessary. Pulp necrosis may develop several weeks or months after injury; however, there is a low incidence of adverse pulpal sequelae.[7,111]

Subluxation. With damage to the supporting structures of the tooth, bleeding is often associated with subluxation (see Fig. 13-15*B*). A subluxated tooth is both sensitive to percussion and mobile, and patients have extreme masticatory sensitivity. Subluxated teeth tend to involve subcutaneous crown fractures.[110] Symptomatic treatment—such as a soft or no-chew diet and, if necessary, occlusal adjustments to remove the involved tooth from any traumatic effects of occlusion—usually allows the tooth to stabilize. Occasionally, nonrigid splinting to adjacent teeth is necessary for 7 to 10 days. Concussed and subluxated teeth have a fairly high reported incidence of pulp complications[111,112]; therefore, teeth with concussion or subluxation injuries require periodic follow-up evaluations (Fig. 13-26*A*).

Intrusive Luxation. Intrusive luxation (see Fig. 13-15*C*) of a tooth involves compression of the tooth into the alveolar socket and through the alveolar bone; this typically occurs when a child falls and the maxillary incisors receive an impact (Fig. 13-26*C*).* Intrusion of the tooth can range from minimal impaction to complete disappearance within the alveolus and supporting jaw. Significant damage is done to the periodontal ligament, resulting in a greater incidence of external root resorption, pulp necrosis, and loss of marginal bone. Partial or total disappearance of the periodontal ligament space is noted in x-rays. A percussion test on the tooth that has

*References 3, 5, 7, 76, and 108.

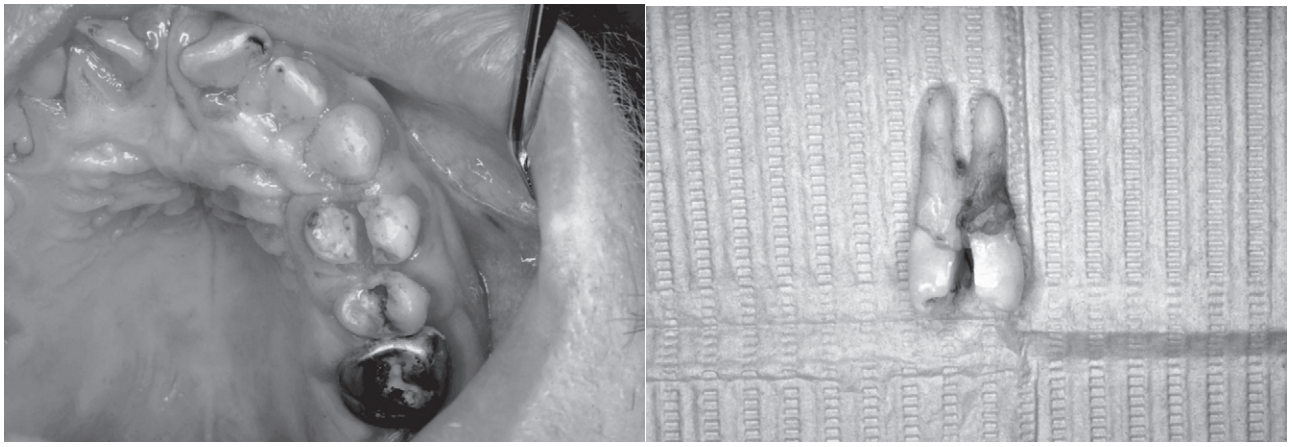


FIGURE 13-25 **A,** The patient reported chewing on a hard piece of raisin bread and had pain associated with the permanent left maxillary first premolar, which had obviously fractured. **B,** Extraction of the involved tooth demonstrated a vertical crown-root fracture, which cannot be repaired.



FIGURE 13-26 **A**, **B,** Bleeding around the gingival crevice is commonly noted with damage to the periodontal and supporting tissue of the tooth in luxation injuries. A tooth that has undergone subluxation is sensitive to percussion and mobile, with patient complaints of extreme masticatory discomfort. **C**, **D,** Intrusive luxation of a tooth can range from minimal impaction to complete disappearance within the alveolus and supporting jaw.

undergone intrusive luxation produces a dull metallic sound, similar to that of an ankylotic tooth, which is useful in differentiating the intruded tooth from a tooth that is partially erupted. This clinical finding is a result of the affected tooth being locked into a firm position in the alveolar socket. When displacement of the apex has occurred in a position as superior as the nasal cavity, hemorrhage from the nares may be seen. No definitive relationship has been found between delay of treatment and post-traumatic complications.[1] Treatment of teeth



FIGURE 13-27 **A,** These maxillary central incisors have undergone extrusive luxation during an altercation. The area is irrigated clean of debris and, with the patient under local anesthetic, the teeth are digitally manipulated into proper position in the alveolar sockets. **B,** Maxillary incisors that have laterally luxated and accompanied fracture of the alveolar support structure. This type of fracture requires digital manipulation to reposition the teeth in proper alignment and reduction of the alveolar fracture with labial and palatal compression forces. Splints that are placed for stabilization should be left in place for 4 to 6 weeks.

that have undergone intrusive luxation is controversial, and no optimal treatment has been determined.[7,112-115]

Recommended treatments include the following[7,111,114-117]:

1. The tooth can be allowed to reerupt if the tooth is immature.
2. Immediate surgical repositioning of the tooth into its proper place in the arch can be carried out. It has been shown that there is a greater incidence of external root resorption, increased risk of sequestration, and marginal bone loss with this technique because of additional trauma to the periodontal structures.
3. The teeth that have undergone intrusive luxation can be splinted to adjacent teeth.
4. Low-force orthodontic repositioning of immature and mature teeth that have undergone intrusive luxation can be carried out over a period of 3 to 4 weeks to allow remodeling of the bone and periodontal fibers, with endodontic therapy performed within 2 to 3 weeks to arrest pulp necrosis and external root resorption, which has been found in 96% of fully formed intruded teeth. Before orthodontic extrusion, the tooth should be slightly repositioned with the use of extraction forceps. Other clinicians have advocated gingival surgical procedures to provide early access for root canal therapy to prevent development of infection. CH therapy, with its antibacterial properties, has been recommended to arrest inflammatory root resorption and promote healing of the adjacent periodontal ligament space.

In the primary dentition, the permanent successor develops lingual to the primary incisor.[9] If the intruded tooth impinges on the permanent tooth, the primary tooth should be extracted immediately and as atraumatically as possible to prevent injury to the permanent tooth bud. Proper extraction techniques are used to prevent further injury of the developing tooth germ. Any instrument that increases the risk of entering the follicular space should not be used. If the intruded tooth is facially displaced and appears not to have involved the permanent successor, the tooth should be allowed to reerupt spontaneously. If, during the eruption phase, the gingiva becomes infected, the tooth should be removed and antibiotics such as penicillin or clindamycin administered to prevent damage to the permanent tooth germ[7] (see Fig. 13-26).

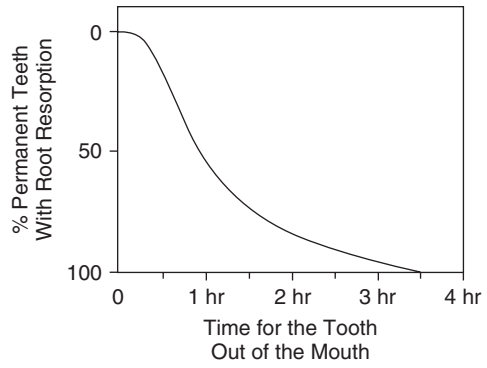
Extrusive Luxation. Extrusive luxation (see Fig. 13-15*D*) results in the apex of the tooth being displaced out of the alveolar socket with complete rupture or stretching of the apical neurovascular bundle, severing of the periodontal ligament fibers, and an intact supporting bone usually remaining (see Fig. 13-26*B*). Radiographically, the periodontal ligament space is increased. The teeth appear elongated in a lingual direction, with a dull percussion sound and evidence of exsanguination around the gingival crevice. The tooth that is partially displaced out of the alveolar socket should be manipulated digitally into proper position as soon as possible. Treatment should be undertaken as soon as possible within the first few hours following injury. Any delay in treatment will alter the prognosis of the involved teeth. Teeth that go untreated beyond 33 hours have shown an increased incidence of pulp necrosis compared with those treated earlier.[1] The tooth should be splinted with a nonrigid material, such as monofilament nylon or thin (28-gauge) wire, for 1 to 2 weeks to allow some physiologic movement of the involved tooth so that ankylosis may be prevented (Fig. 13-27). The fixation prevents the tooth from migrating incisally after being repositioned. It is highly probable that the tooth will require endodontic therapy.[5,112] Follow-up examinations with radiographic and vitality evaluation must be performed periodically to prevent loss of the tooth. The extruded primary tooth should be removed to prevent damage to the succedaneous tooth.[7]

Lateral Luxation. A laterally luxated tooth (see Fig. 13-15*E* and *F*) shows a radiographic appearance similar

to that of extruded teeth. In an occlusal exposure, the periodontal ligament space is increased apically when the apex of the tooth is displaced labially. The crowns are often displaced lingually, and percussion and mobility test results are similar to those from intrusions, again because of the locked position in the alveolar socket (see Fig. 13-27*B*). Lateral luxation of the tooth is usually accompanied by comminution or fracture of the alveolar socket. In lacerations of the gingiva, the tooth can be forced through the alveolar plate. The tooth and alveolar bone can typically be manipulated digitally (usually with force) into proper position, with concurrent compression forces on labial and palatal bone plates, and splinted to adjacent stable teeth. If treatment is delayed more than 48 hours, it is difficult to reposition the tooth manually.[1] Studies have shown that treatment should then be deferred; the tooth eventually realigns itself or orthodontic intervention is necessary.[114] Any large gingival laceration, which is common with this type of injury, should be sutured. The tooth should be stabilized for 2 to 8 weeks, depending on the extent of the displacement.[7,112] As with extrusion injuries, pulp necrosis is prevalent. Endodontic therapy may be required and follow-up examinations are necessary.

Avulsion (Exarticulation). A true dental emergency exists with complete avulsion of the tooth from the alveolar socket (see Fig. 13-15*H*). Avulsion injuries make up about 15% of all traumatic injuries to the permanent dentition, usually associated with fights, sports, and MVAs, and 7% to 13% in the primary dentition, with falls against hard objects the primary causative factor.[118] The prognosis of the pulp and periodontal tissue is directly related to proper diagnosis and to the action taken at the scene of the accident, which usually is not performed by individuals trained in dental techniques.* The most frequently involved teeth in both dentitions are the maxillary central incisors. Avulsion of these teeth usually occurs between the ages of 7 and 10 years, when the permanent incisors are erupting. According to Andreasen, the loosely structured periodontal ligaments and resilient alveolar bone surrounding erupting teeth provide minimal resistance, which favors avulsion over other injuries.[7]

The goal of reimplanting teeth after traumatic avulsion is to maintain the viability of the cells of the pulp and periodontal ligament, which would assist reattachment and prevent post-traumatic complications of root resorption.† The success of reimplantation is inversely related to how long the tooth is out of the socket (Fig. 13-28*A*). The sooner the tooth is reimplanted, the better the prognosis. The single most important factor that will affect treatment outcome is speed. Ideally, the avulsed teeth should be replanted within 20 minutes of injury. The longer the periodontal tissue is allowed to dry out, the less opportunity there is for regeneration of the periodontal fibers within the alveolus and tooth, with loss of normal physiologic metabolism and morphology of periodontal ligament cells. Improved prognosis is based on management of inflammation, which is stimulated by



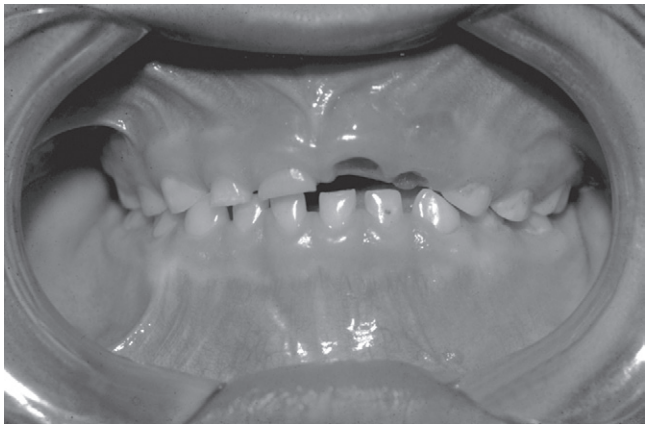


FIGURE 13-28 **A,** Successful reimplantation is inversely related to the length of time a tooth is out of the socket. Reimplantation of a tooth within the first 15 to 20 minutes produces a favorable long-term prognosis. **B,** Avulsed primary teeth should not be reimplanted. The socket site should be inspected, any debris or fragments should be removed, and the area should be irrigated. Appropriate sutures should be placed for reapproximation of soft tissue.

damage to the attachment apparatus and infection of pulpal tissue.[83,123] The width and length of the root canal, stage of root development of the tooth, and type of extra-alveolar storage once the tooth has been avulsed are significant factors determining tooth survival. Andreasen and Hjorting-Hansen[122] have observed that after a period of 2 or more years, 90% of teeth reimplanted in less than 30 minutes after avulsion exhibit no discernible resorption of roots. In contrast, root resorption was seen in 95% of teeth reimplanted after an extraoral period longer than 2 hours. Root resorption has been attributed to cemental damage on the external surface of the root, with the necrotic pulpal tissue providing a possible source for infection. Reimplanting the tooth within the first 15 to 20 minutes prevents the loss of normal periodontal ligament cells and has a favorable long-term prognosis.[124,125]

Because time is critical, reimplantation at the site of injury has the best prognosis.* Most parents and caregivers have little information regarding avulsion and reimplantation guidelines, so direct instruction is usually required.[126]

*References 5, 7-9, 73, 76, 86, 112, and 119.
†References 7, 63, 112, and 120-122.

*References 7, 63, 73, 112, and 120-122.

TABLE 13-1 pH and Osmolality of Various Storage Media

Medium	pH	Osmolality (mOsm/kg)
Saline	7.0	295
Tap water	7.5	12
Saliva	6.3	110–120
Viaspan	7.4	320
Gatorade	3.0	280-360
Milk	6.75	275
Coconut water	6.2	288
Blood plasma	7.2-7.4	290
Hanks' balanced salt solution 7.0	270-290	

From Hiremath G, Kidiyoor KH: Avulsion and storage media. *J Invest Clin Dent* 2:89, 2011.

Telephone instructions to the patient, parent, caregiver, or supervising personnel should be to inspect the tooth for debris by holding the tooth by the crown, to cleanse with saline or milk or simply sucking or spitting on the tooth, and to place the tooth back into the socket immediately. After reimplantation, the tooth should be held in place with light pressure en route to the office or clinic. If the tooth cannot be replaced, it should be stored in the buccal vestibule or under the tongue of the patient unless, because of the extent of injury or the age and cooperation level of a child, there is a concern that the tooth may be aspirated or swallowed. The tooth can then be stored in the mouth of the parent or supervising adult for transportation without adverse consequences. If neither option is available, the same effect can be achieved if the tooth can be stored in milk, saline (1 teaspoon of salt added to 8 ounces of water), or a saliva-soaked towel. Storage in tap water has been documented to have adverse effects on periodontal healing and is not recommended for cleansing or transportation of avulsed teeth.* Water is the least desirable storage medium because of its hypotonic environment, which can cause cell lysis.[124] Milk is considered the best medium because of its availability, pH compatibility to vital cells, freedom from bacteria, and function of maintaining the vitality of the periodontal ligament cells 3 hours in the postavulsion period.[128] A comparison of different storage media is shown in Table 13-1. A storage medium that has a similar pH and osmolality to those of blood plasma is desired to preserve the periodontal ligament cells.[129]

The tooth should never be allowed to dry. Even short drying periods can do irreversible damage to the periodontal ligament of the avulsed tooth and can cause rapid loss of the tooth through resorption.[7,112] Another transport medium is a tooth-preserving system known as Hank's balanced salt solution (HBSS), which is considered to have excellent storage potential while a history and physical examination is conducted. The advantage of this system is an inner suspension netting and removable basket that permit gentle washing without crushing

*References 7, 80, 81, 118, and 127.



FIGURE 13-29 Save-A-Tooth emergency tooth-preserving system (3M Health Care, St. Paul, Minn). (From Scuderi GR, McCann PD, Bruno, PJ: *Sports medicine: Principles of primary care*, St. Louis, 1997, Mosby.)

of the periodontal ligament. A cell culture medium in a transport container, Viaspan (Belzer UW-CSS, DuPont Pharmaceuticals, Wilmington, Del), claims to be superior to milk with healing results found with HBSS.[130] Data have shown that HBSS is able to preserve the vitality of periodontal ligament fibroblast (PDLF) cells to a greater extent when compared with Viaspan and Eagles solution, but clinical relevance has not yet been established.[80,81] Approximately 75% of dentoalveolar trauma occurs close to home or school. Availability of the media in kits at homes, schools, and day care centers, which have a large number of children between the ages of 8 and 12 years, may be prudent.[83] A plan should be developed with the staff to handle such an emergency efficiently because time and tissue management are critical to success (Fig. 13-29).

The primary goal of the emergency visit is to reimplant the tooth with a maximal number of vital periodontal ligament cells that will regenerate and repair the ligamentous attachment of the tooth to bone (Fig. 13-30). Initially, a thorough medical history along with a full history of the accident, is essential, including information about where the tooth was recovered, "dry time," and storage media. This information governs treatment choices. The teeth and traumatized tissue should be inspected and evaluated. If the patient does not have the

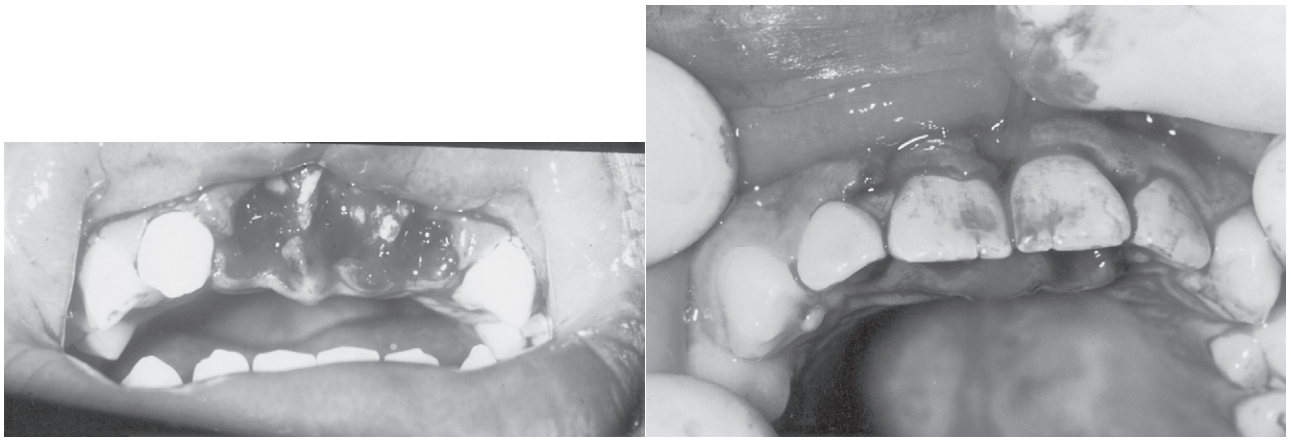


FIGURE 13-30 **A,** Avulsive trauma of anterior maxilla showing degree of tissue damage, debris, and blood clots. These sites were examined for fractures and then gently irrigated. **B,** The avulsed teeth were gently manipulated into position and splinted for 7 to 10 days with a semirigid acid etch resin splint.

tooth, careful clinical examination must be performed to rule out aspiration (see Fig. 13-11), swallowing of the tooth (see Fig. 13-10*B* and *C*), intrusive luxation, or displacement of the tooth in surrounding soft tissue of the head and neck (see Fig. 13-10). Chest, abdominal, panoramic, and facial films should be obtained to rule out adverse possibilities. If the tooth has been left at the scene of the accident, every attempt should be made to retrieve it.

The tooth should be evaluated to determine whether it is of the primary dentition. The root of the tooth should be inspected for evidence of resorption or a radiograph should be taken of the alveolar bone to locate a succedaneous tooth. Avulsed primary teeth should not be reimplanted because there is risk of pulp necrosis and possible interference with the development of succedaneous teeth (see Fig. 13-29).[7,8,63,112] No space problems will develop in the permanent dentition with premature loss of primary teeth, but eruption often proceeds in a labial direction.

Andreasen[7] has proposed that the following conditions be considered before reimplanting a permanent tooth:

1. The avulsed tooth should be without advanced periodontal disease.
2. The alveolar socket should be reasonably intact to provide a seat for the avulsed tooth.
3. There should be no orthodontic considerations, such as significant crowding of the teeth.
4. The extra-alveolar period should be considered. If the tooth is reimplanted within 30 minutes of avulsion, there is a good chance of successful reimplantation. For extra-alveolar periods longer than 2 hours, complications associated with notable root resorption increase greatly.
5. The stage of root development should be assessed. Survival of the pulp is possible in teeth with incomplete root formation if reimplantation is accomplished within 2 hours after injury.

If the tooth has been successfully reimplanted before evaluation by the dentist, a radiograph should be obtained to verify the position within the alveolar socket and assess the extent of damage to surrounding structures (e.g., root fractures, intrusive luxations). A clinical examination of the socket site is mandatory to ascertain whether it is intact and acceptable for replantation of the avulsed tooth. The socket site is rinsed gently with saline and examined for the presence or absence of alveolar bony wall and the extent of fracture. The health of the soft tissue of the socket affects the prognosis of reimplantation and should be left unaltered. The soft tissue should not be manipulated with instrumentation and the site should be gently irrigated to remove debris and blood clots. However, if a collapse is evident, a blunt instrument should be inserted into the socket carefully in an attempt to reposition the fractured piece of bone. If the tooth position is unacceptable, the tooth should be removed gently and reimplanted into the correct position. The involved tooth should be splinted with a semirigid acid etch resin splint for 7 to 10 days. Studies have indicated that rigid splinting of reimplanted teeth increases the extent of root resorption; thus, a minimum of 1 week is sufficient. If there has been a notable concomitant alveolar fracture, a rigid splint should be used for 3 to 4 weeks.[124]

If the tooth is transported to the office or requires repositioning, it should be gently cleansed of debris with saline-soaked gauze or a stream of saline from a syringe until visible contaminants are removed. To prevent damage to vital periodontal tissue and cementum, no effort should be made to scrape the tooth surface or sterilize it with solutions before reimplantation. To prevent further damage to the root surface, the tooth should fit loosely within the alveolus. The tooth should then be manipulated gently into proper position using digital pressure. It is not necessary to suction the blood clot from the socket before reimplantation.

The tooth should be splinted and gingival and soft tissue lacerations should be sutured as indicated. The traumatized teeth should be removed from the occlusion and the patient should receive a soft diet for 2 to 3 weeks. Care should be taken with removal of the splint after 7 to 10 days because the involved tooth will still be mobile. Splinting longer than 7 to 10 days may promote root resorption (Figs. 13-31 and 13-32).[7]

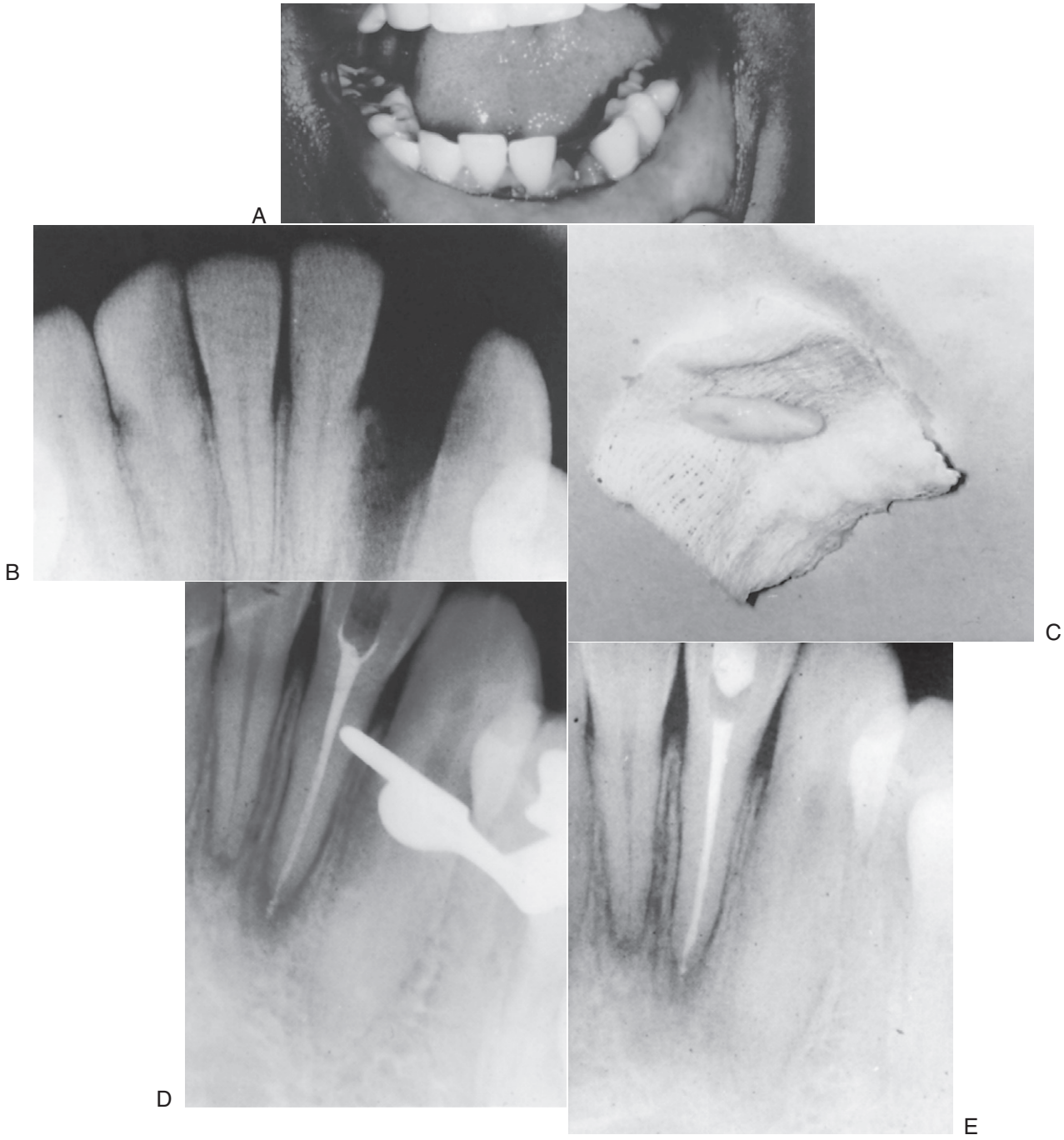


FIGURE 13-31 **A,** This lateral incisor was completely avulsed during an altercation. The tooth was located and the patient was instructed over the telephone to place the tooth in his mouth and get to the dental clinic as quickly as possible. **B,** On arrival at the dental clinic, a radiograph revealed no obvious damage to the supporting bone or surrounding teeth. **C,** The tooth is gently cleansed of debris with saline-soaked gauze without scraping or the use of a sterilization solution to prevent damage to the periodontal tissue and cementum. The tooth is then gently manipulated into the alveolar socket and splinted for 1 week. **D,** Endodontic therapy is initiated 2 weeks after reimplantation of the tooth. The canal is first filled with calcium hydroxide paste and treatment is eventually completed with a gutta-percha filling material. **E,** The tooth remains functional without evidence of root resorption 5 years after implantation.

Factors that affect healing of the avulsed tooth include extraoral time, use of suitable storage media, and type and duration of splinting.[1,131] Andreasen[7] has categorized periodontal healing of avulsed teeth into groups based on histologic evaluation. A useful flow chart for treating avulsion can be seen in Figure 13-33. This provides a succinct and useful guide for treating a patient who sustains an avulsion injury.[132]

Healing With a Normal Periodontal Ligament. The periodontal ligament of the avulsed tooth repairs completely. Small areas of resorption on the root surface are repaired by new cementum deposits, with eventual



FIGURE 13-32 A comprehensive evaluation of the soft tissue is imperative during the initial examination because of the nature of force in avulsion injury. This patient not only suffered avulsion of the mandibular lateral incisor but also a complex laceration of the ventral aspect of the tongue, which was overlooked in the initial examination and was discovered on follow-up.

complete regeneration of the periodontal ligament. A normal periodontal space can be seen in a radiograph and a normal percussion sound can be elicited. It is important to note that this type of healing is rare with tooth avulsion trauma, because trauma to even the innermost layer of the periodontal ligament leads to surface resorption.

Healing With Surface Resorption. This is a self-limited process that affects superficial localized areas of damage to the periodontal ligament. The tooth radiographically and clinically appears to be in normal position, eliciting a normal percussion tone.

Healing With Ankylosis (Replacement Resorption). The development of replacement resorption depends on the degree of damage to the periodontium at the time of avulsion and the extent that viable periodontal cells on the root surface are maintained.[132] The blood clot in the damaged periodontal ligament of the avulsed tooth is organized into granulation tissue, which is replaced by bone, usually 2 weeks after implantation. The tooth undergoes progressive replacement resorption if the entire periodontal ligament space is removed before replantation or transient replacement resorption if a limited amount of periodontal ligament space is removed with the avulsion. The tooth becomes ankylosed and the root is gradually replaced by bone. The normal periodontal space is not evident in a radiograph. The areas of root resorption can be detected within 2 months of injury, beginning in the apical third of the root, and most cases are evident 1 year after reimplantation. The degree of replacement resorption varies with the age of the patient (e.g., reimplanted teeth with a damaged periodontal ligament space in younger patients are resorbed within 3 to 7 years, whereas in older patients, reimplanted teeth remained functional for a longer time).[133] To minimize replacement resorption of avulsed teeth, studies have used materials placed between the tooth and socket, such as silicone grease and methyl methacrylate, surgical sponge (Gelfoam), fascia, and cutaneous connective tissue, with limited success.[118]

In the fully developed jaw, ankylosis of teeth presents no difficulty. The teeth may be maintained until root replacement leads to loosening and exfoliation. However, in younger patients in whom maturation of the alveolar structures has not occurred, ankylosis may interfere with the normal growth of the alveolar process. When infraocclusion is diagnosed in these individuals, the tooth should be extracted to prevent malocclusion.[63] Clinically, the percussion tone is high and may reveal replacement resorption in the initial phases.

Inflammatory Resorption. The cause of inflammatory resorption is a communication between surface resorption and pulp via the dental tubules. Toxic byproducts and bacteria from the necrotic pulp tissue seep into the periodontal tissue and cause an inflammatory reaction.[134] Resorption is accelerated and, within a few months, the root may be completely resorbed. Inflammatory resorption is common after reimplantation of the permanent incisors in the 6- to 7-year-old child because of wide dentinal tubules, a thin protective cementum layer, or both. Clinically, the reimplanted tooth is loose, extruded, and sensitive to percussion, producing a dull sound. Affected reimplanted teeth may undergo replacement and inflammatory resorption processes simultaneously.

Root resorption is the major complication with traumatically avulsed teeth, usually associated with long extraoral storage times or an unsuitable storage environment at the time of avulsion.[124,135] The loss of vitality of the periodontal ligament influences the progression of resorption of the root surface. Even short drying periods of the avulsed tooth can have an adverse effect on the periodontal ligament. Unless treated, inflammatory resorption can result in rapid loss of the reimplanted tooth as early as 3 months after reimplantation. In cases in which there has been a significant period when the avulsed tooth has been allowed to dry more than 20 minutes, successful reimplantation of the tooth has been reported, including regrowth of the periodontal ligament, with the use of Emdogain (Biora, Malmo, Sweden), especially in the 20- to 60-minute drying time period.[136,137]

Appropriate endodontic therapy can arrest the resorptive process. Treatment is based on the removal of the source of infection.[138] All reimplanted teeth with complete root formation should be treated endodontically within 7 to 10 days, prior to splint removal.[7,122,139] CH is recommended as an interappointment medication before the placement of gutta-percha because of its antibacterial effects. Controversy exists regarding how long CH should be used. Hammarstrom et al have claimed claim that because of the necrotizing effect on the cells repopulating the root surface, the use of CH should be contraindicated on damaged root surfaces—for example, avulsion injuries.[132] Lengheden et al have studied the prolonged use of CH in avulsed teeth and concluded that because of the relatively high pH of CH , necrosis in growing periodontal healing cells and damaged cells occurs and ankylosis develops.[140] Other agents that have been used to provide a temporary canal-filling material include an antibiotic-corticosteroid paste (Ledermix, Lederle, Wolfratshausen, Germany) and the hormone calcitonin, both of which help minimize inflammatory root resorption. Andreasen has demonstrated that

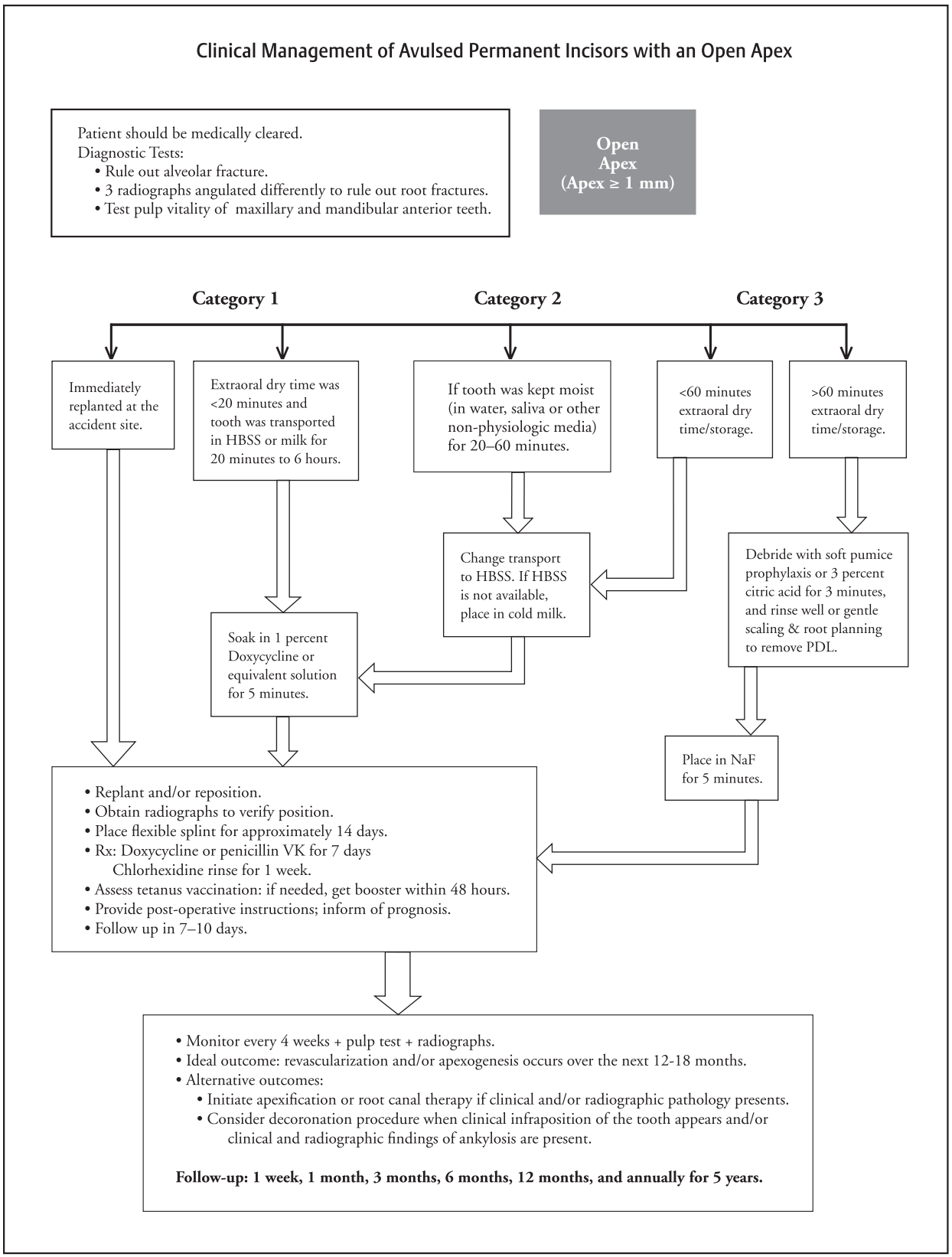


FIGURE 13-33 Flow charts of closed apex and open apex avulsion. (From McIntyre JD, Lee JY, Trope M, Vann WF Jr. Permanent tooth replantation following avulsion: Using a decision tree to achieve the best outcome. *Pediatr Dent* 31:137, 2009.)

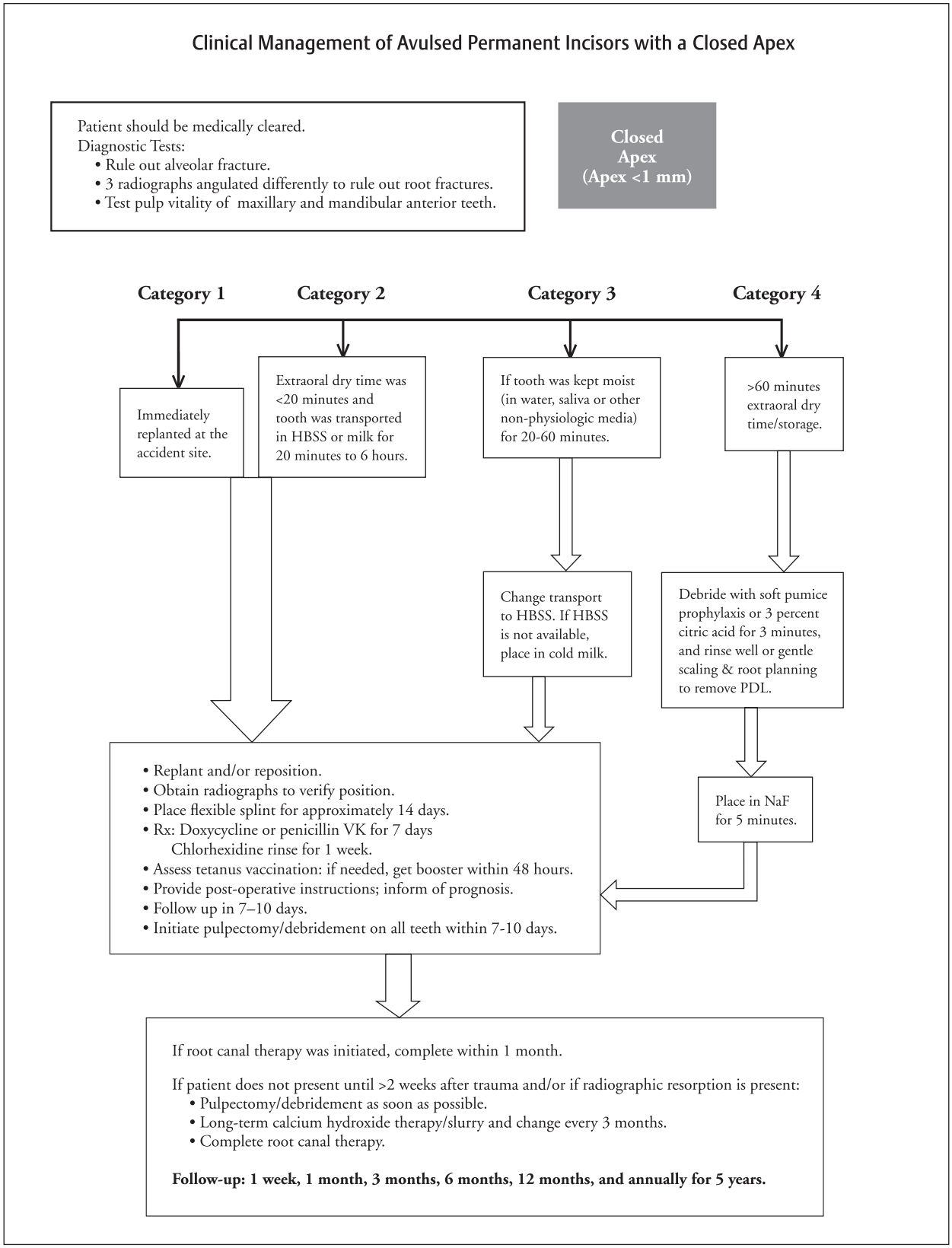


FIGURE 13-33, cont'd

extraoral root canal procedures and root canal filling materials injure the periodontal ligament, resulting in increased ankylosis when compared with nonendodontically treated avulsed teeth.[7] This study recommended that endodontic treatment should be initiated 1 to 2 weeks after reimplantation to halt the development of inflammatory resorption and allow reformation of periodontal fibers. However, a recent study has shown that extraoral endodontic treatment is not detrimental for avulsed permanent teeth that are replanted after more than 60 minutes of extraoral dry time.[141]

The key to retarding the inflammatory resorption process is early initiation of endodontic therapy to eliminate a potential bacterial source within the necrotic pulpal tissue. The periodontal ligament space is allowed to heal and a CH filling material is placed, followed by the definitive canal filling. Other techniques to overcome the resorptive process include the replacement of the apical part of the root with cast Vitallium and ceramic implants. These procedures do not prevent resorption but prolong the survival time of the reimplanted tooth.[118]

If more than 2 hours elapse before reimplantation can be achieved, extraoral endodontic therapy should be accomplished[141] and the canal should be filled with CH, which has been shown to be effective in arresting inflammatory resorption.[7] In teeth with prolonged extraalveolar time, application of 2% phosphate-acidulated sodium fluoride (pH, 5.5) for 20 minutes has been shown experimentally in animal models to slow the replacement resorption to approximately 50%.[142,143] Studies have yet to confirm similar results in human teeth. Doxycycline has been advocated for root preparation to reduce microorganisms in the pulp lumen and increase pulp revascularization in immature teeth.[144] Other agents used to protect the root from inflammatory resorptive changes include tetracycline-doxycycline, stannous fluoride, citric acid, hypochloric acid, formalin, alcohol, diphosphonates, and indomethacin solutions.[118]

Immature teeth with wide open foramina that are reimplanted less than 2 hours after avulsion should be reimplanted and splinted without endodontic therapy for possible revascularization of the pulp. Radiographic examination should be performed after 2 or 3 weeks and, if root resorption is noted, endodontic therapy should be initiated immediately and CH paste packed into the root canal to eliminate inflammatory resorption.[7] Revascularization of the root is promoted with a solution of 1 mg of doxycycline in 20 mL of saline for 5 minutes. After arresting the resorption and closing the apex, a final endodontic restoration should be completed.

Tetanus prophylaxis or booster injection should be administered after injury if the last injection was given more than 5 to 10 years previously. Human tetanus antitoxin, 250 units given intramuscularly, is recommended for dirty wounds untreated for more than 25 hours.[145,146] Antibiotic coverage with penicillin or clindamycin is indicated to minimize bacterial activity in the periodontal and pulp tissue and twice-daily use of a 0.12% chlorhexidine rinse for 7 to 10 days is indicated to improve oral hygiene and overall oral health.

The prognosis of reimplanted permanent teeth is guarded because many teeth are destroyed by root resorption. Successful reimplantation requires expedient treatment at the time of the accident and close follow-up visits every 6 months for at least 3 years and annually for as long as possible. If more than 2 hours have elapsed since avulsion of the tooth or if the periodontal ligament has not been kept moist, there should still be an attempt to reimplant the tooth, with the patients and parents, if necessary, being made aware of the poor prognosis (Fig. 13-34).[124]

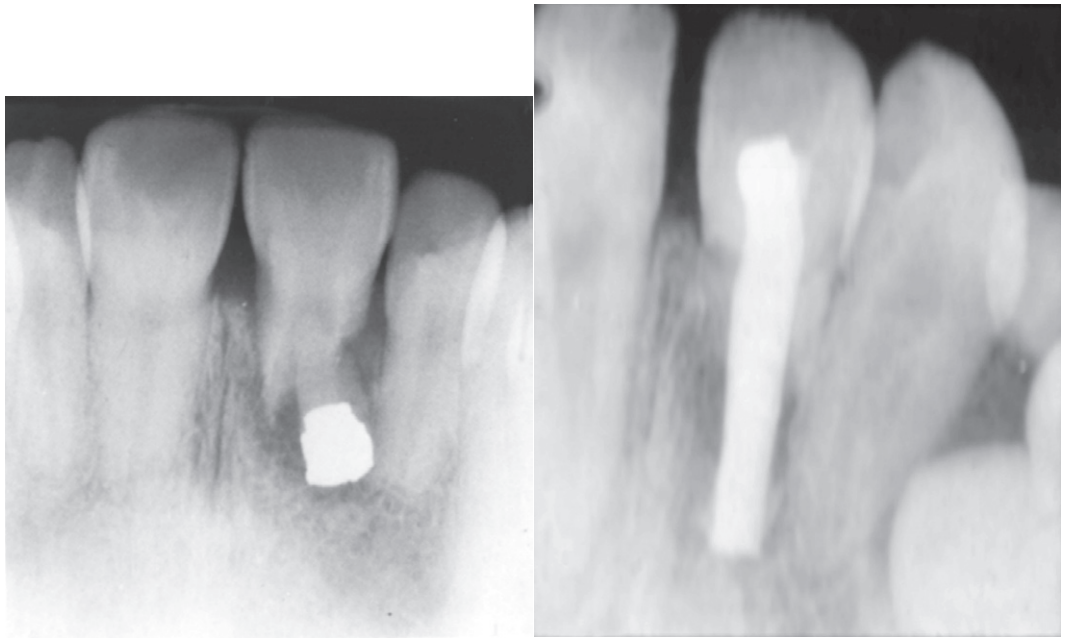
Many factors, however, still favor replanting such teeth. First, reliability of failure predictors has not yet been tested in prospective studies. Second, preservation of even resorbing replanted teeth may offer significant long-term advantages in preparation for definitive treatment. Also, for psychological reasons, replantation can significantly reduce the anxiety and despair of the injured child and parents. Furthermore, decoronation of a resorbing anterior tooth will allow it to serve as a matrix for alveolar bone formation and preserve an otherwise resorbing alveolar process, thereby leaving an environment of bone and soft tissue that is optimal for single-implant insertion or fixed prosthesis. Finally, replantation and subsequent decoronation, if indicated, appear to be cost-effective in comparison with nonreplantation combined with subsequent repeated prosthetic tooth replacements because of vertical alveolar growth of adjacent ridge areas, with eventual definitive implant placement or a fixed prosthesis.[147]

Preservation of roots in the alveolar process seemed to maintain the bone volume, enabling better conditions for later implant placement; 41 implants (97.6%) were integrated successfully. Given that patients have finished growth, with careful treatment planning and timing, the functional and aesthetic outcome of single-tooth implant treatment today is excellent and can be recommended for replacing tooth losses after trauma in the anterior region of the maxilla.[148]

INJURIES TO THE SUPPORTING BONE

Comminution of the Alveolar Bone. Comminution of the alveolar socket (see Fig. 13-16*A*) is usually associated with lateral or intrusive luxation injuries. The fractures are generally reduced with digital manipulation and the luxation injury is treated. Follow-up for evidence of root resorption of the involved teeth is indicated.

Fractures of the Alveolar Socket Wall. Alveolar socket wall injuries (see Fig. 13-16*B* and *C*) are frequently seen in the upper incisor region, usually affecting several teeth, and typically are associated with luxation injuries.[149] Generally, there is mobility of the buccal osseous plate with the involved teeth and evidence of contusion of the gingiva or mucosa. Lateral extraoral radiographs best demonstrate the location of the fractured site. Reduction of the fracture involves simultaneous digital pressure of the coronal aspect of the crown and apex along the fracture site. After reduction of the fracture, the occlusion should be checked and the involved teeth removed from the forces of traumatic occlusion. Soft tissue lacerations should be sutured and the involved teeth should be in a rigid splint for 4 weeks to allow osseous healing. Alveolar process fractures with primary teeth that are not notably displaced or easily



A B

FIGURE 13-34 **A,** This central incisor was avulsed in a playground accident when the patient was 8 years old. The patient reported that she put the tooth in her pocket and got to her family dentist after approximately 3 hours, with the tooth still in her pocket. After informing the family of the poor prognosis, her dentist filled the canal retrograde with amalgam extraorally and replaced the tooth. The tooth functioned for almost 20 years without difficulty, except discoloration. Mobility of the tooth eventually resulted because of resorption of the root, requiring extraction. **B,** A similar case showing root resorption involving a central incisor that had been in function for a number of years following root canal therapy. The tooth was ultimately extracted because of mobility. (**B,** Courtesy Dr. Seth Canion, Case Western Reserve University, School of Dental Medicine, Department of Pediatric Dentistry, Cleveland.)

manipulated back into proper position may not require splinting because the bone heals quickly in children. However, splinting of the involved segment may be difficult in children because of the lack of sufficient tooth support. The child should maintain a soft diet for 2 weeks, with periodic follow-up examinations required to monitor pulp health.

Fractures of the Alveolar Process. Fractures of the alveolar process (see Fig. 13-16*D* and *E*) are found predominantly in the anterior teeth and premolar region of older children.[7] These injuries may be isolated or may be seen in conjunction with other dental and/or facial injuries. The patient frequently reports that the occlusion has been changed as the involved segment has shifted from its original position (Fig. 13-35). The fracture is readily identified with an intraoral radiograph.

Treatment involves reduction and immobilization of the involved segment and stabilization for 4 weeks to allow osseous healing. Closed reduction of the alveolar fracture may be accomplished with digital manipulation of the alveolar and dental segment. Stabilization can be accomplished satisfactorily with a rigid acid etch resin splint or lingual splint (Fig. 13-36). An open reduction of the alveolar fracture may be necessary if the alveolar segment is notably displaced or the involved teeth are displaced so that the roots are locked over the osseous plate (Fig. 13-37). A vestibular incision is made below the fracture line and a subperiosteal flap is elevated to expose the fracture site. A no. 1 or 9 periosteal elevator can be used in the fracture line to elevate the segment and place the alveolus in proper position. Frequently, no osseous



FIGURE 13-35 Alveolar process fracture associated with anterior mandibular teeth. The segment moves as one unit with teeth and bone. Note the contusion of the gingival and mucosal tissues.

fixation is necessary but, if indicated, the segment can be wired or a bone plate placed after reestablishment of the proper occlusion is verified. Rigid splinting of the involved teeth or use of an Erich arch bar for 4 weeks provides adequate stability for osseous healing (see Fig. 13-37).

Careful follow-up is mandatory to evaluate pulp necrosis and periapical inflammation. These complications are rather frequent and are apparently related to the time interval between injury and fixation. The best prognosis is associated with alveolar fractures treated within 48

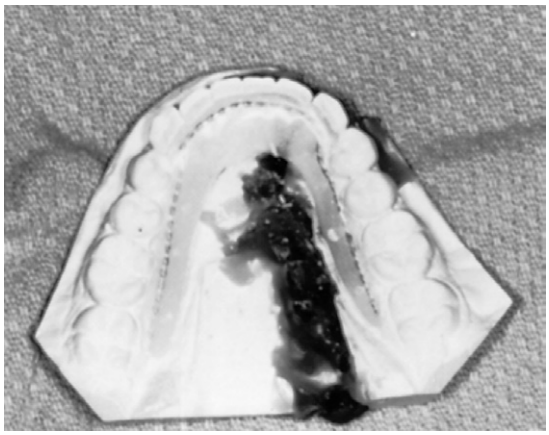


FIGURE 13-36 This segment was digitally manipulated into proper occlusion and position. A lingual splint was fabricated to fix the involved segment rigidly.

hours after injury.[1] Teeth splinted within 1 hour after injury develop pulp necrosis less frequently than teeth splinted after longer periods.[7] Common complications after treatment include pulp necrosis, pulp obliteration, root resorption, and loss of supporting bone. A tetanus booster usually is not indicated unless the wound has contaminants such as road debris or dirt. Coverage with penicillin or clindamycin and the use of chlorhexidine oral rinse are recommended.

Fractures of the Maxilla and Mandible. The management of maxillary and mandibular fractures (see Fig. 13-16*F* and *G*) is discussed in Chapters 14 and 18.

SPLINTING TECHNIQUES

Splinting provides stabilization of traumatized teeth and prevents further damage to the pulp and periodontal tissue during the healing period, allowing the attachment apparatus time to regenerate.[150] It is important that the splint allow slight flexibility, or physiologic mobility, to accelerate the healing of periodontally injured teeth.[151] The maintenance of oral hygiene and prevention of infection are important in promoting periodontal

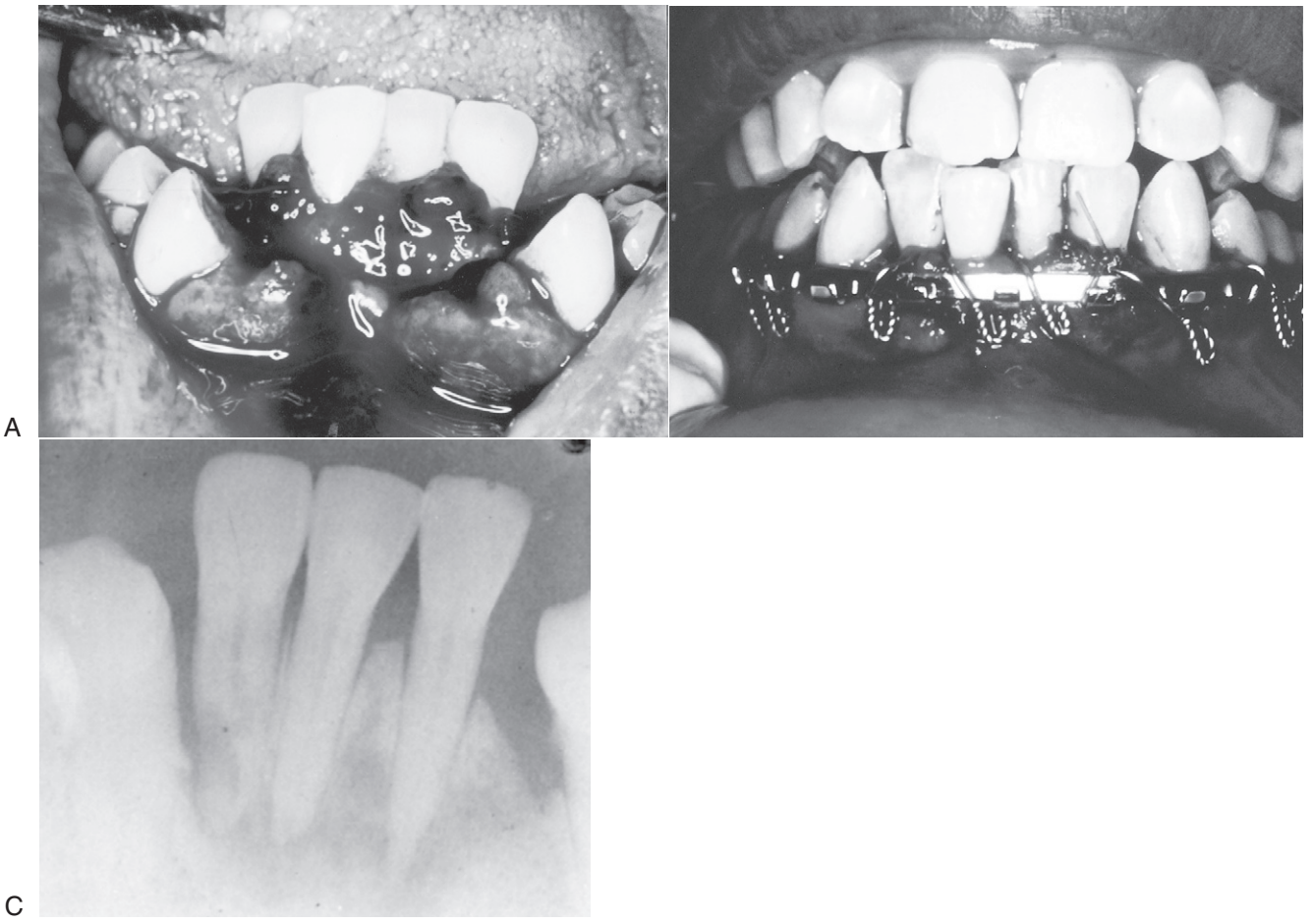


FIGURE 13-37 **A,** Mandibular dentoalveolar fracture following traumatic injury. **B,** After the segment is out of traumatic occlusion, an Erich arch bar is used for 4 weeks to stabilize the segment. **C,** This patient was involved in an accident 5 years before this radiograph, during which the mandibular incisors and alveolar bone were lingually displaced. The teeth were repositioned digitally without splinting or removal from traumatic occlusion. The segment healed to a fibrous nonunion, with root resorption of the lateral incisors that required removal of the entire segment.

healing during stabilization. No portion of the acrylic composite or wire of the stabilization device should impinge on the gingival margins of the teeth. Splints that irritate the gingiva and cause gingival inflammation prevent the resolution of the inflammatory response in the marginal gingiva. The patient must be able to keep the gingival tissue as clean as possible, which is difficult with cap splints, foil splints, and cold-cured acrylic splints. Fixation methods used for dental splints may vary with the type of dental trauma; however, in most cases, a splint of simple design in place for 7 to 10 days is recommended. In cases of root and alveolar bone fractures, longer periods of fixation are necessary, usually 2 to 4 months and 6 weeks, respectively. However, controversy exists concerning the duration of fixation and posttraumatic complications. Studies have shown that the duration of fixation is unrelated to the success of posttraumatic healing and does not affect any negative sequelae, and that the period for fixation is wholly dependent on the specific clinical situation presented.[111]

Erich arch bars, lingual splints, or both, can be used for stabilization of alveolar process fractures if the teeth within the segment are stable. Arch bar splints are not recommended for tooth fixation because the tied wire tends to loosen with time and rest on the marginal gingiva, causing mechanical irritation and a site for bacterial deposition. If the teeth are mobile, the supporting wires, if positioned apically to the cervical prominence, may have a tendency to elevate the tooth slowly. The Schuchardt splint was developed for the fixation of luxated teeth but has proved too rigid and complicated to construct because it was made with an aluminum-brass alloy bar 2 mm in diameter. An acrylic cap splint cemented to the noninjured teeth has been used in luxation injuries but is also too rigid.[150]

Interdental wiring techniques, such as figure-eight wiring and loop wiring,[73,145] can be used but are technically difficult and troublesome. The patient may have difficulty in cleaning around the wires and the wires may slip apically below the cervical prominence of the tooth and elevate the tooth or damage the cementum surface.

The requirements for an acceptable splint are as follows[7,114,152]:

- It is easy to fabricate directly in the mouth, without lengthy laboratory procedures.
- It can be placed passively without force to the teeth.
- It does not contact the gingival tissue and thus cause gingival irritation.
- It does not interfere with normal occlusion.
- It is easily cleaned and allows proper oral hygiene.
- It does not traumatize the teeth or gingiva during application.
- It allows an approach for endodontic therapy.
- It is easily removed.
- It provides good aesthetic results.
- It does not injure the pulp of the traumatized teeth or adjacent teeth.
- It does not interfere with intraoral radiographic techniques.
- It allows placement of a rubber dam in all types of dentition.
- It does not promote root resorption.
- It allows slight mobility so that the position of the tooth after reimplantation exerts minimal pressure between the root surface and the alveolar bone.
- It is economical and requires minimal specialized equipment.

Acid Etch Resin Splint

Acid etch resin fixation techniques provide a relatively easy, versatile method for stabilization of teeth with effective, aesthetic composite resin materials.[7,150,153] The labial surfaces are cleansed of blood and debris as much as possible; the use of a cotton roll or gauze in the vestibule and over gingival lacerations assists in keeping the surface as clean and dry as possible. The teeth are air-dried, and a 35% phosphoric acid gel is applied to the incisal third of the traumatized and adjacent teeth, as indicated.[154] After the gel has been on the surface for approximately 20 seconds, it is removed with a water spray. The teeth are then air-dried to reveal a frosty white surface, signifying a successful etch. Depending on the composite resin system used, the teeth are stabilized with a band of the material along the etch surfaces. The composite resin material is then allowed to cure or is cured using ultraviolet light with a light-activated system.

This type of system provides longer working time and better control of the material. The occlusion should be checked and the splint altered if it interferes with proper occlusion. The splint should be smoothed and polished for improved patient hygiene and comfort; however, if the teeth are still slightly loose and tender, the polishing procedure might better be postponed to a later visit.

This method provides excellent stabilization and allows the patient to keep the teeth and gingiva clear because the splint is away from the periodontal tissue.[155] The procedure is simple and efficient, may not require anesthesia, is hygienic, and serves as definitive treatment.[156] One disadvantage of the method is that the material may fracture because the acrylic is brittle when exposed to occlusal masticatory forces. It has been suggested that other cold-cured resin materials offer greater stability for prolonged splinting periods (e.g., Sevriton, Ash, Dentsply AG, Milford, Del) of fixation. The material is then removed after the indicated stabilization period and the teeth are smoothed with pumice. Care must be taken during removal of the acrylic material from the involved teeth because they will still be fairly mobile after the stabilization period and may accidentally be extracted.

A modification of this technique involves the use of a wire instead of a composite bridge to splint the traumatized teeth[154] (Fig. 13-38). The wire should be of proper stiffness to provide rigid fixation (24 gauge) or semirigid fixation (28 gauge), as indicated by the injury. The etch wire composite splint is aesthetic, hygienic, and quick to construct. This technique is useful with missing teeth or in a mixed dentition in which teeth are not fully erupted and the edentulous area has to be spanned (Fig. 13-39*A*). Metal bars, nylon lines, fiberglass, polycarbonate, or synthetic fibers can be used to span the space and fuse with the composite material. This type of splint should be used with all types of dentoalveolar trauma, luxation injuries, root fractures, autotransplantation, and alveolar fractures in which good stabilization can be obtained.

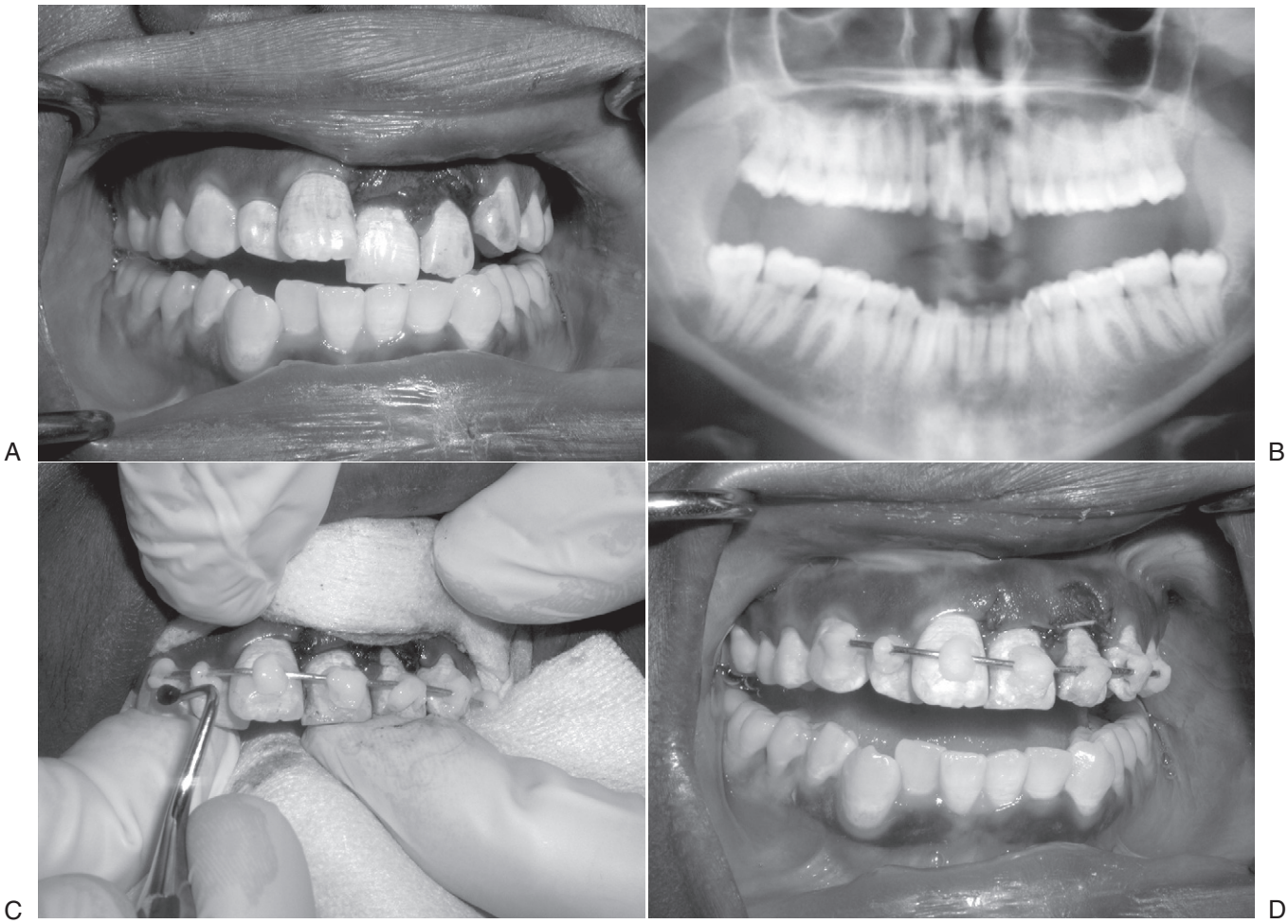


FIGURE 13-38 **A,** Dentoalveolar fracture involving the maxillary incisors and alveolar bone, requiring a period of rigid fixation for 4 weeks. The patient did not seek treatment for 24 hours following the trauma. **B,** Panoramic radiograph reveals the extent of the trauma and degree of extrusion of the involved teeth. **C,** The labial incisors of the involved teeth to be used as an abutment are cleansed of blood and debris. The cotton roll is placed in the vestibule and over the gingival lacerations. The teeth are air-dried and a 35% phosphoric acid gel is applied to the facial surfaces of the teeth for approximately 20 seconds. The composite resin and rigid wire (24 gauge) are then introduced and teeth are manipulated into position with digital pressure. **D,** After the resin has been cured, any excess material is removed and polished. The occlusion is checked and modified to resolve any potential traumatic occlusion. Lacerations should also be closed at this time. (Courtesy Dr. Kevin Stanton, New York.)

The only teeth that might not be suitable for this type of splint are those with artificial crowns or large fillings because they cannot be etched and composite material bonded to the surface.

The wire is cut and modified to lie passively along the facial aspect of the teeth to be splinted. The facial surfaces are prepared by the acid etching method described earlier. The wire or line should be placed passively and the patient asked to bite gently into a bite block of wax or similar material to force the avulsed tooth gently into the socket site. The wire is then bonded to the teeth with a composite resin restorative material. Light-activated composite resin systems allow flexibility and control of the involved segments. The wire can first be secured to the anchor teeth with composite resin and activated with the ultraviolet light. The involved teeth can then be individually repositioned correctly and bonded to the wire. The occlusion is checked for interferences and the composite material is smoothed. A wire splint can also be fixed to the teeth with orthodontic brackets and square or round stainless steel wires. The wire provides excellent stabilization and clear gingival margins and interproximal areas to allow hygiene in these areas. A radiograph should be obtained following reduction and stabilization to verify proper alignment of the teeth and alveolar segments. Cold-cured material may provide greater stability but the bond strength may be compromised because of a longer setting time, especially in the presence of traumatized tissues in which field control can be difficult. In an emergency room setting, a flowable, light-cured composite resin and wire are often the most effective for splinting teeth.

Semirigid Splint

If there are no associated alveolar fractures, a semirigid splint that allows physiologic movement of the traumatized tooth is indicated.[112] Andreasen[7,134] has shown that rigid splinting of reimplanted mature and autotransplanted

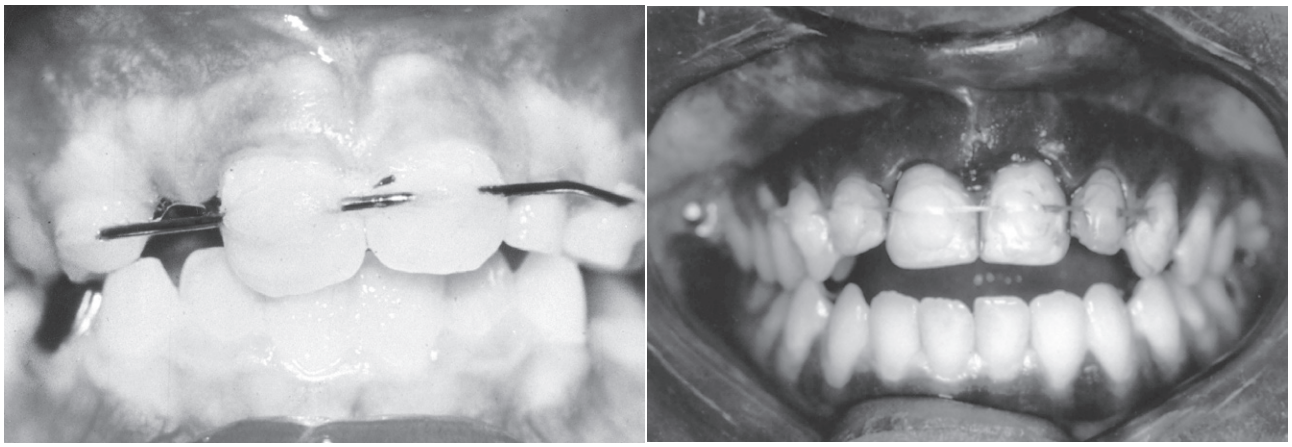


FIGURE 13-39 **A,** An acid-etched wire composite splint provides rigid fixation when a 24-gauge wire is used when there are missing teeth or in a mixed dentition in which the teeth are not fully erupted and the edentulous space has to be maintained. **B,** To allow physiologic movement of the involved maxillary central incisors, the teeth are fixed semirigidly with dental floss to the stable abutment teeth. The teeth are all prepared in the usual fashion with an acid etch technique and the dental floss is secured to the teeth in order from canine to canine. The involved teeth are usually removed from traumatic occlusion (not required in this case because of the existing anterior open bite), and the patient receives a soft diet.

immature teeth increases the incidence of root resorption because of uncontrolled forces on the tooth, periodontal fibers, and lamina dura; there are negative influences on pulp revascularization and periodontal ligament healing, with an increased incidence of pulp necrosis and canal obliteration. The acid etch composite resin technique with materials such as waxed dental floss, suture,[63] flexible braided orthodontic wire,[73] or monofilament nylon line provides stabilization of the traumatized tooth and allows physiologic movement of the tooth under function to minimize ankylosis and root resorption. Various other fixation materials have been evaluated with respect to flexibility. Kevlar (B-W Dental, Frederiksberg, Denmark) and fiber splints allow more horizontal movements, with Protemp (Protemp II, Espe, Germany) and flexible wire composite splints providing adequate lateral support and vertical flexibility, which improve periodontal healing of luxated teeth.[157]

The involved tooth and two stable adjacent teeth are acid-etched to provide support (see earlier). A single strand of 20- to 30-lb test monofilament nylon line is cut to size and secured to the first abutment tooth with a composite resin system. The remaining abutment and traumatized teeth are secured to the nylon line with the composite resin system as tension is maintained with hemostats on the free end of the nylon line. The resin is then smoothed and polished after all teeth are secured, and the occlusion is verified (see Figs. 13-27 and 13-39).[158] A radiograph should be obtained to check for proper root position after splinting.

Single-tooth fixation techniques include endosteal (transdental) pin fixation, amalgam splints, and the Maryland type of etched cast framework, which generally should be considered unacceptable fixation for most clinical situations. Unacceptable splints include the self-curing, circumferential, Essig, arch bar, intracoronal, and orthodontic splints. Recommended splints include the dental floss, suture, resilient, clear resin, Hawley with incisal spring, and nylon line splints. Other variations are

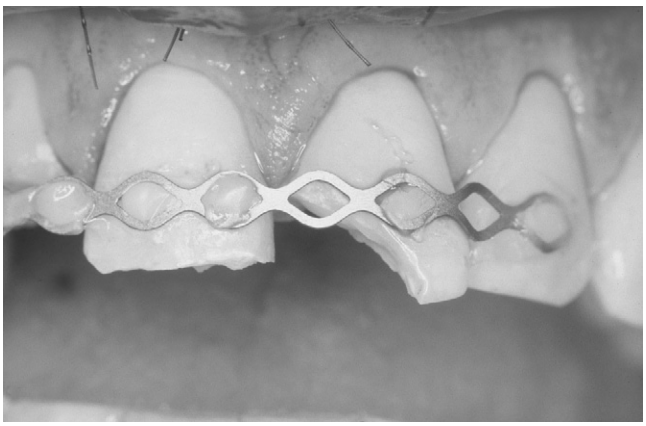


FIGURE 13-40 Titanium trauma splinting bars. (Courtesy Dr. Andreas Filippi, Department of Oral Surgery, Oral Radiology and Oral Medicine, University of Basel, Basel, Switzerland.)

commercial splints such as the Anterior and Posterior Splint-Grids, Splint-Lock System, and Titanium Splinting Bars. Titanium trauma splinting (TTS) bars have been advocated by some practitioners for splinting teeth because of ease of application with light-cured composite resin and removal, better patient compliance with oral hygiene, and fewer patient complaints with alterations in masticatory function.[4] However, oral hygiene effectiveness was similar among patients splinted with conventional wire-composite splints and those splinted with TSS.[154,159] The practicality and clinical use of these materials remain to be seen (Fig. 13-40).

RESTORATION

Following dentoalveolar trauma, the normal form and function of the masticatory system should be restored by the restorative dentist. Restorations for crown injuries include composite acid etch buildups, porcelain veneers, and full coverage crowns.

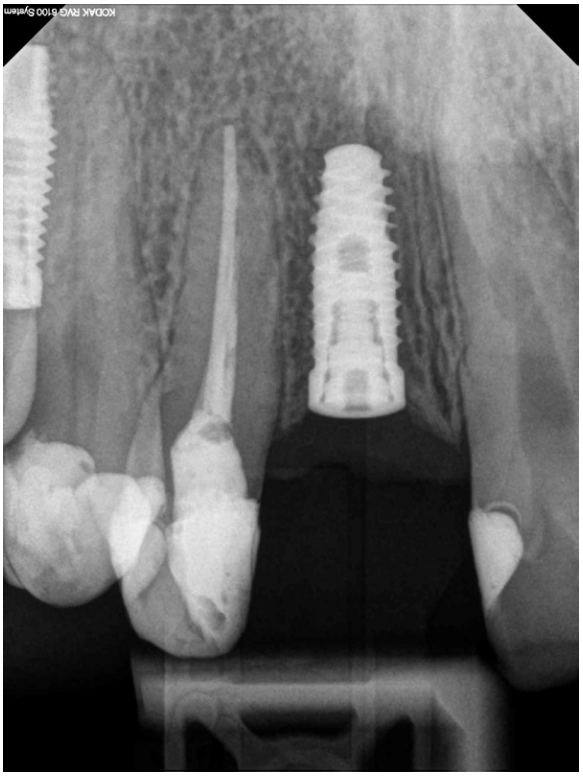


FIGURE 13-41 Restoration of function following dentoalveolar trauma and loss of teeth can be accomplished with various implants or implanted materials, such as autogenous and allogeneic graft materials.

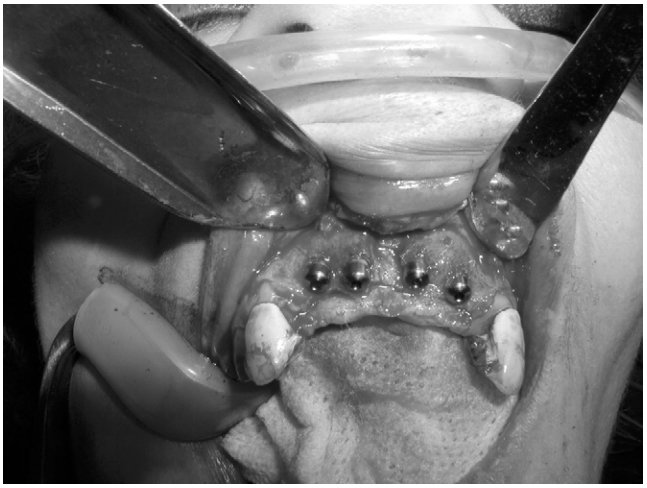


FIGURE 13-42 The use of dental implants, especially in the aesthetic zone of the maxillary anterior teeth, should be considered as the required reconstruction of patients suffering dentoalveolar injury. Four implants have been placed in the maxillary anterior region following traumatic loss of the maxillary anterior teeth and alveolar bone in this patient.

Crown-root fractures are usually restored, after endodontic therapy, with a customized cast core cemented into the retained root and coverage with a full crown.[7] Alveolar process fractures can be malaligned after healing, resulting in malocclusion and occlusal cant. Orthodontic treatment and leveling of the occlusal plane with restorations (crowns, veneers) have been shown to be effective.[160]

With the loss of the dentition, supporting alveolar bone, or both, various dental implant systems can be used to support a bridge and restore function.[161] If large amounts of alveolar bone have been lost, especially if the facial osseous plate has been avulsed, bone grafts may be necessary to provide adequate osseous support for the implants (Figs. 13-41 and 13-42). Long-term effects of dentoalveolar trauma can also include inadequate space in the dental arch. Orthodontic treatment may be required; the use of skeletal orthodontic anchorage devices have facilitated treatment, with more rapid results.[162]

MOUTH PROTECTORS

The most frequent of all anesthesia-related medicolegal claims are dental injuries because they often occur from excessive pressure on the incisors while using a laryngoscope.[163] Damage to the incisors and dentoalveolar structures was found to comprise 33% of all anesthetic claims; the use of preanesthetic evaluation and prophylactic measures is recommended to prevent damage to the incisors and dentoalveolar structures.[38] Preanesthetic dental evaluation and the use of mouth protectors in patients undergoing tracheal intubation for general anesthetic have been shown to reduce the incidence of dental trauma.[42-44] The use of flexible fiberoptic-lighted stylets or nasopharyngeal airways may help reduce the risk of dental injury.[50] Seat belts, shoulder harnesses, air bags, and padded dashboards in automobiles have been advocated for reducing injuries in MVAs, including facial and dental trauma as the victim hits the steering wheel, dashboard, or windshield during impact. Preventive orthodontics should be considered for children with increased maxillary overjet as a precaution against traumatic injury to the exposed teeth.[7]

The use of intraoral mouthguards has been demonstrated to be an effective means of reducing the degree and incidence of dentoalveolar trauma associated with contact sports.[164-173] There is an apparent awareness by athletes in high-risk sports for possible dentoalveolar injuries, but there is considerable lack of the use of mouthguards by athletes.[19] In the United States, participation in youth athletic programs continues to increase by substantial numbers each year, with a greater emphasis placed on injury prevention and rehabilitation. Studies have reported that 33% of all dental trauma occurs in sporting activities.[174,175] In one 10-year study, 31% of craniomaxillofacial trauma was attributed to sporting activities.[102] In 1960, a joint commission of the Bureau of Dental Health Education and the American Association for Health, Physical Education, and Recreation reviewed the literature on football injuries and found that when high school football players did not wear face masks or mouth protectors, 50% of all their injuries occurred around the mouth. The committee concluded that most of these injuries could be prevented by a properly fitted mouthguard.[172] Even a simple boil and bite mouthguard

BOX 13-1 National Collegiate Athletic Association Guideline for Mouthguards

The NCAA has mandatory equipment rules, including the use of mouthguards for selective sports. Various studies of properly fitted mouthguards have indicated that they may reduce dental injuries and mild traumatic brain injuries (concussions) when blows to the jaws or head are received.

The American Dental Association has urged the mandatory use of mouthguards for those engaged in athletic activities that involve body contact and has endorsed their use in sporting activities in which a significant risk of oral injury may occur. When considering the optimal protection for an athlete, it is important that a thorough medical history be taken and the demands of his or her position and sporting activity be considered.

Specific objectives for the use of properly fitted mouthguards as protective devices in sports are as follows:

- Properly fitted mouthguards could reduce the potential chipping of tooth enamel surfaces and reduce fractures of teeth, roots, or bones.
- Properly fitted mouthguards could protect the lip and cheek tissues from being impacted and lacerated against tooth edges.
- Properly fitted mouthguards could reduce the incidence of a fractured jaw caused by a blow delivered to the chin or head.
- Properly fitted mouthguards could provide protection to toothless spaces, so support is given to the missing dentition of the student-athlete.

Stock, mouth-formed, and custom-fitted mouthguards are three types recognized by the American Dental Association. All need to be fitted properly for maximum protection. Student athletes should be advised about which properly fitted mouthguard is best for them and how it is best maintained to ensure maximum fit and protection for daily practices and game-day wear. Medical staff personnel should regularly oversee and observe the student athletes and the properly fitted mouthguards.

To realize fully the benefits of wearing a mouthguard, the coach, student athlete, and medical staff need to be educated about the protective functions of a mouthguard and the game rules regarding mouthguard use must be enforced.

Adapted from National Collegiate Athletic Association: *Sports medicine handbook: Guideline 4c: Mouthguards*, Indianapolis, 2007, National Collegiate Athletic Association.

has been shown to reduce the incidence and severity of orofacial injuries by 50%.[176]

In 1962, the National Alliance Football Rules Committee required high school football players to use mouthguards and face guards.[169] They stated that "each player shall wear an intraoral mouth and tooth protector, which includes both an occlusal (protection and separation of the biting surfaces) and labial (protection of the lip) portion."[177,178] The most recent National Collegiate Athletic Association (NCAA) guidelines regarding mouthguards, revised in August 2007, have defined the use of "properly fitted mouthguards" (Box 13-1). In 1967, it was estimated that 25,000 to 50,000 injuries were prevented by the use of intraoral mouth protectors.[179] Studies carried out with the Notre Dame football program—when faceguards were mandatory but mouth protectors were seldom worn or accepted on a team basis—indicated that more than one of five injuries to football players were to the hard and soft tissues of the oral cavity.[164,180] The NCAA Football Rules Committee recommended the mandatory use of mouthguards in 1970, and the mouthguard rule went into effect with the 1973 football season. In the 1970 season, 280 cases of oral injury requiring treatment were reported. With a mandatory rule for mouthguard protection in the 1973 season, the reported number of cases decreased to 55.[170]

Currently, organized football programs at some elementary and junior high schools, and backyard and sandlot football, are the only levels of the sport not covered by mandatory mouthguard rules. If well-accepted injury figures are conservatively projected, more than 200,000 injuries are being prevented annually. The incidence of mouth injuries among football players in 1981 was between 0.35% and 0.45%.[169] Studies have suggested that the prevalence of dentofacial injuries to football players is higher than previously recorded. The Academy for Sports Dentistry was formed in 1983; it conducts studies on the incidence and prevention of sports injuries, promotes the wearing of mouthguards in more than 40 sports, and encourages the team dentist concept.[181]

Enforcement of the mandatory mouthguard rules is difficult because many officials report that they cannot determine whether a player is using a mouthguard if the material is transparent.[167] Athletes occasionally are reluctant to use a mouthguard because they believe that the guards are troublesome, prevent adequate breathing and speaking, cause nausea, are expensive and, overall, are unaesthetic. There has been an increased use of mouthguards with the incorporation of the team's colors on the guards. The NCAA modified the mouthguard rule for the 1990 football season to require that all mouthguards be yellow to facilitate their visualization by the officials on the playing field. Research at the University of Michigan has revealed that yellow mouth protectors are easier to detect at various distances than clear mouth protectors or none at all.[182] It now appears that the mandatory mouthguard rule is not being enforced at the NCAA division I football level, with an alarming number of players, especially quarterbacks, not using a mouth protector or wearing it on the side of their helmet during the game. Enforcing the use of mouthguards should be the responsibility of coaches and trainers, not officials or referees during games. Compliance should be enforced at all times during practice, when the chance of dental injury is equal to or greater than that found during the actual game.

Currently, mouth protectors are required only in organized football, ice hockey, field hockey, lacrosse, and boxing matches.[183,184] Mouth protectors are recommended for most sports such as wrestling, rugby, soccer, basketball, gymnastics, racquetball, martial arts, skiing, and weight lifting to minimize tooth injury and supporting structures through direct or indirect trauma from falls, collisions, or clenching of the teeth.[185] Several studies have confirmed the decrease in football and other sports injuries when a mouthguard was worn. The participation of girls and women in competitive athletics

has increased dramatically. The incidence of injuries to female athletes has been found to be similar to that of male athletes involved in the same athletic activity.[186] Female athletes should be educated and encouraged by coaches and officials to use mouth protectors to prevent dental injury.[169]

A properly constructed and used mouthguard should do the following[163,175,176]:

- Hold the lips and cheeks away from the teeth, preventing laceration or bruising.
- Cushion teeth from direct blows and redistribute the forces.
- Prevent opposing teeth from coming into excessive contact.
- Provide the mandible with resilient support to absorb impact.
- Provide protection against neck injuries.
- Assist with the prevention of concussion and resultant neurologic injuries.

Mouthguards may be divided into three types: (1) ready-made or stock protectors; (2) mouth-formed protectors; and (3) custom-made protectors.* The most desirable qualities of a mouthguard are retention, comfort, allowance for ease of speech and breathing, resilient material, and provision of protection for the teeth, gingiva, and lips. Mouthguards that are not stock varieties are usually fabricated for the maxillary arch. For the athlete with a class III malocclusion, the mouthguard should be manufactured to cover and protect the prominent mandibular teeth.[168] A properly fitted mouthguard must be protective, comfortable, retentive, resilient, tear-resistant, odorless, tasteless, inexpensive, and easy to fabricate and should not encroach on the airway or interfere with breathing or speech.[178]

For maximal protection, the mouthguard should be designed and constructed according to the following specifications[183,192]:

- The occlusal surfaces of all teeth are covered for protection and to prevent possible overeruption of teeth.
- The flanges cover the alveolus and are trimmed ⅛ inch (2 mm) short of the depth of the buccal vestibule for maximal retention and to protect the lip and gingiva.
- The material extends distally to include the second molars and is relieved for clearance of the frena.
- The material extends lingually no more than ¼ inch onto the palatal mucosa, tapering at the margins to prevent lingual bulk, which may interfere with speech and breathing or trigger a gag reflex.
- The edge of the labial flange should be rounded in a cross section.

Mouthguards have changed over time from vacuum-formed mouthguards to two-layer ethylene vinyl acetate (EVA) mouthguards fabricated on a high-pressure machine. The EVA mouthguards' main advantages are that they fit better and have better protection because of improved impact absorption.[193] The thickness of the mouthguard at the incisal edge and cusp tips should ideally be 4 mm because of the stability of EVA (Shore A hardness, 80). A thickness of less than 4 mm has decreased energy absorption potential and more than a 4-mm thickness has a marginal increase in absorption potential but less wearer comfort because of the bulkiness of the mouthguard.[191,194] However, Duhaime and colleagues[195] have reported that it might be possible to construct a thinner EVA mouthguard that provides protection equal to that offered by those currently in use.

A number of plastic materials have been used for the construction of mouthguards. Current materials used include EVA, polyvinyl acetate–polyethylene copolymer, polyurethane, polyvinyl chloride, soft acrylics, and natural rubber. The various materials' high-energy absorption properties do not necessarily indicate that the material will provide maximal protection as a mouthguard, and some of the absorbed energy may be directly transmitted to the underlying dentoalveolar structures.[191,194]

TYPES OF MOUTH PROTECTORS

Stock Mouth Protectors

The stock mouth protector is the simplest and cheapest type of mouthguard (Fig. 13-43). It is purchased over the counter and does not require fitting. These mouthguards are constructed of latex or silicone and provide minimal protection because they are loose-fitting and can be held in place simply by keeping the teeth together and the jaws closed. The stock type of protector is uncomfortable to the participant because it is difficult to breathe and speak around the mouthguard and it tends to irritate the gingiva and buccal vestibule. There is no evidence that these mouthguards are effective in redistributing forces of impact, and they may result in soft tissue injury. Thus, the Academy for Sports Dentistry has declared that typical stock mouthguards are not acceptable.[164,169,183,187-190]

Mouth-Formed Protectors

The mouth-formed protector is probably the most popular and universally used mouthguard.[164] There are two major types of mouth-formed protectors, the shell-liner type and the thermoplastic type (Fig. 13-44).[171,183,189]

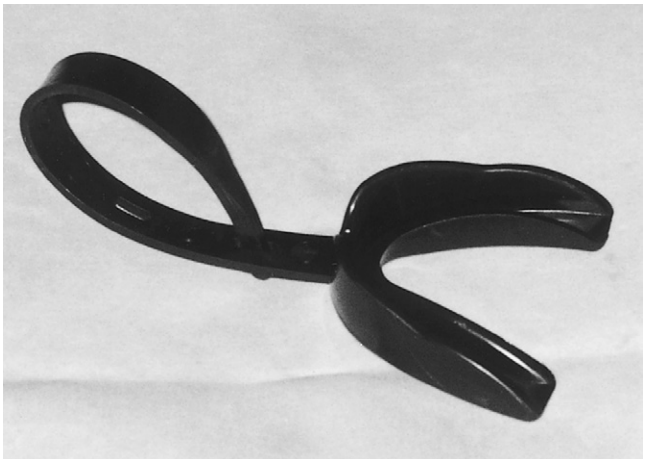
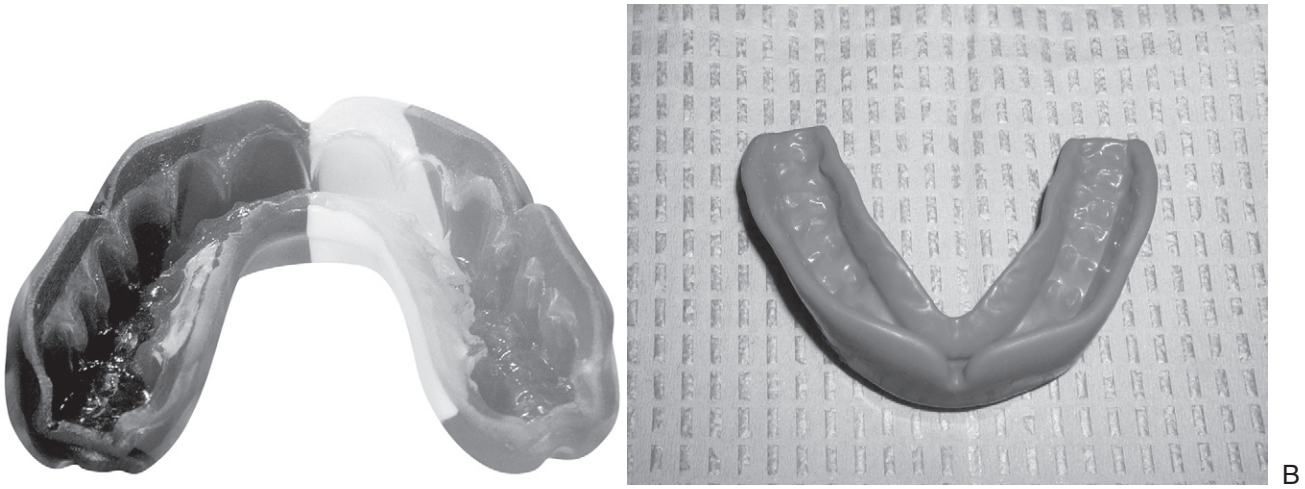


FIGURE 13-43 Ready-made or stock mouth protector.

*References 164, 169, 172, 183, and 187-191.



A B

FIGURE 13-44 Two major types of mouth-formed protectors, **A,** Shell-liner mouth-formed protector. **B,** Thermoplastic mouth-formed protector. (**A,** Courtesy Glidewell Laboratories, Newport Beach, Calif.)

Shell-Liner Mouthguard. The shell-liner mouthguard (see Fig. 13-44*A*) consists of a preformed outer shell of latex rubber or plastic that fits loosely around the maxillary teeth. It provides a smooth durable surface and soft resilient lining that is adapted to the teeth. The tray tends to be bulky with sharp edges but can be trimmed for comfort and improved fit in the vestibule. The shell is then relined with a polymer-monomer soft-curing material similar to that used for soft relining of dentures, self-curing methyl methacrylate, or silicone rubber. The materials are mixed, placed in the outer shell, and carried to the mouth to set in 5 to 7 minutes.* The disadvantages of this type of protector limit its usefulness. Repeated biting on the relining material causes it to spread and lose retention of the teeth. The liner can gradually become hard because it is continually exposed to oral fluids. The protector tends to be bulky and opens the bite if not properly fitted. This type of protector is not recommended for athletes with orthodontic braces or other intraoral appliances.

Thermoplastic Mouthguard. The thermoplastic type of mouth-formed protector (see Fig. 13-44*B*) is the most widely used mouthguard because it is cheap and durable and can be resoftened and refitted if it loses retention. The mouthguard is a thermoplastic shell of polyvinyl acetate ethylene that is immersed in boiling water for 30 seconds, dipped quickly in cold water, and placed in the mouth over the maxillary teeth. Suction develops in the patient around the mouthguard to mold it into shape around the teeth and dentoalveolar structures. The material is then adapted to the teeth with the fingers. The vestibule extension can be trimmed and molded for comfortable coverage of the alveolus and soft tissue. The teeth can be occluded into the material while it is warm to provide minimal opening of the closed bite.[164,169,189] The major advantages over the stock trays are a closer fit and greater retention; however, a quality thermoplastic mouthguard is technique- sensitive, and care is necessary during the molding process. Other advantages include a low bulk of material and relative ease of fabrication. Disadvantages of the thermoplastic mouthguard include possible distortion over time and hardening of the material with continued contact with oral fluids[175] (Fig. 13-45).

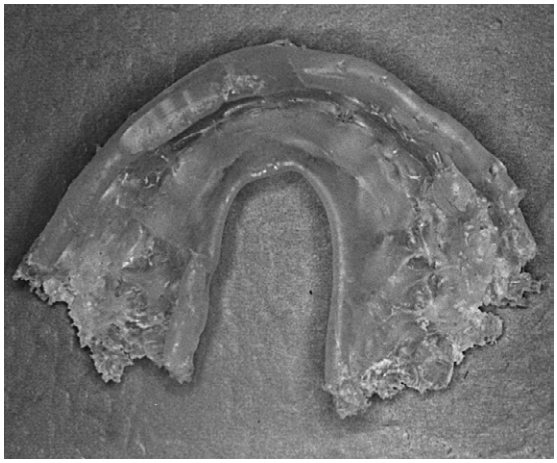


FIGURE 13-45 Thermoplastic mouthguard showing distortion and hardening of the material. (From Pinkham JR: *Pediatric dentistry: Infancy through adolescence*, ed 3, Philadelphia, 1999, WB Saunders.)

Bimaxillary Mouthguard

Another type of mouth-formed protector covers both arches and is known as the bimaxillary mouthguard (Fig. 13-46). The position of the mandible is fixed at a predetermined, relaxed, maximal breathing position that allows the athlete to maintain adequate ventilation. The mouthguard also rotates the position of the condylar head within the glenoid fossa to help reduce the transmission of forces to the glenoid fossa and cranium, which protects the athlete from concussion-type injuries and from injuries to the mandibular condyle.[176] The anterior mandibular teeth are also protected from frontal impact.

*References 54, 169, 183, 189, and 190.

Custom-Made Mouth Protectors

The custom-made protector (Fig. 13-47) has the best mouthguard qualities when compared with all other types of mouthguards, including retention, protection, taste, lack of odor, comfort, allowance of speech, cleanliness, and retention.[196] However, there is no evidence that they are more effective in preventing injuries. A study that compared rebound in custom-fabricated mouthguards versus commercial mouthguards has found that there is greater rebound with all the commercial mouthguards when compared with the custom mouthguard.[197] A custom-made mouthguard is fabricated in a laboratory to a stone model made from an alginate impression of the athlete's maxillary teeth. A thin sheet of thermoplastic material, commonly polyvinyl-polyethylene with a thickness of 1/16 to 1/8 inch,[189] is adapted to the model to construct a comfortable, individualized mouth guard for protection. There is no excessive bulk, so the athlete can breathe, speak, and be understood and not have to hold the mouthguard continually in position with the teeth or cheeks. The interocclusal thickness remains thin and uniform to allow even occlusion when the athlete bites into the protector. Excellent retentive properties add to its comfort and do not cause the wearer to take the mouthguard out of his or her mouth continually, which would increase the chances of soiling or misplacing it.[164]



FIGURE 13-46 Pro Air mouthguard, a bimaxillary mouthguard. (Courtesy Brain-Pad, Conshohocken, Pa.).

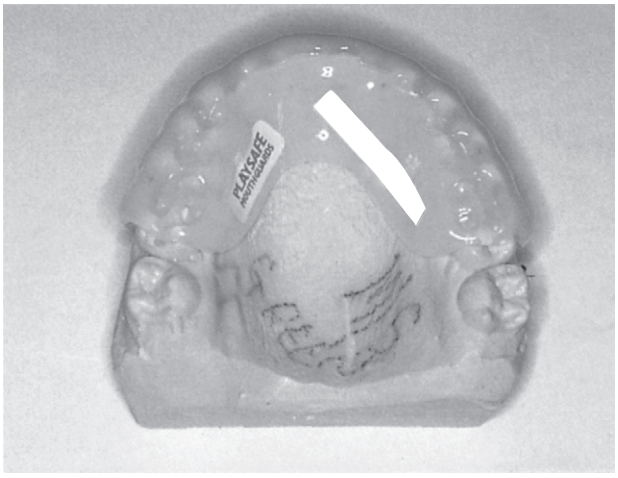
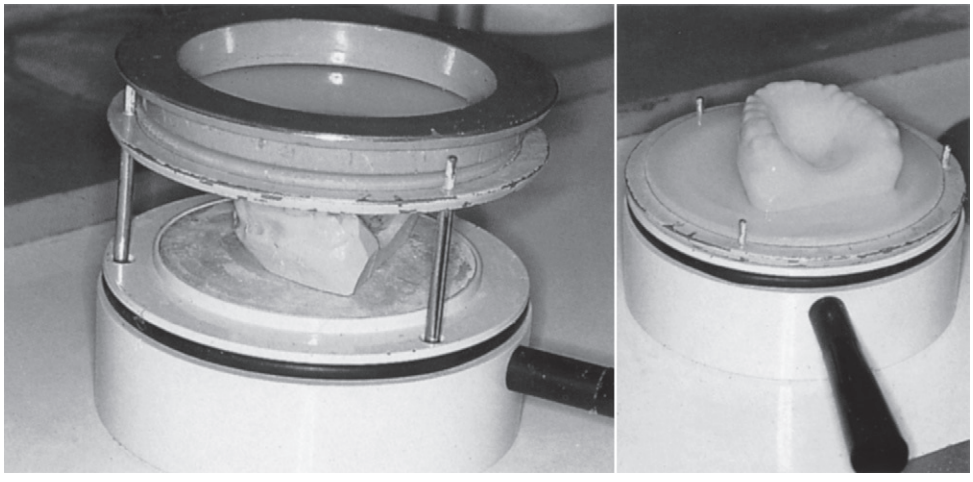


FIGURE 13-47 A custom-made mouth protector. (From Andreasen JO, Andreasen FM: *Textbook and color atlas of traumatic injuries to the teeth*, ed 3, Copenhagen, 1994, Munksgaard.)

After the stone model has been made from an alginate impression, a thin sheet of thermoplastic material is heated and a vacuum is formed over the model to cover all the teeth (Fig. 13-48). The vacuum method of adapting the material produces a guard of uniform thickness and maximal retention around the natural contours of the teeth.[189] The material is allowed to cool completely on the model to prevent distortion.

The mouthguard is then trimmed from the palate and 1/8 inch short of the buccal vestibule, with clearance for the buccal and lingual frena (Fig. 13-49). The edges are smoothed and rounded with a stone wheel or are placed on the model and flamed with an alcohol torch and adapted with the fingers.[166,189] The athlete's name should be secured to the mouthguard for identification and, if desired, a strap can be added (Fig. 13-50). The stone models are kept by the coach, trainer, or dentist so that a replacement mouthguard can be made if necessary. The disadvantages of the custom-fitted mouthguard are



A B

FIGURE 13-48 A thin sheet of thermoplastic material is heated **(A)** and vacuum is formed over a stone model of the patient's teeth **(B).** (From Andreasen JO, Andreasen FM: *Textbook and color atlas of traumatic injuries to the teeth*, ed 3, Copenhagen, 1994, Munksgaard.)

the time and expense involved with management, construction, and delivery of the protector. Materials such as Light Line (LD Caulk [urethane diacrylate], Dentsply) have been used to provide additional comfort because the material softens intraorally.[196]

A mouthguard must be easy to fabricate, comfortable, able to accommodate the needs of an individual's dentition, durable, easily held in place, and able to provide adequate protection to the teeth, jaws, and cranial structures. Regardless of which mouthguard is used, the athlete should be instructed to rinse the protector under cold tap water, a mouthwash, or 0.2% chlorhexidine rinse and wash with soap after each use. It should then be stored in a rigid perforated container.[189]

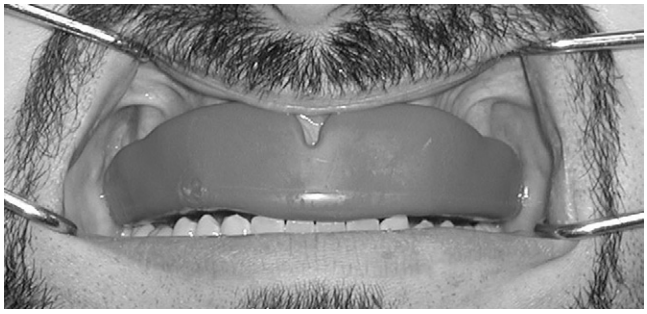


FIGURE 13-49 The mouthguard should extend to ⅛ inch short of the depth of the buccal vestibule, with clearance of the frena.

The dentist should be involved in the prevention of sports-related injuries with preseason screenings, availability for emergency trauma care, and fabrication of custom-fitted mouthguards.[178] Unfortunately, apparently some school officials believe that dentists are willing to assist with oral protection only if custom-fitted mouthguards are used. Regardless of the type of protector used, players will benefit if a dentist is available to offer advice on its fit and form and on the health of the player's mouth. The thermoplastic variety of mouthguard is recommended if it is to be placed or formed by the athlete; however, supervision of the fitting procedure by a dentist is recommended. The most desirable protector is the custom-made mouthguard, fabricated by a dentist from a thermoplastic material.[169] Mouthguards are also recommended for patients who suffer self-inflicted injuries, such as mental retardation in Lesch-Nyhan syndrome, comatose patients, and patients in whom dentoalveolar-intraoral injuries are caused by involuntary movements, as in Parkinson's disease. There is sufficient evidence to suggest that a correctly made mouthguard considerably reduces the severity of oral injuries. Prevention of dental trauma requires educational campaigns to broaden the lay public's knowledge of emergency management procedures (Fig. 13-51). The goals of these campaigns should include the following: (1) raising public awareness; (2) emphasizing the necessity for cooperation between the emergency and private dental services; and (3) emphasizing the necessity for adequate emergency treatment.[198]

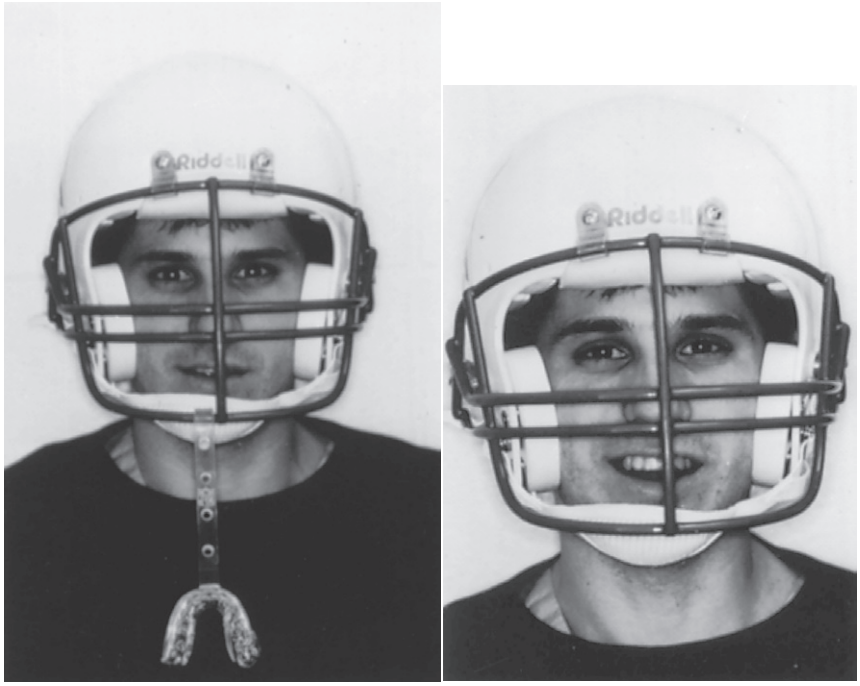


FIGURE 13-50 **A,** A thermoplastic mouth-formed or custom-made mouthguard may have a strap attached to the helmet. The strap secures the mouthguard to the facemask and allows the athlete to remove the mouthguard during breaks in competition without misplacing it or soiling it on the ground. **B,** Enforcement of a mandatory mouthguard rule is difficult if the mouthguard material is transparent or has no strap to the helmet.



FIGURE 13-51 The burden of educating patients regarding the importance of oral health and prevention depends on our professions. Continued awareness of this can be shown by making April National Facial Protection Month and the endorsement from the American Academy of Pediatric Dentistry, American Association of Orthodontists, and American Association of Oral and Maxillofacial Surgeons.

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CHAPTER

14

Mandibular Fractures

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OUTLINE

Historical Considerations
History of Treatment
Demographics and Epidemiology
Causes
Location of Mandibular Fractures
Facial Fractures Associated With Mandibular Fractures
Number of Fractures Per Mandible
Nonmaxillofacial Trauma Associated With Mandibular Fractures
Classification of Mandibular Fractures
Dictionary Classification
Classification by Anatomic Region
Diagnosis of Mandibular Fractures
Patient History
Clinical Examination
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General Principles of Treatment
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Treatment of Mandibular Fractures
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Complications of Mandibular Fracture Treatment
Infection and Teeth in the Line of Fracture
Other Complications

HISTORICAL CONSIDERATIONS

Management of mandibular fractures has continued to evolve since on of the earliest descriptions in Egyptian literature. The first described case in 1650 BC, discussed the examination, diagnosis, and treatment of mandibular fractures and other surgical complications. Treatment options were limited and quite often the condition led to the demise of the patient.[1,2]

It was not until the advent of the Hippocratic concept for reapproximation and immobilization that management of mandibular fractures was revolutionized. Hippocrates described direct reapproximation of the fracture segments with the use of circumdental wiress, similar to today's bridle wire. He advocated wiring of the adjacent teeth with external bandaging to immobilize the fracture.[3,4] Hippocrates had the insight to realize that reapproximation and immobilization are paramount in the treatment of mandibular fractures.

In 1180, a textbook written in Salerno, Italy, described the importance of establishing a proper occlusion.[5] In 1492, an edition of the book *Cirurgia,* printed in Lyons, France, first mentioned the use of maxillomandibular fixation in the treatment of mandibular fractures.[6] In 1795, Chopart and Desault described the effects of elevator and depressor muscles on mandible fragments.[7] Chopart was also the first to use dental prosthetic devices in an attempt to immobilize fracture segments (Fig. 14-1).

Advancements for treatment stagnated, through the nineteenth century. Emphasis remained on some form of external bandage or wrap occasionally used in conjunction with a bridle wire. Attention was being directed toward the development and improvement of intraoral and extraoral devices.

Various techniques were advocated however, most were variations of bandages and external appliances, extraoral and intraoral appliances, monomaxillary wiring, including bars, monomaxillary splints, and intermaxillary wiring and splints, guides or glides, and internal fixation, including wires, plates, and screws. A brief overview of these techniques are discussed below.

HISTORY OF TREATMENT

Bandages and External Appliances

Bandages, first mentioned by Hippocrates,[3] gained notoriety and acceptance as a standard of care when John Rhea Barton described his Barton bandage[15] (Fig. 14-2). This bandage provided posterior directional forces on the fractured mandible, resulting in deformities such as the "bird face" and malunions.[16,17]

External or extraoral appliances also failed to provide stable reduction of fractures. These appliances had chin



FIGURE 14-1 Chopart and Desault (1795) used viselike devices to treat mandibular fractures.



FIGURE 14-2 The Barton bandage, named for John Rhea Barton.

cups and an assortment of straps and external supports that like the bandages, produced an undesirable posterior directional force.[18-25]

Extraoral and Intraoral Appliances

A primitive approach combined a rigid splint on the occlusal surface of the teeth and one on the undersurface of the mandible. This viselike device was then used to apply pressure to the two splints, theoretically immobilizing and fixating the fractured segments. Dorrance and Bransfield[26] wrote about the multiple disadvantages of this method, however, including the inability to gain proper occlusion, its inapplicability for fractures of the posterior mandible and for bilateral fractures, its promotion of drooling, and the lack of immobilization necessary for proper union.

Gunning[27] was the first to use a custom-fitted intraoral dental splint for immobilization. He used the splint in conjunction with an external head appliance, and these splints could also be applied to the maxilla and mandible, resulting in IMF (Fig. 14-3). An anterior space was provided for nourishment.[27-29] This basic principle, of using splints for intermaxillary fixation, although modified, is still used routinely in the treatment of edentulous and partially edentulous mandibular fractures.

Monomaxillary Wiring, Bars, Arches, And Splints

Originally advocated by Hippocrates,[3] and then later supported in the writings of numerous authors,[30-33] monomaxillary wiring was popular in the treatment of mandibular fractures. It was similar in concept to today's bridle wire and the Risdon wire of the early 1920s, and monomaxillary wiring afforded some element of reduction, but without supplemental fixation, fractures were inadequately immobilized.

Splints became popular in the middle of the nineteenth century and were usually cast of metal and custom-fitted for the patient. Splints were fabricated with the use of models made after reduction of the fragments. Some were cemented to the teeth and others involved sophisticated clamps that would tie buccal and lingual segments together.[17,34-43] The use of monomaxillary wiring, bars, arches, and splints is limited to fractures containing stable teeth on both sides of the fracture.

Intermaxillary Wiring

Guglielmo Salicetti (William of Saliceto)[44] has been credited with being the first to use intermaxillary fixation. Gilmer,[45] also credited with being the first to use this technique, passed wires around individual teeth of both arches and then ligated these opposing wires.

Orthodontic bands and arches were used in patients with loose, fractured, or missing teeth. The famous American orthodontist Angle[46] described many methods of IMF that used bands and other orthodontic techniques. A recent study by Cousin revisited this methodology and noted success using nonwire fixation for IMF.

Sauer[47] has described the ligation of an iron wire to the teeth with fine ligature wires. However, it was Gilmer[48] who took this idea a step further and fixated full arch bars on both the mandible and maxilla, revolutionizing the treatment of fractures.

Open Reduction and Internal Fixation Using Sutures

Historically, mandibular body, ramus, or angle fractures were treated with closed reduction and intermaxillary fixation (IMF). Currently, screw and plate osteosynthesis is standard procedure to stabilize these fractures by open reduction and fixation.[8-10] A fracture of the condyle can be treated conservatively with IMF, surgically by an open

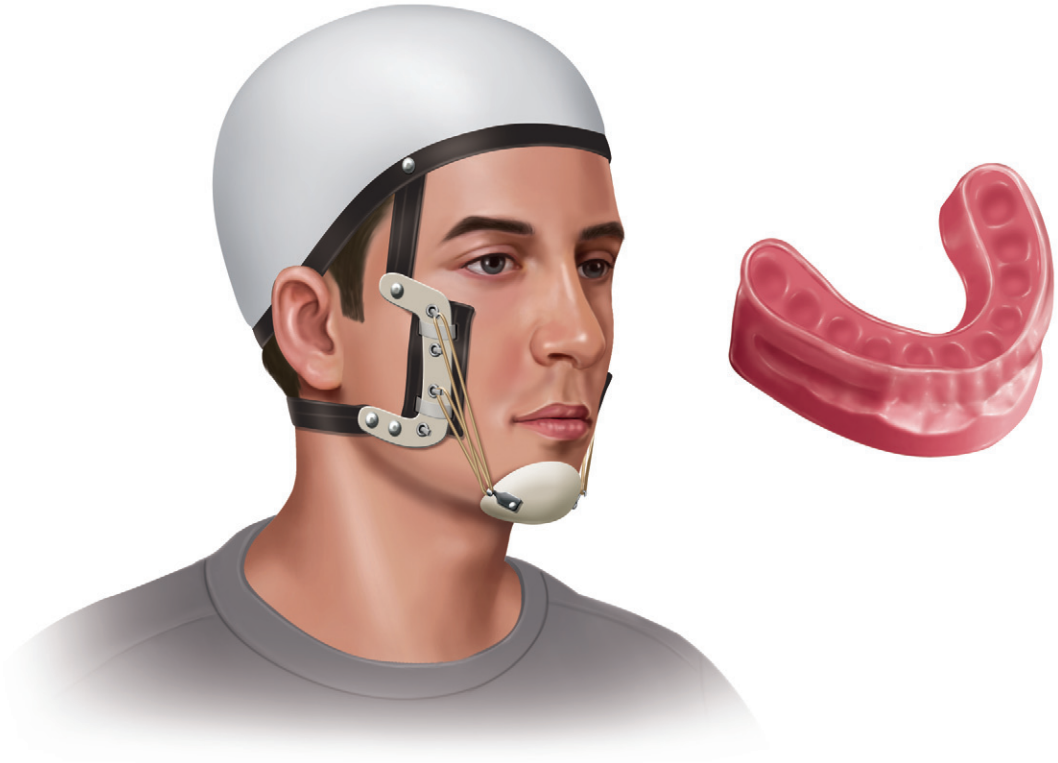


FIGURE 14-3 Thomas Brian Gunning (1840-1889) was the first to use vulcanite in a custom-fitted splint to immobilize mandibular fragments. He used double arms extraorally for anchorage to a head cap and soft rubber chin support.

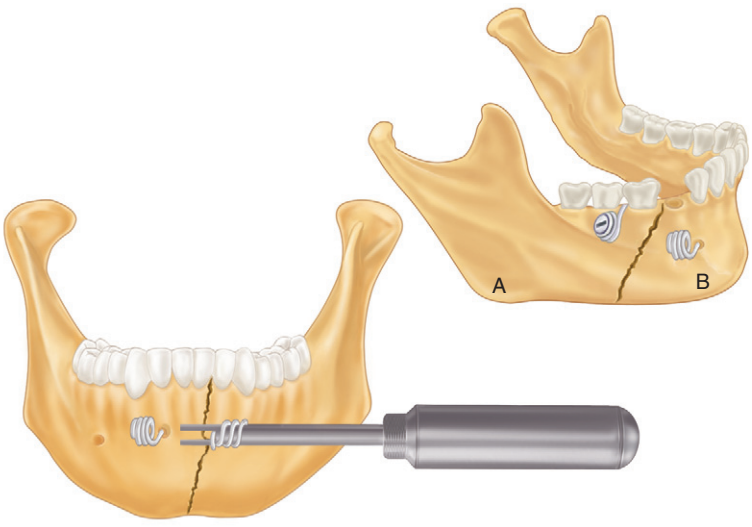


FIGURE 14-4 The Thomas principle (1869) presented a technique of intraoral open reduction with silver wire osteosynthesis. The spiral springlike termination was tightened intermittently "until union was secured." **A,** Proximal segment. **B,** Distal segment.

reduction using a submandibular, preauricular, retroauricular, or transparotid approach, or by a transoral approach or endoscopically.[11-14]

Transosseous wires

In the middle of the nineteenth century, Buck and Kinlock have described the use of wire ligature for the immobilization of mandibular fractures.[17,49] Using this method, one would drill a hole on both sides of the fracture site and then pass a wire. The wire, which was iron or silver, was then tightened periodically by creating pigtails on each end of it (Fig. 14-4). This method was hampered by a high incidence of infection.[26]

Bone Plates

In 1881, Gilmer[36] described a method of mandibular fracture fixation that used two heavy rods placed on either side of the fracture and wired together. The rods were pushed through skin, mucous membranes, and bone and were wired on both the mouth and skin sides.

Dorrance and Bransfield stated that the earliest reference to the use of true bone plates was that of Schede, who, around 1888, used a solid steel plate held by four screws.[26] During World War I, Kazanjian[50] used wire sutures through bone fragments and tied the wire to an arch bar for fixation. In 1900, Mahé[51] used multiple plates to secure multiple mandibular fragments after applying a monomaxillary splint. A similarity to the plating systems used today can be noted.

In 1915, Ivy[52] attempted the use of Sherman's steel plates but abandoned the method because of infection and necrosis. In 1917, Cole[53] used silver plates and screws on each side of the fracture and then silver wires attached to the plates to immobilize the fracture. In 1934, Vorschutz introduced two long screws through the skin into the bone, reduced the fracture, and held the screws in position with the use of a plaster of Paris bandage (similar to the Joe Hall Morris appliance of today).[54]

Biodegradable Plates and Plating Systems

Internal rigid fixation of the facial skeleton is a reliable method for obtaining osteosynthesis. Historically, metallic plates and screws have been used to allow for early passive and active function. To date, titanium plating remains the reference standard of mandibular fracture treatment because of its high biocompatibility, stability[55], resistance to corrosion[56,57], and cost-effectiveness (in comparison to resorbable plating systems).[58] However, these titanium plating systems have certain disadvantages, including migration from the original position, infection, palpability, and exposure, all which might require revision surgery, and can cause distortion in magnetic resonance imaging (MRI) and computed tomography (CT) scanning.[59] It may become necessary to remove plates because of stress protection–induced osteopenia in the cortex beneath the plate, causing a reduction in cortical thickness and shaft caliper. Others have reported that metallic alloys containing nickel, cobalt, and chromium may sensitize the patient and lead to allergic reactions. Physicians must take backscatter into consideration in patients in whom reconstruction with metallic implants has been performed following tumor resection and who will require radiation therapy. The same population will also have artifacts on future scans.

Resorbable plating systems or biodegradable fixation devices have been used in orthopedics and craniomaxillofacial fields for over 25 years.[60] These materials not only reduce the risk of complications and mitigate the need for implant removal, but add the benefit of rigid fixation with biodegradation. Most of the studies included polymers (e.g., poly-L-lactide, polyglycolide, polyglycolic acid, polylactic acid, and dioxanone). Although reports of the use of resorbable plates in the mandible have been sparse, a prospective study of 50 fractures has reported success in achieving union with 2.5-mm resorbable plates and screws.[61] Resorbable plates now consist of amorphous molded copolymer of L-lactide–D-lactide and trimethylene carbonate for malleability and stability.[61] Initially, the load is carried entirely by the resorbable plate and ultimately by the healed biological union.[62] In 1991, Wittenberg et al[63] found that poly-L-lactide plates were successful in areas of low stress and no compression.

The dimensions of biodegradable plates were usually large compared with miniplates. This would limit their use in the maxillofacial region. These materials have less strength, which would limit their use in loaded and functional bone, such as the mandible. The stability, strength, and elasticity of resorbable osteosynthesis have been extensively investigated in vitro.[63,64] Araujo et al have reported that elastic stiffness and surgical stability of bioresorbable plates may change after surgery, however, and the reduced stiffness could permit relapse after surgery.[64] Polymer chemists and clinicians continue to evaluate biodegradable materials in the hope of developing implants with clinical potential equal to that of metallic implants.

The cases of plate fixation after World War II were sporadic. Before the 1960s, the biomechanical knowledge of internal rigid fixation for the facial skeleton was adapted from the orthopedic literature. In the mid-1960s, Luhr[65] pursued research in rigid fixation for the facial skeleton and developed the Vitallium mandibular compression plate using glide screw principles. Throughout the 1960s, Luhr continued research on rigid fixation and also contributed the self-threading screw. In the 1970s, several investigators, including Spiessl,[67] studied AO-ASIF principles. They found that adaptation of a compression plate on the lateral cortex, at the inferior border of the mandible, gave excellent compression and adaptation at the inferior border but the superior border (tension zone) splayed. Many investigators thought that a second tension zone plate would be necessary, whereas others believed that arch bars in tooth-bearing areas were sufficient to limit tension zone splaying. In 1973, Schmocker and Speissl[68] developed the eccentric dynamic compression plate, which provided compression at the tension and compression zones of the mandible. When the screws closest to the fracture were tightened, the fracture line would be placed under compression. When the eccentric terminals were tightened, the alveolar segment would be reduced.

Further development of the this plate fixation technique introduced reconstruction plates. These were also called load-bearing plates because of their rigidity and application over complex comminuted fractures. Further Innovation by Raveh et al introduced a system that allowed the heads of screws to lock into the titanium plate holes.[69-71] The advantage of this system was that the vascularity of periosteum was not endangered by plate pressure. In the early 1970s, research was initiated to evaluate the use of smaller plating systems. In 1973, Michelet et al[72] placed bendable monocortical miniplates to treat mandibular fractures. The advantages of miniplates were their thinness and the fact that they could be placed through intraoral incisions. Also known as load-sharing plates, miniplates transferred the path of static compression to one of dynamic compression. Fixation to the alveolar ridge allows tensile stress placed on the miniplate to cause an increase in dynamic compression and stability.

Edentulous Mandibular Fractures

Treatment of the fractured edentulous jaw began with a report by Baudens[73] (1844), who used circumferential wiring to reduce and fix the bone. Gunning used splints,

as described earlier.[27-29] In 1851, Robert[74] used silver wire passed circumferentially around the mandible with a needle and tied the wire around a piece of lead that had been molded to the edentulous mandible. Gilmer[36] (1881) described a gutta-percha or vulcanite splint used by Black that was maintained by circumferential wires of silver or platinum. Pickerill[75] (1913) passed screws into the bone on either side of the fracture and maintained them with a connecting steel bar. Circummandibular wiring to maintain a denture splint was often described by Ivy and Curtis in the early 1900s.[39,40]

Another technique, described by Partridge[41] in 1976, involves the use of a nylon circumferential strap wrapped around the body of the mandible and tightened. Two or more of these straps are placed on the fracture and tightened. This method can be applied only to fractures that have extreme obliquity, but the amount of periosteal stripping involved may outweigh any potential benefit.

DEMOGRAPHICS AND EPIDEMIOLOGY

CAUSES

The causes of maxillofacial fractures have changed over the past decades and will continue. Different societies and cultures show different patterns of facial trauma. Varying socioeconomic conditions combined with behavioral differences, however, make the aggregate comparison of mandibular fractures difficult. Obtaining analysis data from various regions can increase the understanding of facial trauma and allow for optimization of treatment.[76,77] Demographic information on maxillofacial injuries has changed with the onset of motor vehicle seat belt and airbag laws, reduced speed limits, and increasing urban violence. Many countries, including Brazil, India, and China, are contributing to this demographic data, along with reports from the United States,[42,43,78-87] England,[88,89] Germany,[90,91] the Netherlands,[92,93] and the Scandinavian countries.[94,98]

The main causes of mandibular fractures worldwide include motor vehicle accidents (MVAs), interpersonal violence (IPV), falls, and sports-related injuries. Past data from industrialized or developed nations with large numbers of vehicles indicated that multiple mandibular fractures were occurring with severe concomitant facial fractures and associated nonmaxillofacial injuries, situations that required extensive treatment. In contrast, assaults and falls have become the predominant mechanism for facial injuries.[99-101] Statistics from smaller developing countries have indicated that MVAs remain the most frequent cause. Ogundare et al[102] retrospectively analyzed mandibular fractures seen in a U.S. urban setting and found that a striking 79% of 1267 mandibular fractures were caused by IPV, whereas Chrcanovic et al[103] found that 44% of mandibular fractures were caused by MVAs in Brazil. It is important to note, however, that local laws and socioeconomic conditions in developed versus developing countries create mixed results for case by case studies.

Despite the many variables associated with the causes of mandibular fractures, MVAs and assaults are undoubtedly the primary causes of mandibular fractures throughout the world. Work-related accidents, firearms, and pathologic conditions are also causative factors. Reports have shown that on average, more than 75% of mandibular fractures are caused by MVAs and IPV, whereas 7% are work-related, 7% occur as the result of a fall, 4% occur in sports-related accidents, and the remainder have miscellaneous causes. Sports-related maxillofacial injuries were especially evaluated by Roccia et al; of 138 patients, 27% sustained a fractured mandible and 6.5% had dental and alveolar fractures. Sane and Ylipaavalniemi[101] studied 8640 accidents that occurred in Finnish soccer players. They found that 6.4% of the injuries occurred in the maxillofacial and dental regions. Of these, 81% affected the teeth or alveolar process and 11% were fractures of the mandible and the middle third of the face. Linn et al[89] have reported on 319 patients treated for sports-related accidents in the Netherlands; 15% of them sustained a mandibular fracture and 5.5% fractured the mandibular alveolar process, had luxated teeth, or both.

Studies of firearm-related mandibular fractures in civilians are limited. Peleg and Sawatari evaluated 92 patients who sustained gunshot wounds to the mandible. In France, Vaillant and Benoist[107] evaluated 14 cases of bullet wounds of the mandible. The age of the patients ranged from 6 to 68 years. Two children were victims of accidents and the adults were suicide or assault victims. The degree of injury was proportionate to the velocity of the projectile. Civilian injuries were not as life-threatening when compared with their high-velocity combat counterparts. Handguns caused 72 injuries, followed by assault rifles and shotguns. Their surgical approach involved fabrication of an occlusal splint, IMF, aggressive débridement of hard and soft tissues, and immediate reconstruction with a titanium plate, which they believed could restore the appropriate function and contour of the patient.

Dental implants have revolutionized the restorative treatment of edentulous patients over the last decade. Unfortunately, a sequela of implant placement and loading is fracture. As implantation procedures have become the norm, fractures of the mandible and other maxillofacial bones are more common. The amount of osseous resorption secondary to long-term use of a prosthesis and the higher incidence of metabolic abnormalities in older adults result in a lower grade of recipient bone. Therefore, patient selection is paramount, and adhering to good osseointegration principles allow for better results. Greenstein et al[103] have stated several predisposing factors for mandibular fracture, such as osteoporosis (reduction of bone mass), stress at the implant location, and trauma (tensile forces placed on the bone during mandibular function). Laskin has suggested that the degree of mobility or displacement determines ultimate treatment; thus, implants that have undergone osseointegration and are involved in the line of fracture should not be removed.[104] Implants that are infected should be removed. The use of open reduction and internal fixation (ORIF) should be avoided because of the amount of periosteal stripping and the lack of bone in the atrophic mandible. Thus, conservative (closed) treatment of these fractures is the best option.

LOCATION OF MANDIBULAR FRACTURES

In the cases evaluated for fracture location, the incidences were as follows: angle (30%), condyle (23%), symphyses (22%), body (18%), ramus (2%), and coronoid process (1%).[105-111] As noted, there are many variables, but generalization fractures that occur in the symphyses, condyle, and angle do not differ much in incidence, and fractures of the ramus and coronoid process are rare.

Individual studies demonstrate how the cause plays a role in fracture location. Chrcanovic et al[100] have shown that when fractures caused by MVAs are considered, the body region is the most common site. When motorcycle accidents are considered, the condyle is the area most often affected. When assault is considered, the body demonstrated the highest incidence of fracture. In another case study, Boole et al have found that assaults and fights lead to a higher percentage of angle fractures, whereas MVAs and motorcycle accidents result in more ramus fractures.[107]

FACIAL FRACTURES ASSOCIATED WITH MANDIBULAR FRACTURES

Mandibular fractures were the only facial bone fractures in an average of 70% of patients. The literature is generally divided between patients with mandibular and midface fractures and those with mandibular and other facial bone fractures. Of the patients reported, 15% had another facial bone fracture, along with the fractured mandible.

NUMBER OF FRACTURES PER MANDIBLE

Studies on the number of fractures per mandible have shown remarkable consistency. The number of mandibular fractures per patient ranged from 1.5 to 1.8. A mean of 53% of patients had unilateral fractures, 37% had two fractures, and 9% had three fractures. However, many authors simply reported single, double, more than two, or single and multiple, so statistical analysis is impossible. It can be concluded that approximately 50% of patients with mandibular fractures have more than one fracture.

NONMAXILLOFACIAL TRAUMA ASSOCIATED WITH MANDIBULAR FRACTURES

The literature dealing with concomitant nonmaxillofacial injuries associated with mandibular fractures is difficult to interpret with the wide variation in reported injuries. The incidence of facial injuries occurring in conjunction with major trauma was shown in several surveys to range from 15% of 1,088 trauma patients in Liverpool[112] to 34% of 87,174 trauma patients in a North American Database on Trauma.[113] In the study of Ellis et al,[110] 90% of patients had no other injuries, probably because the cause was primarily assault. Olson et al[84] have reported associated injuries in 46.6% of all patients treated, most of whom were involved in MVAs. Mulligan and Mahabir[113] have reported associated cervical spine injuries in 6.5% of patients, whereas Elahi et al[114] noted such injuries in only 2.6%. Kapoor and Srivastava[115] noted other (nonfacial) fractures in 40% of patients and Adekeye[116] found other fractures in 9% of patients.

CLASSIFICATION OF MANDIBULAR FRACTURES

Mandibular fractures have been classified in several ways, with the use of terminology that has not been standardized. For example, the term *simple fracture* has been defined by Assael and Tucker as "… a complete transection of the bone with minimal fragmentation of the fracture site."[117] Rowe and Killey defined simple fractures as "…linear fractures which are not in communication with the exterior."[85] Kruger has stated that a simple fracture "… is one in which the overlying integument is intact. The bone has been broken completely but it is not exposed to air. It may or may not be displaced. It may be comminuted."[118] Whitestone and Raley have defined a simple fracture as follows: "…the overlying mucosa is intact with no potential sources of direct communication."[119]

DICTIONARY CLASSIFICATION

For the sake of this discussion, the following fracture terms have been adopted from *Dorland's Illustrated Medical Dictionary*[120]:

1. Simple or closed: A fracture that does not produce a wound open to the external environment, whether it be through the skin, mucosa, or periodontal membrane
2. Compound or open: A fracture in which an external wound, involving skin, mucosa, or periodontal membrane, communicates with the break in the bone
3. Comminuted: A fracture in which the bone is splintered or crushed
4. Greenstick: A fracture in which one cortex of the bone is broken, the other cortex being bent
5. Pathologic: A fracture occurring from mild injury because of preexisting bone disease
6. Multiple: A variety in which there are two or more lines of fracture on the same bone not communicating with one another
7. Impacted: A fracture in which one fragment is firmly driven into the other
8. Atrophic: A spontaneous fracture resulting from atrophy of the bone, as in edentulous mandibles
9. Indirect: A fracture at a point distant from the site of injury
10. Complicated or complex: A fracture in which there is considerable injury to the adjacent soft tissue or adjacent parts; may be simple or compound

CLASSIFICATION BY ANATOMIC REGION

Mandibular fractures are also classified by the anatomic areas involved, as follows: symphysis, body, angle, ramus, condylar process, coronoid process, and alveolar process. Dingman and Natvig[78] have defined these regions as follows:

1. Midline: Fractures between central incisors
2. Parasymphyseal: Fractures occurring within the area of the symphysis
3. Symphysis: Bounded by vertical lines distal to the canine teeth

4. Body: From the distal symphysis to a line coinciding with the alveolar border of the masseter muscle (usually including the third molar)
5. Angle: Triangular region bounded by the anterior border of the masseter muscle to the posterosuperior attachment of the masseter muscle (usually distal to the third molar)
6. Ramus: Bounded by the superior aspect of the angle to two lines forming an apex at the sigmoid notch
7. Condylar process: Area of the condylar process superior to the ramus region
8. Coronoid process: Includes the coronoid process of the mandible superior to the ramus region
9. Alveolar process: The region that would normally contain teeth

Kazanjian and Converse[120] have classified mandibular fractures by the presence or absence of serviceable teeth in relation to the line of fracture. They thought that their classification was helpful in determining treatment. Three classes were defined:

Class I: Teeth are present on both sides of the fracture line.
Class II: Teeth are present on only one side of the fracture line.
Class III: The patient is edentulous.

They thought that class I fractures could be treated by a variety of techniques, using the teeth for monomaxillary or intermaxillary fixation. Class II fractures, usually involving the condyle-ramus angle or partially edentulous body of the mandible, require intermaxillary fixation. Class III fractures require prosthetic techniques, open reduction methods, or both for stabilization.

Rowe and Killey[85] have divided mandibular fractures into two classes: (1) those not involving basal bone; and (2) those involving basal bone. The first class primarily is comprised of alveolar process fractures. The second class is divided into single unilateral, double unilateral, bilateral, and multiple.

Kruger[118] has classified mandibular fractures into simple, compound, and comminuted. Kruger and Schilli[121] took into account many of the aforementioned classifications described and developed four categories of mandibular fractures:

1. Relation to the external environment
   a. Simple or closed
   b. Compound or open
2. Types of fractures
   a. Incomplete
   b. Greenstick
   c. Complete
   d. Comminuted
3. Dentition of the jaw with reference to the use of splints
   a. Sufficiently dentulous jaw
   b. Edentulous or insufficiently dentulous jaw
   c. Primary and mixed dentition
4. Localization
   a. Fractures of the symphysis region between the canines
   b. Fractures of the canine region
   c. Fractures of the body of the mandible between the canine and angle of the mandible
   d. Fractures of the angle of the mandible in the third molar region
   e. Fractures of the mandibular ramus between the angle of the mandible and sigmoid notch
   f. Fractures of the coronoid process
   g. Fractures of the condylar process

Shetty et al[122] combined six significant injury criteria to create the acronym FLOSID, which essentially allowed for ease of assessment and defined fracture characteristics. They assessed mandibular fractures using the taxonomy described and added weighting factors to address severity (mandible injury severity score):

1. Fracture type (F)
   a. Incomplete
   b. Simple
   c. Comminuted
   d. Bone defect
2. Location of fracture (L)
   a. Left from midline (L1) to condylar head (L8)
   b. Right from midline (R1) to condylar head (R8)
3. Nature of occlusion (O)
   a. Normal
   b. Malocclusion
   c. Edentulous
4. Extent of soft tissue damage (S)
   a. Closed
   b. Open intraorally
   c. Open extraorally
   d. Open intra and extraorally
   e. Soft tissue defect
5. Presence of infection (I)
   a. Yes
   b. No
6. Radiographic analysis of interfragmentary displacement (D)
   a. Mild
   b. Moderate
   c. Severe

An important classification of mandibular angle and body fractures relates to the direction of the fracture line and the effect of muscle action on the fracture fragments. Angle fractures may be classified as (1) vertically favorable or unfavorable or (2) horizontally favorable or unfavorable. Figures 14-5 through 14-8 demonstrate the various types.

In fractures of the angle of the mandible, the muscles attached to the ramus (masseter, temporal, and medial pterygoid) displace the proximal segment upward and medially when the fractures are vertically and horizontally unfavorable (Fig. 14-9). Conversely, these same muscles tend to impact the bone, minimizing displacement in horizontally and vertically favorable fractures. The farther forward the fracture occurs in the body of the mandible, the more the upward displacement of those muscles is counteracted by the downward pull of the mylohyoid muscles. In bilateral fractures in the canines areas, the symphysis of the mandible is displaced inferiorly and posteriorly by the pull of the digastric, geniohyoid, and genioglossus muscles (Fig. 14-10).

Condylar fractures are generally classified as extracapsular, subcondylar, or intracapsular. Condylar fractures are influenced by location and muscle action. The lateral

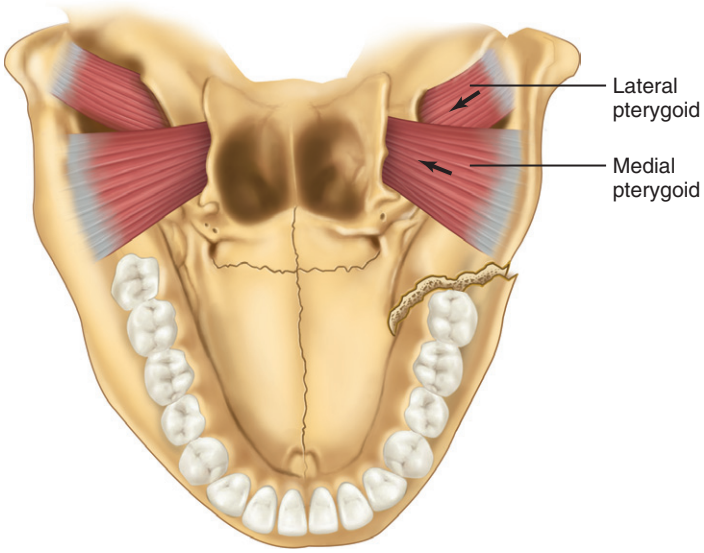


FIGURE 14-5 Vertically unfavorable fracture.

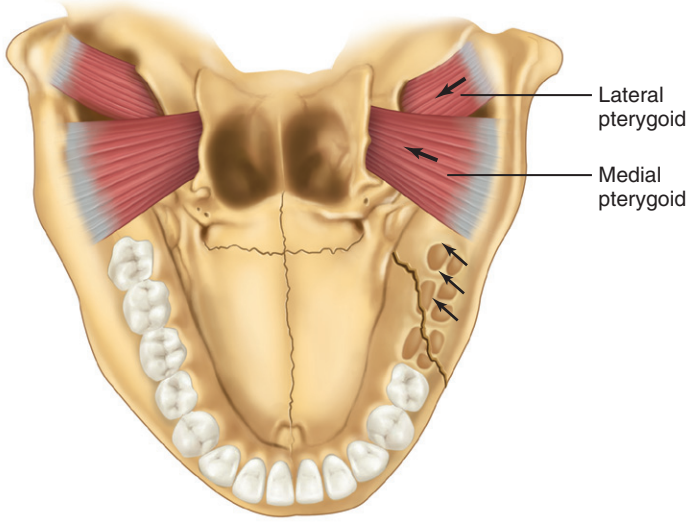


FIGURE 14-6 Vertically favorable fracture.

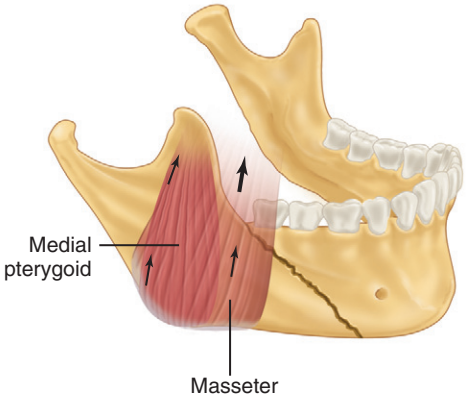


FIGURE 14-7 Horizontally favorable fracture.

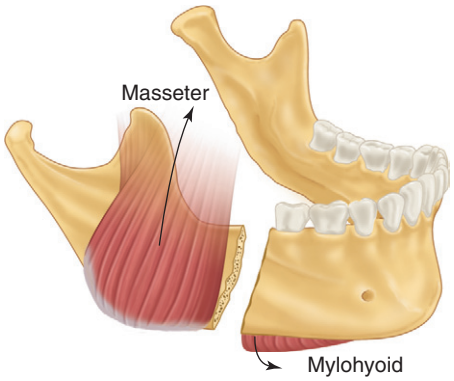


FIGURE 14-8 Horizontally unfavorable fracture.

Lateral pterygoid:
Upper head inserts into articular disk and upper part of condylar head

Lower head inserts into pterygoid fovea

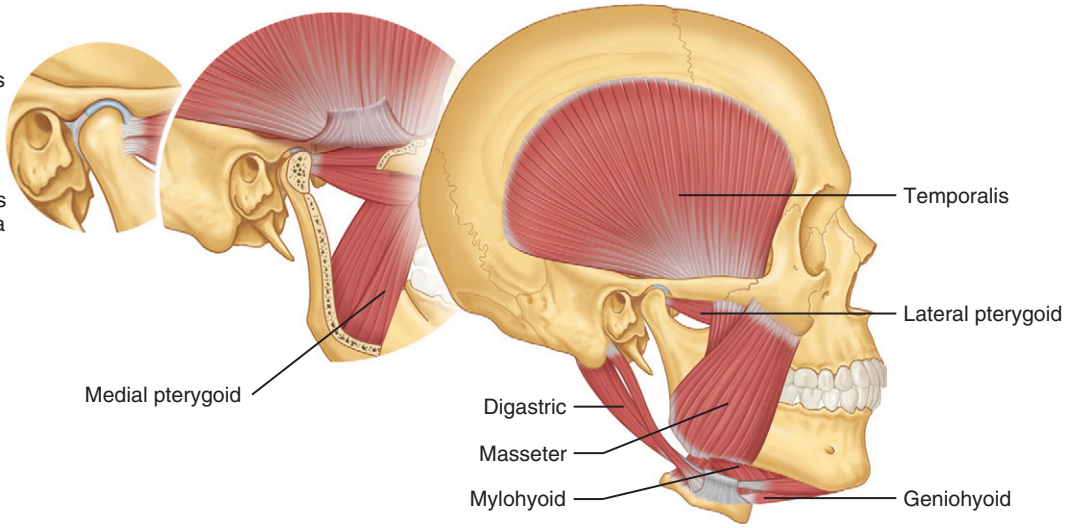


FIGURE 14-9 Muscles of mastication, which have a displacing influence on mandibular fractures.

pterygoid muscle has a tendency to cause anterior and medial displacement of the condylar head, depending on the location, severity of the fracture, and effect of the supporting capsule (Fig. 14-11). In 1934, Wassmund has described five types of condylar fractures.[17] Type I is defined as a fracture of the neck of the condyle, with relatively slight displacement of the head. The angle between the head and axis of the ramus varies from 10 to 45 degrees. These fractures tend to reduce spontaneously. Type II fractures produce an angle from 45 to 90 degrees, resulting in tearing of the medial portion of the joint capsule. In type III fractures, the fragments are not in contact and the head is displaced mesially and forward because of traction of the lateral pterygoid muscle. The fragments are generally confined within the area of the glenoid fossa. The capsule is torn and the head is outside the capsule. An open reduction is recommended for this fracture type. Type IV fractures of the condylar head articulate on, or in a forward position with regard to, the articular eminence. The type V group consists of vertical or oblique fractures through the head of the condyle, and a bone graft is suggested to reconstitute the condylar head when considerable displacement of the fragments has occurred.[17]

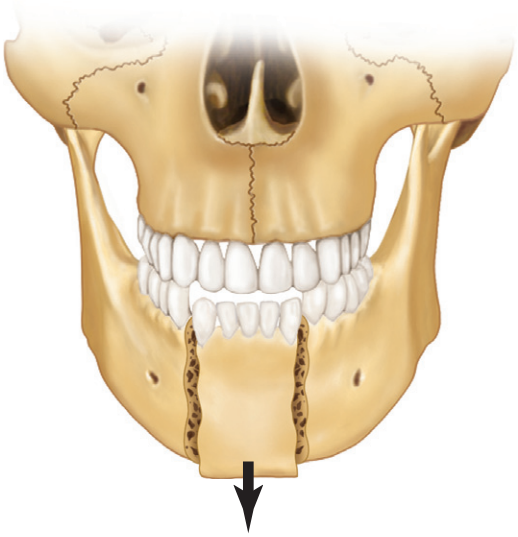


FIGURE 14-10 Inferior and posterior displacement of the symphysis of the mandible in bilateral fractures in the canine area.

As a means of simplifying the classification of high and low condylar fractures, Loukota et al have proposed a three-part system.[123] The classification system revolves around a reference line, which is a linear line that extends from the posterior border of the condylar neck through the sigmoid notch to the tangent of the ramus. First, the diacapitular type describes a fracture through the head of the condyle, because it may start on the articular surface and extend outside the capsule. Second, a condylar neck type describes a fracture that is at minimum over 50% above the reference line. Finally, a condylar base type refers to a fracture line that runs behind the mandibular foramen and is at minimum over 50% below the reference line (Fig. 14-12).

DIAGNOSIS OF MANDIBULAR FRACTURES

PATIENT HISTORY

A thorough history is imperative for the proper diagnosis of mandibular fractures. The patient's health history may reveal preexisting systemic bone disease, neoplasia with potential metastases, arthritis and related collagen disorders, nutritional and metabolic disorders, and endocrine diseases that may cause or be directly related to the

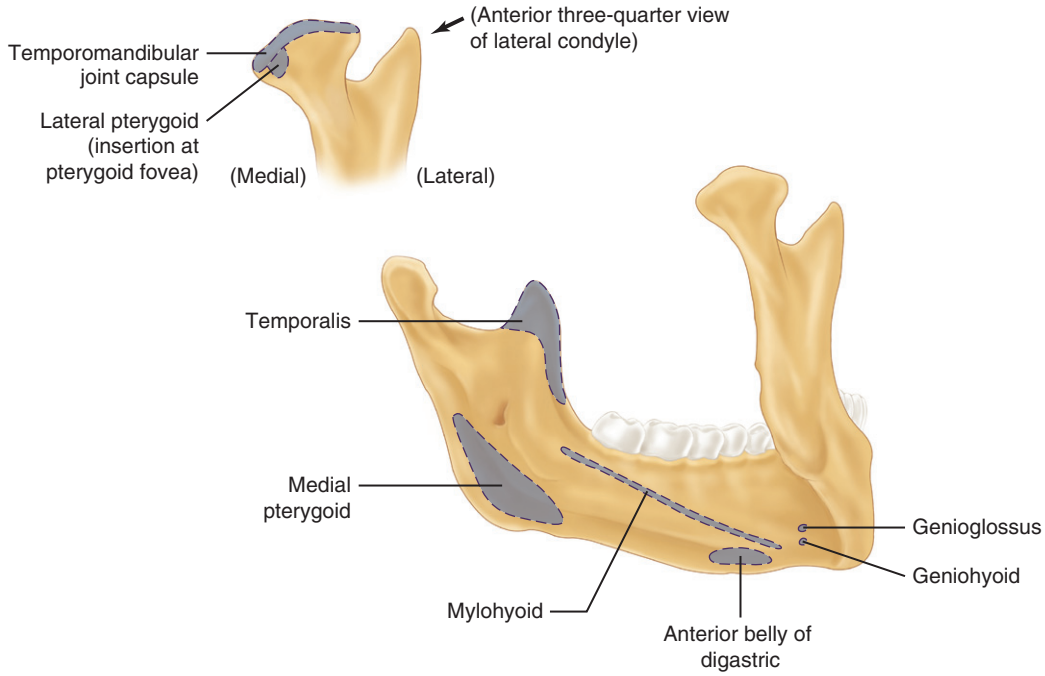


FIGURE 14-11 The lateral pterygoid muscle tends to cause anterior and medial displacement of the condylar head.

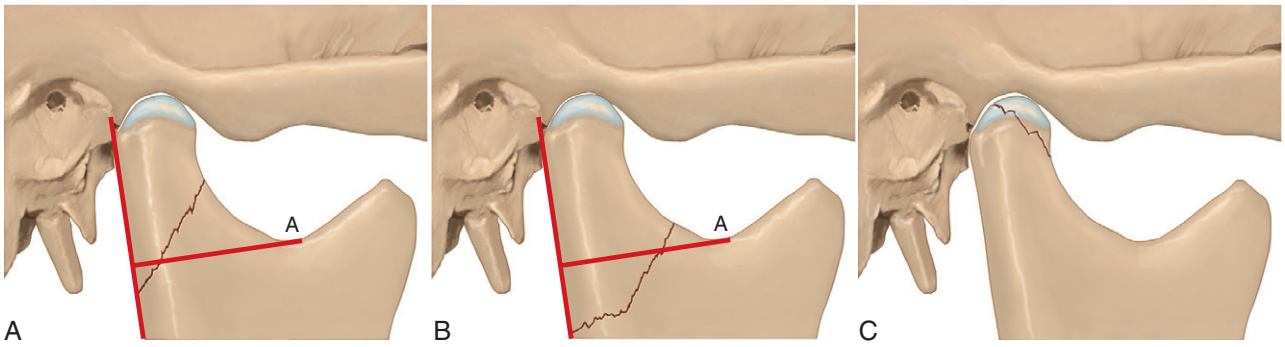


FIGURE 14-12 Classification of condylar fractures. **A,** High or condylar neck fracture. **B,** Low or condylar base fracture. **C,** Diacapitular fracture. (From Ward Booth P, Eppley BL, Schmelzeisen R: *Maxillofacial trauma and esthetic facial reconstruction*, ed 2, St. Louis, 2012, Saunders.)

fractured jaw. The history also reveals significant medical and psychiatric problems that will influence the management of the patient and perhaps even dictate treatment modalities.

A history of temporomandibular joint (TMJ) dysfunction can have significant legal and post-treatment ramifications. A number of clinicians and radiologists have experienced problems by not obtaining an adequate history of previous mandibular trauma or fracture.

The type and direction of traumatic force can be extremely helpful for the diagnosis. Fractures sustained in MVAs are usually different from those sustained in IPVs. Because the magnitude of the force can be much greater, victims of automobile and motorcycle accidents tend to have multiple, compound, comminuted mandibular fractures, whereas the patient hit by a fist may sustain a single, simple nondisplaced fracture.

The object that caused the fracture can also influence the type and number of fractures. A blow from a broad blunt object (e.g., a two by four piece of wood) may cause several fractures (e.g., symphysis, condyle) because the impact of the force is sustained throughout the bone, whereas a smaller, well-defined object (e.g., hammer, pipe) may cause a single comminuted fracture becausethe impact of the force is concentrated in a smaller area.

Knowing the direction of force can help the clinician diagnose concomitant fractures. An anterior blow directly to the chin can result in bilateral condylar fractures (Fig. 14-13) and an angled blow to the parasymphysis may cause a contralateral condylar or angle fracture. A patient with teeth clenched together at the moment of impact is more likely to have dental and alveolar process fractures than basal bone fractures. Even knowing where the patient was sitting in an automobile may aid in the diagnosis of mandibular or other injuries. Chest injuries caused by noncollapsible steering wheels, facial fractures caused by striking unpadded dashboards, and facial lacerations from non–safety glass are a few examples of predictable injuries that have been eliminated by the use of seat belts and more effective automotive safety engineering.[124,125]

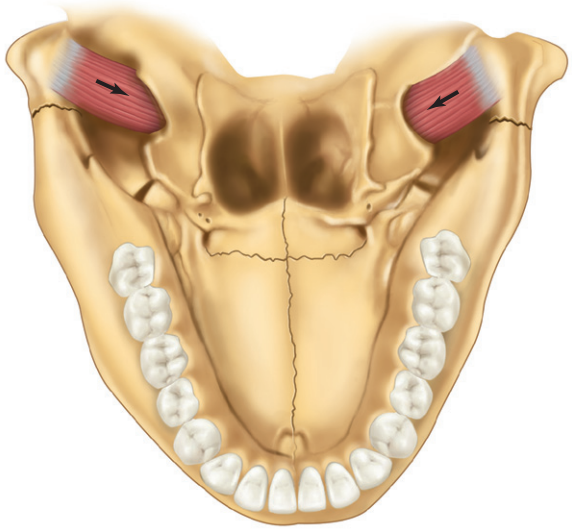


FIGURE 14-13 Trauma in the form of an anterior blow directly to the chin can cause bilateral condylar fractures.

CLINICAL EXAMINATION

Initial evaluation is part of the secondary survey when adhering to the Advanced Trauma Life Support (ATLS) protocol. Stabilization of the airway may require tracheotomy in severe crushing injuries, such as the hallmark flail mandible. The mandible should be evaluated after the patient is stabilized and more life-threatening injuries have been addressed. The signs and symptoms of mandibular fractures are as follows.

Change in Occlusion. Any change in occlusion is highly suggestive of mandibular fracture. The clinician should ask the patient whether his or her bite feels different. A change in occlusion can result from fractured teeth, fractured alveolar process, fractured mandible at any location, and trauma to the TMJ and muscles of mastication. Post-traumatic premature posterior dental contact or an anterior open bite may result from bilateral fractures of the mandibular condyle or angle (Fig. 14-14) and from maxillary fractures with inferior displacement of the



FIGURE 14-14 Bilateral angle fractures can cause an open bite.

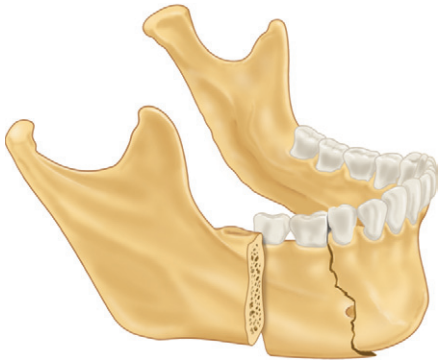


FIGURE 14-15 Unilateral open bite may be the result of ipsilateral angle and parasymphyseal fractures.

posterior maxilla. A posterior open bite may occur with fractures of the anterior alveolar process or parasymphyseal fractures. A unilateral open bite may occur because of ipsilateral angle and parasymphyseal fractures (Fig. 14-15). A posterior crossbite can result from midline symphyseal and condylar fractures, with splaying of the posterior mandibular segments.

Retrognathic occlusion is associated with condylar or angle fractures (and forward displaced maxillary fractures); prognathic occlusion can occur with effusion of the TMJ joints and with protective forward posturing of the mandible (also with retropositioning of the maxilla). These are only a few of the many occlusal disharmonies that can exist, but any change in occlusion has to be considered the primary diagnostic sign of mandibular fracture.

Anesthesia, Paresthesia, or Dysesthesia of the Lower Lip. Although changes in sensation in the lower lip and chin may be related to chin and lip lacerations and blunt trauma, numbness in the distribution of the inferior alveolar nerve after trauma is almost pathognomonic of a fracture distal to the mandibular foramen. Conversely, most nondisplaced fractures of the mandibular angle, body, and symphysis are not characterized by anesthesia, so the clinician must not use lip anesthesia as the sole feature in diagnosis.

Abnormal Mandibular Movements. Most patients with a fractured mandible have limited opening and trismus because of guarding of the muscles of mastication. However, certain mandibular fractures or associated facial fractures result in predictable abnormal mandibular movements. A classic example is deviation on opening toward the side of a mandibular condylar fracture. Because lateral pterygoid muscle function on the unaffected side is not counteracted on the opposite side by the nonfunctioning lateral pterygoid muscle, deviation results. Inability to open the mandible may be caused by the impingement of the coronoid process on the zygomatic arch from fractures of the ramus and coronoid process or from depression of a zygomatic arch fracture. Inability to close the jaw can be the result of fractures of the alveolar process, angle, ramus, or symphysis, causing premature dental contact. Lateral mandibular movements may be inhibited by bilateral condylar fractures and fractures of the ramus with bone displacement.

Change in Facial Contour and Mandibular Arch Form. Although facial contour may be masked by swelling, the clinician should examine the face and mandible for abnormal contours. A flattened appearance of the lateral aspect of the face may be the result of a fractured body, angle, or ramus. A deficient mandibular angle can occur with unfavorable angle fractures in which the proximal fragment rotates superiorly. A retruded chin can be caused by bilateral parasymphyseal fractures. The appearance of an elongated face may be the result of bilateral subcondylar, angle, or body fractures, allowing the anterior mandible to be displaced downward. Facial asymmetry should alert the clinician to the possibility of a mandibular fracture. The same holds true for mandibular arch form. If there is a deviation from the normal U-shaped curve of the mandible, fracture should be suspected.

Lacerations, Hematoma, and Ecchymosis. Trauma significant enough to cause loss of skin or mucosal continuity, or subcutaneous-submucosal bleeding, can result in trauma to the underlying mandible. Lacerations should be carefully inspected before closure. The direction and type of fracture may be visualized directly through the laceration, with the clinician thereby obtaining diagnostic information that might be impossible to ascertain clinically or radiographically. The common practice of closing facial lacerations before treating underlying fractures should be discouraged from diagnostic and treatment standpoints. The diagnostic sign of ecchymosis in the floor of the mouth indicates a mandibular body or symphyseal fracture (Fig. 14-16).

Loose Teeth and Crepitation on Palpation. A thorough examination of the teeth and supporting bone can help diagnose alveolar process, body, and symphyseal fractures. A force strong enough to loosen teeth can fracture the underlying bone. Multiple fractured teeth that are firm indicate that the jaws were clenched during traumatic insult, thus lessening the effect on the supporting bone. The clinician should palpate the mandible using both hands, with the thumb on the teeth and the fingers on the lower border of the mandible. By slowly and carefully placing pressure between the two hands, the clinician can detect crepitation in a fracture. However, this simple diagnostic technique is often overlooked in favor of extensive (and expensive) radiologic diagnostic methods.

Dolor, Tumor, Rubor, and Color. Pain, swelling, redness, and localized heat have been noted as signs of inflammation since the time of the ancient Greeks. All these findings are excellent primary signs of trauma and can greatly increase the index of suspicion for a mandibular fracture.

RADIOLOGIC EXAMINATION

The following radiologic studies are helpful in the diagnosis of mandibular fractures:

1. Panoramic radiograph
2. Lateral oblique radiograph
3. Posteroanterior radiograph
4. Occlusal view
5. Periapical view
6. Reverse Towne's view
7. TMJ, including tomograms
8. CT—high-resolution spiral or helical CT

The single most informative radiologic study used in diagnosing mandibular fractures is the panoramic radiograph, showing the entire mandible, including condyles (Fig. 14-17).[126] The advantages are simplicity of technique, ability to visualize the entire mandible in one radiograph, and generally good detail, but there are several disadvantages: (1) the technique usually requires the patient to be upright (machines that allow the patient to be prone are available), which may make it impractical in the severely traumatized patient; (2) it is difficult to appreciate buccal-lingual bone displacement or medial condylar displacement; and (3) fine detail is lacking in the TMJ , symphysis (depending on the type of equipment), and dental and alveolar process regions. A secondary but important disadvantage is that panoramic radiographic equipment is not available in all hospital radiology departments. In addition, because of the sensitivity of the panoramic radiographic technique, superimposition of structures can lead to interpretation errors.[127]

The lateral oblique view of the mandible can be helpful in the diagnosis of ramus, angle, and posterior body fractures (Fig. 14-18). The technique is simple and can be done in any radiology department. The condyle region is often unclear, as are the premolar and symphysis regions. The Caldwell posteroanterior (PA) view demonstrates any medial or lateral displacement of fractures of the ramus, angle, body, and symphysis (Fig. 14-19). The condylar region is not well demonstrated on this view, but midline or symphyseal fractures can be well visualized. The anteroposterior view is occasionally used for patients who cannot be placed in the supine position; however, considerable magnification and distortion occur with this view. The mandibular occlusal view demonstrates discrepancies in the medial and lateral position of body fractures and also shows anteroposterior (AP)

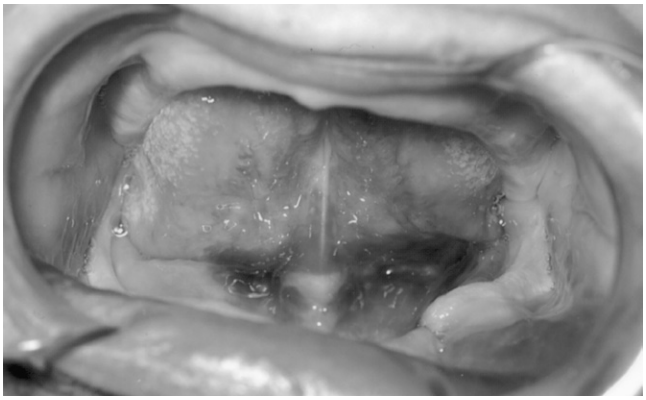


FIGURE 14-16 Ecchymosis in the floor of the mouth is a significant diagnostic sign of a mandibular body or symphyseal fracture.

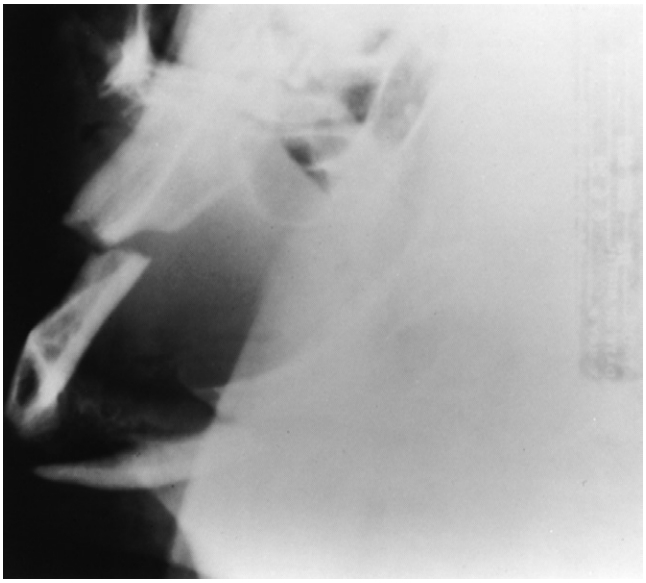


FIGURE 14-18 The lateral oblique view of the mandible is helpful in diagnosing ramus, angle, and posterior body fractures.

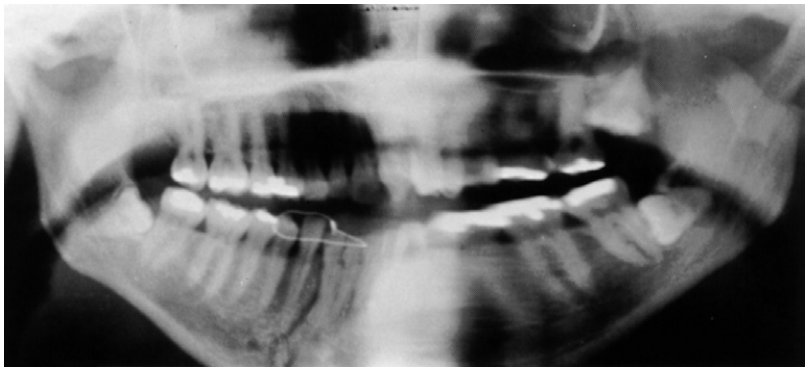


FIGURE 14-17 Panoramic radiograph, the single most informative radiograph for the diagnosis of mandibular fractures.



FIGURE 14-19 The Caldwell posteroanterior view shows medial or lateral displacement of fractures of the ramus, angle, body, and symphysis.

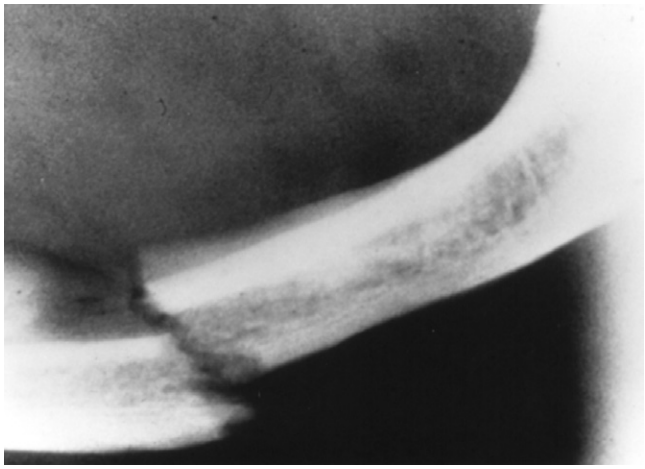


FIGURE 14-20 With the mandibular occlusal view, discrepancies in the medial and lateral positions of body fractures and anteroposterior displacement of the symphysis can be shown.



FIGURE 14-21 The reverse Towne view is most helpful for showing medial displacement of the condyle and condylar neck fractures.



FIGURE 14-22 CT scanning is ideal for condylar fractures.

displacement in the symphysis region (Fig. 14-20). The reverse Towne view is ideal for showing medial displacement of condyle and condylar neck fractures (Fig. 14-21). Transcranial lateral views of the TMJ are helpful in detecting condylar fractures and anterior displacement of the condylar head. Periapical dental films show the most detail and can be used for nondisplaced linear fractures of the body and alveolar process and dental trauma. Plain tomograms can be used in an AP and lateral direction when greater detail is necessary. The traditional CT scan is ideal for condylar fractures that are difficult to visualize; however, greater expense and radiation exposure limit its use to cases that cannot be diagnosed with plain films and panoramic tomography (Fig. 14-22).[128] With the advent of newer technology, helical CT (HCT) is faster, more accurate, and less expensive and delivers decreased radiation exposure as compared with conventional CT scans. According to Roth et al, it also provides enhanced resolution because of less motion misregistration. Hence, the trend toward making HCT the current gold standard is underway.[128]

In summary, as with most other imaging procedures, it is usually optimal to have views of the mandible in two planes oriented at 90 degrees to each another. Ideal imaging includes sections in the axial and coronal planes but practical considerations of time, the patient's condition, and cost mandate selectivity. Thus, the history, physical examination, and understanding which structures are best shown by certain imaging methods dictate the proper radiologic technique.[129,130]

GENERAL PRINCIPLES OF TREATMENT

Throughout this text, specific chapters have been devoted to preoperative, operative, and postoperative treatment of facial fractures. This section presents general principles used in the management of mandibular fractures.

1. The patient's general physical status should be carefully evaluated and monitored before any consideration of treating mandibular fractures.

   It must be emphasized that any force great enough to cause a fractured mandible is capable of injuring other organ systems in the body. This is obvious when dealing with massive crush injuries of the face, with concomitant multiorgan system involvement. However, it is easy for the clinician to focus on an obvious isolated mandibular fracture without noting a fractured cervical spine, subdural hematoma, pneumothorax, cardiac tamponade, or ruptured spleen. The downward spiral to disaster can begin by not following this principle.

   The literature is replete with case reports of isolated fractures of the mandible in which life-threatening medical conditions became apparent after treatment was instituted. Post-traumatic thrombotic occlusion of the internal carotid artery associated with mandibular fractures (and no other apparent injuries) was diagnosed 24 hours after injury in one case and 9 hours after injury in another.[131] Banna[131] has also reported a case and reviewed the literature on post-traumatic thrombotic occlusion of the internal carotid artery after minimal neck trauma. A basal skull fracture associated with an undisplaced body fracture of the mandible became apparent 48 hours after injury.[132] Gordon et al[132] have described a patient with a unilateral body fracture of the mandible who had symptoms of a ruptured spleen 5 days after injury and 3 days after arch bars had been placed. Bertolami and Kaban[133] have reported on a case of a 20-year-old woman involved in an MVA who sustained a 3-cm chin laceration without malocclusion or limitation of mandibular movement. After 8 hours, radiographs were reviewed and showed a nondisplaced fracture of the angle of the mandible and a fracture of the second cervical vertebra. Minton and Tu[134] have noted a case of bilateral cervical subcutaneous emphysema, pneumothorax, and pneumomediastinum secondary to a bilateral fractured mandible without other apparent injuries.
2. Diagnosis and treatment of mandibular fractures should be approached methodically, not with an emergency-type of mentality.

   Patients rarely die of mandibular fractures, so the clinician has time to evaluate the nature and extent of mandibular injuries carefully and thoroughly. Diagnosis on the basis of the history and local physical and radiologic examination should be expedited in an orderly and efficient manner, and treatment should be instituted in a controlled fashion. However,this is not to condone prolonged unnecessary delay, which can increase the potential for infection and nonunion.
3. Dental injuries should be evaluated and treated concurrently with treatment of mandibular fractures.

   Teeth are often injured with mandibular fractures and, although the teeth may not have to be restored immediately, dental knowledge is vitally important for determining which teeth can and should be maintained.
   a. Fractured teeth can become infected and jeopardize bone union; however, an intact tooth in the line of fracture that is maintaining bone fragments can be protected with antibiotic coverage (Fig. 14-23).
   b. A second molar on an otherwise edentulous posterior fracture segment should be maintained to prevent superior displacement of the fragment in intermaxillary fixation.
   c. Mandibular canines are the cornerstone of occlusion and should be maintained at all costs.
   d. Some teeth are not critical to restoration and can be removed when their prognosis is doubtful and when maintenance may adversely affect fracture treatment. For example, a lone mandibular incisor adds little to future bridge or partial denture construction; however, a single molar tooth in an otherwise edentulous posterior quadrant can be critical to dental rehabilitation.
   e. Some fractured teeth cannot be salvaged, no matter how critical they may be. For example, a molar tooth may be split mesially and distally, so reconstruction would be impossible. Maintenance of this tooth during intermaxillary fixation could result in severe discomfort and perhaps infection.
4. Reestablishment of occlusion is the primary goal in the treatment of mandibular fractures.

   Probably because of the mandible's excellent blood supply, nonunion of mandibular fragments is rare, so it is apparent that bone fragments do not have to be in tight approximation to heal. In addition, in most cases, facial aesthetics will not be adversely affected by slight fragment displacement. However, function can be seriously compromised when improper treatment results in malocclusion. Impressive appearing radiographic bone adaptation should not be the primary treatment goal.
5. With multiple facial fractures, mandibular fractures should be treated first.

   The old adage "inside out and from bottom to top" applies to the proper sequence to follow when treating facial fractures. To build a foundation on which the facial bones can be laid, the mandible should be reconstructed first, although with the use of rigid fixation, deviation from this principle can be allowed. All intraoral surgery should be done before any extraoral open reductions or suturing of facial lacerations. Lip and skin wounds that have been meticulously closed in an emergency room are often inadvertently, or even necessarily, reopened during the treatment of mandibular fractures. Gross débridement and control of hemorrhage should be combined with temporary measures to reapproximate extraoral wounds, thus allowing definitive treatment to be carried out after the intraoral procedures are completed.
6. Intermaxillary fixation time should vary according to the type, location, number, and severity of the

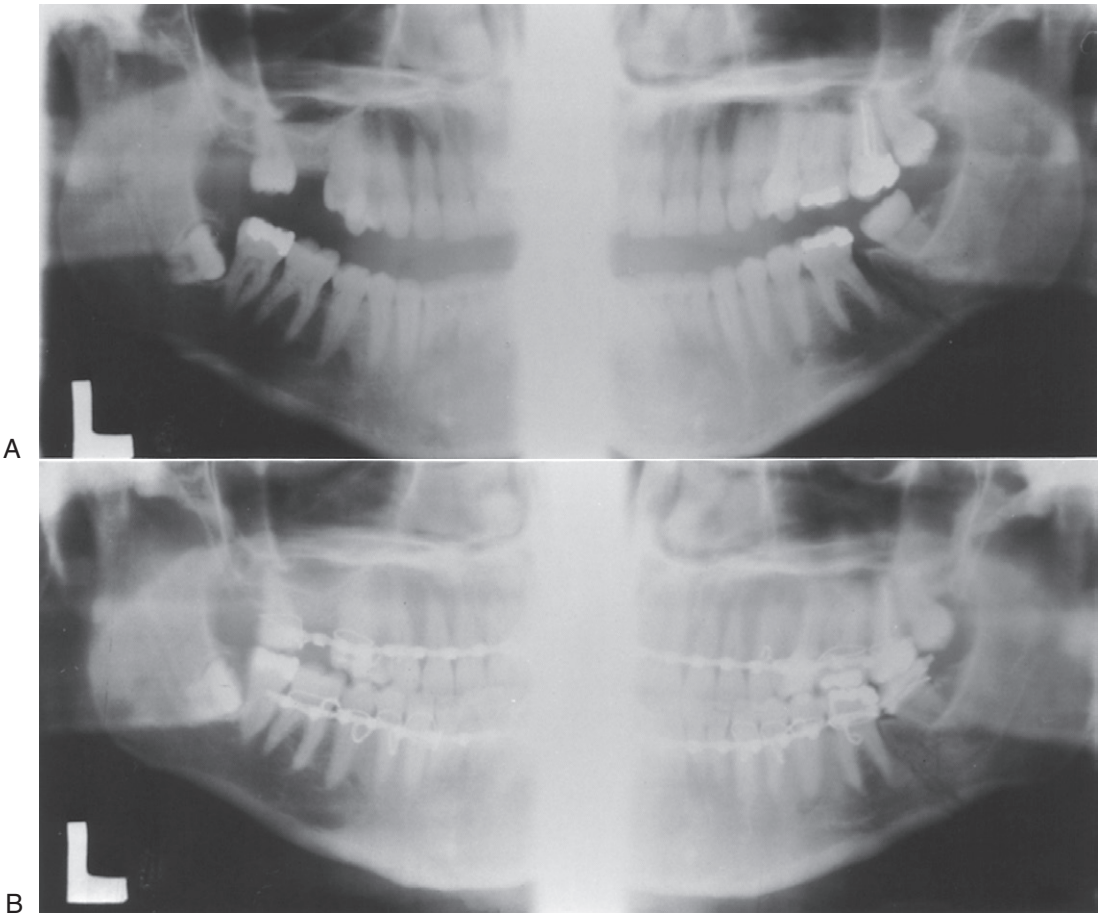


FIGURE 14-23 An intact tooth in the line of fracture can be maintained to prevent displacement of the segment.

mandibular fractures, the patient's age and health, and the method used for reduction and immobilization.

Historically, a period of 6 weeks of intermaxillary fixation has been used to allow healing to occur. However, this length of time is only empirical and should vary with the patient and clinical situation.[135] A simple, nondisplaced, greenstick mandibular fracture in a healthy child would certainly require less intermaxillary fixation time than multiple, grossly comminuted, compound mandibular fractures in an older unhealthy patient.[136] With the advent of rigid fixation techniques, intermaxillary fixation may be eliminated or maintained with light elastics for short periods.

7. Prophylactic antibiotics should be used for compound fractures.

   The benefit of pre- and perioperative antibiotics has been extensively analyzed and verified to lower postoperative infection rates. Numerous literature studies have demonstrated the advantages of antibiotics in the management of compound mandibular fractures; despite the number of new antibiotics, however, penicillin remains the agent of choice.[96,137-140] However, much debate remains in regard to assessing the efficacy and duration of a postoperative antibiotic regimen.[138,141-145]

8. Nutritional needs should be closely monitored postoperatively.

   Excellent reduction and fixation techniques may fail in a patient who has undergone notable weight loss and has a catabolic nutritional status.

9. Mandibular fractures can be treated by closed reduction.

   With enthusiasm for open reduction and rigid fixation in the treatment of mandibular fractures, it is important to remember that closed reduction techniques have a long history of success.[146,147] Although open techniques have advantages, such as more exacting bone fragment reapproximation and earlier return to function by the patient, significant disadvantages exist as well. These may subject the patient to prolonged anesthesia, increase the risk of infection and metal rejection, cause damage to adjacent teeth and nerves, result in intraoral or extraoral scarring, and increase hospitalization time and cost. The following sections present relative indications for open or closed techniques.

INDICATIONS FOR CLOSED REDUCTION

Nondisplaced Favorable Fractures. The simplest means possible should be used to reduce and fixate mandibular fractures (Fig. 14-24). For the reasons specified earlier,

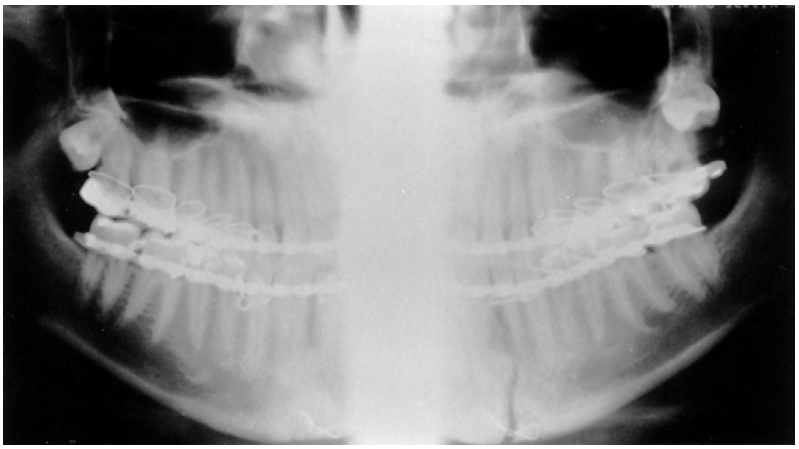


FIGURE 14-24 Nondisplaced favorable fracture.

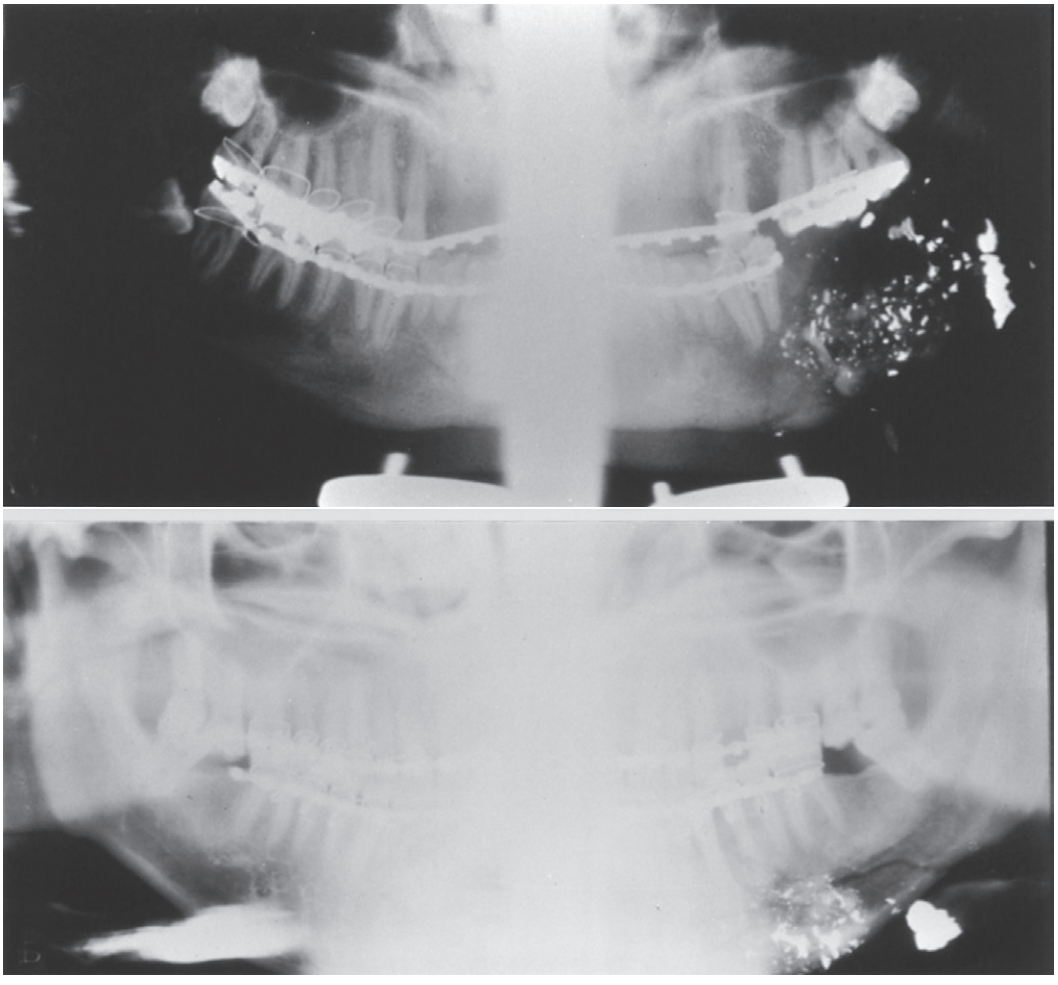


FIGURE 14-25 Grossly comminuted mandibular fractures.

open reduction can carry an increased risk of morbidity, so closed techniques should be used for treatment. if possible.

Grossly Comminuted Fractures. Because of the excellent blood supply to the face, small fragments of bones will coalesce and heal if the associated periosteum is not disturbed(Fig. 14-25). Comminuted fractures should be managed as a so-called bag of bones, with the clinician using closed techniques to establish normal occlusion without violating integrity.

Fractures Exposed by Significant Loss of Overlying Soft Tissue. Fracture repair is somewhat dependent on soft tissue coverage and vascular supply. Soft tissue coverage should be established by rotational flaps, microvascular grafts or, if the area is small, secondary granulation. Wires, screws, and plates may decrease the chance of

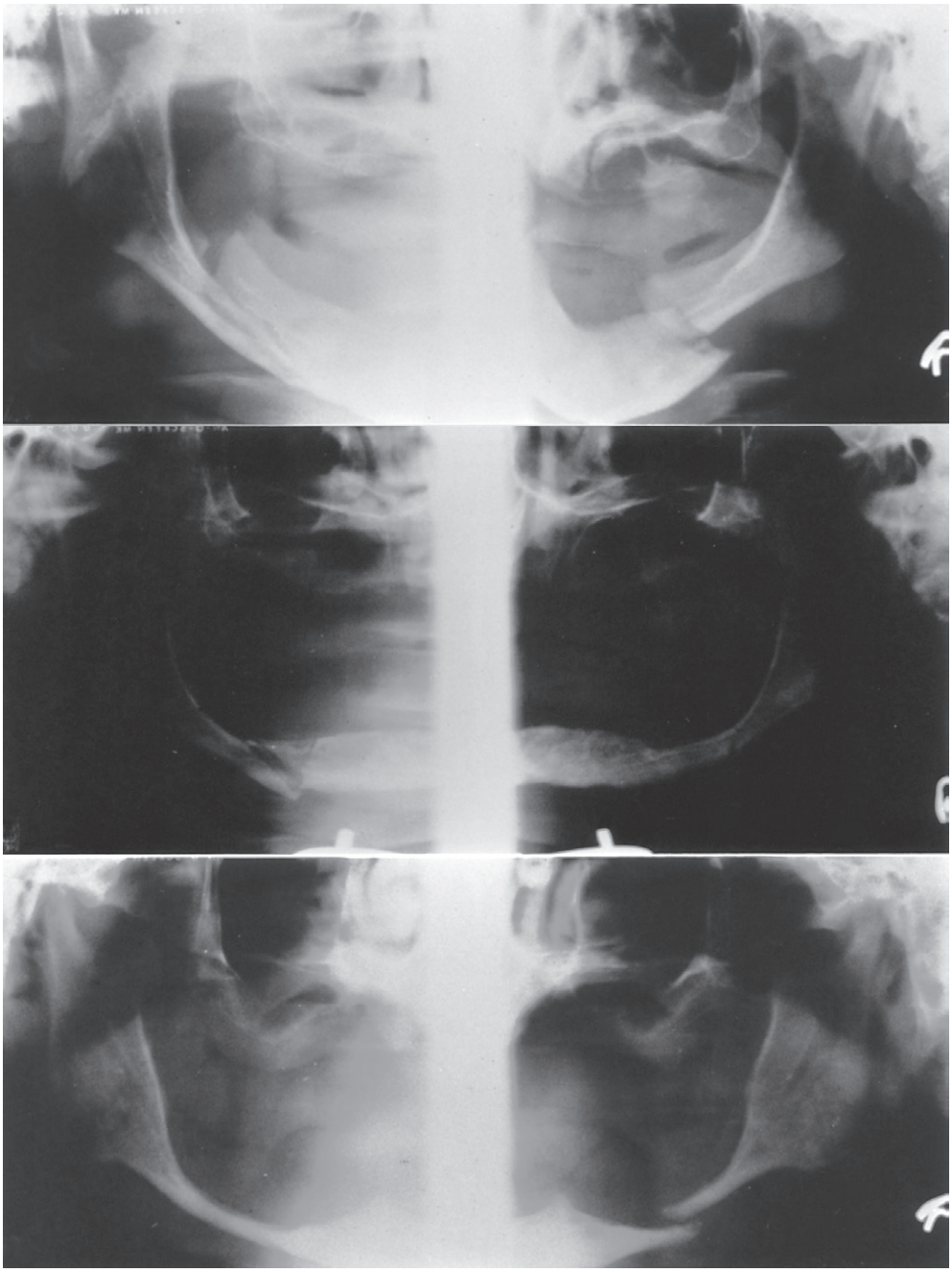


FIGURE 14-26 Edentulous mandibular fractures. **A,** Minimal atrophy. **B,** Moderate atrophy. **C,** Severe atrophy.

successful bone union by disrupting the covering soft tissue further.

Edentulous Mandibular Fractures. These fractures (Fig. 14-26) present a special challenge because the inferior alveolar vascular supply to the bone is severely compromised, there is little cancellous bone (with associated osteoblastic endosteum) for repair, and the fractures usually occur in older adults, in whom the normal healing potential can be retarded. Open reduction requires stripping of the covering periosteum, which further inhibits osteogenesis. Closed reduction with the use of a mandibular prosthesis held in place by circummandibular wires offers a more conservative approach if amendable to the clinical presentation. If open reduction is necessary, a supplemental bone graft across the fracture site should be considered in addition to minimal periosteal stripping. In severely atrophic edentulous ridges, open reduction with primary bone grafting may be indicated, because proper alignment of the fractured ends of bone may be impossible because of the small cross-sectional diameter of the mandibular body.

Mandibular Fractures in Children With Developing Dentition. Open reduction with wires or plates carries the risk of damage to developing tooth buds, which occupy a major portion of the mandible in children (Fig. 14-27). If open reduction is necessary because of gross



FIGURE 14-27 **A,** Mandibular fracture in a child with developing dentition. **B,** Treatment with closed reduction and lingual splint.

displacement of the fragments, fine wires should be placed at the most inferior border of the mandible, engaging only the cortex. Closed reduction is indicated with special wiring techniques (continuous loop or Risdon wire) or fabricated acrylic splints maintained by circummandibular wiring. A special concern in children is fractures of the mandibular condyle. Damage to the condylar growth center can result in retarded growth of the mandible and facial asymmetry. Intracapsular condylar fractures in children can also lead to ankylosis of the joint, so early mobilization (after 7 to 10 days of intermaxillary fixation) is indicated.

Coronoid Process Fractures. Fractures of the coronoid process are rarely isolated and are usually simple and linear, with little displacement, although with extreme trauma the bone may be displaced into the temporal fossa. Isolated fractures of the coronoid process cause trismus and swelling in the region of the zygomatic arch. There may be swelling in the retromolar area and a lateral crossbite. Treatment is usually initiated only if the occlusion is compromised or if the fractured coronoid process impinges on the zygomatic arch, inhibiting mandibular movement.[148]

Condylar Fractures. Most condylar fractures (Fig. 14-28) can and should be treated via closed techniques if the occlusion is not compromised. Early jaw mobilization and physical therapy are indicated to prevent ankylosis or limited jaw movements. This subject is covered in detail in Chapter 15.

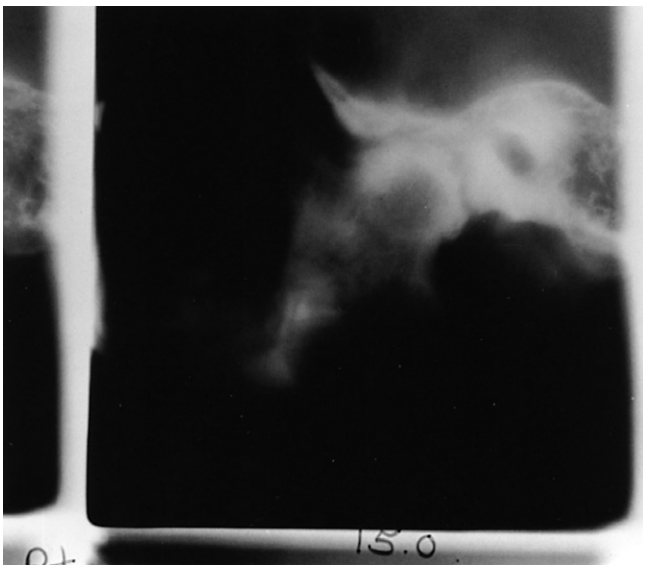


FIGURE 14-28 Condylar fractures can result in ankylosis if they are improperly treated or go untreated.

INDICATIONS FOR OPEN REDUCTION

The indications for open reduction as outlined in the following examples are frequently based on the anatomy of the fracture rather than the severity of the injury or the amount of displacement. Control of the edentulous or proximal segment is the overriding factor in selecting

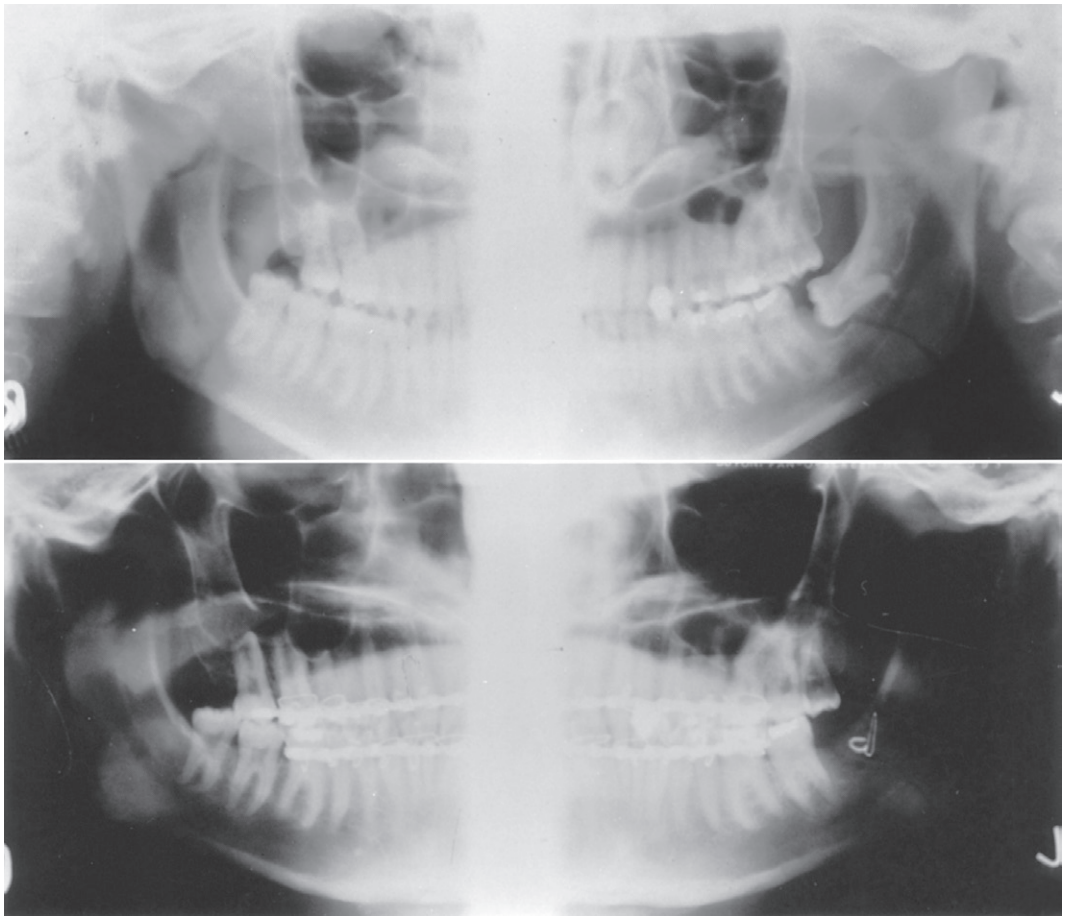


FIGURE 14-29 Indication for open reduction. The proximal segment is minimally displaced superiorly, a third molar is in the proximal segment, and reduction cannot be maintained without intraosseous wires, screws, or plating.

this surgical option. Additional factors such as the necessity of early mobilization or medical considerations such as seizure disorders may dictate open reduction treatment.

Displaced Unfavorable Fractures Through the Angle of the Mandible. Open reduction is indicated for this fracture when the proximal fragment is displaced superiorly or medially and reduction cannot be maintained without intraosseous wires, screws, or plating (Fig. 14-29).

Displaced Unfavorable Fractures of the Body or Parasymphyseal Region of the Mandible. The mylohyoid, digastric, geniohyoid, and genioglossus muscles may further displace the fragments (Fig. 14-30). When treated with closed reduction, parasymphyseal fractures tend to open at the inferior border, with the superior aspects of the mandibular segments rotating medially at the point of fixation. With medial rotation of the body of the mandible, the lingual cusps of all premolars and molars move out of occlusal contact. If the constriction is not corrected, masticatory inefficiency and abnormal periodontal changes occur.[149,150]

Multiple Fractures of the Facial Bones. In multiple fractures of the facial bones, open fixation of the mandibular segments provides a stable base for restoration.

Midface Fractures and Displaced Bilateral Condylar Fractures. With midface fractures and displaced bilateral condylar fractures, one of the condylar fractures should be opened to establish the vertical dimension of the face. If this procedure is not done, any type of suspension wiring, such as that from the frontozygomatic suture area to the mandible, would tend to collapse and telescope the fractures of the midface and condyles, resulting in a foreshortened facial appearance.

Fractures of an Edentulous Mandible With Severe Displacement of the Fracture Fragments. In fractures of an edentulous mandible with severe displacement of the fracture fragments, open reduction should be considered to reestablish continuity of the mandible (Fig. 14-31). The technique is especially helpful with a nonatrophic mandible when there are no dentures, so the occlusion is not an immediate concern. In this situation, plating of the mandible without IMF should be a strong possibility. As the mandible becomes extremely atrophic, consideration must be given to the status of blood supply to the bone and the effect of an open surgical procedure on the compromised vascularity. Supplemental bone grafts have to be considered for extremely atrophic mandibular fractures.

Edentulous Maxilla Opposing a Mandibular Fracture. When a maxilla opposing a mandibular fracture is edentulous or contains insufficient teeth to allow intermaxillary fixation, open reduction should be considered. Open reduction with rigid fixation of the mandibular fractures would eliminate the need for IMF. However, if the patient's condition warrants closed reduction, a prosthesis could be constructed for the maxilla, it could

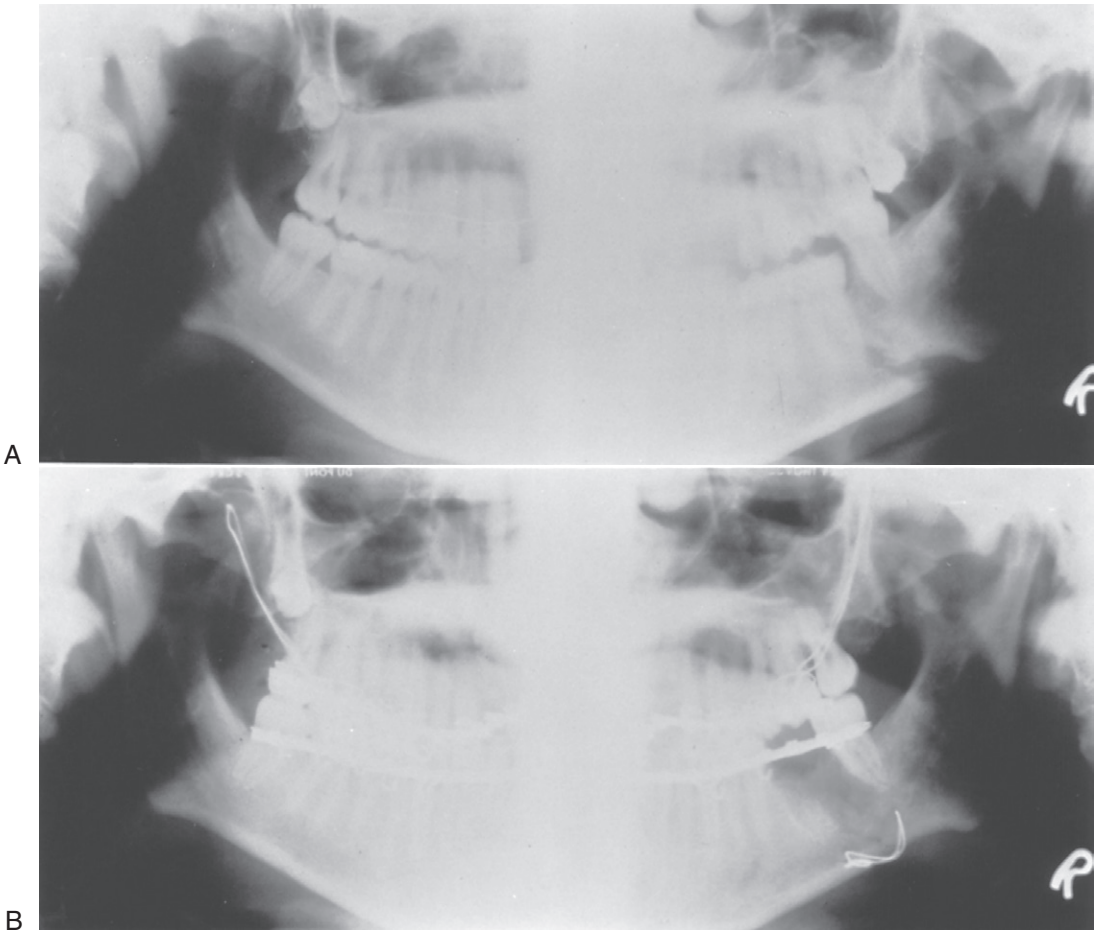


FIGURE 14-30 Displaced unfavorable fractures of the body region frequently require open reduction.

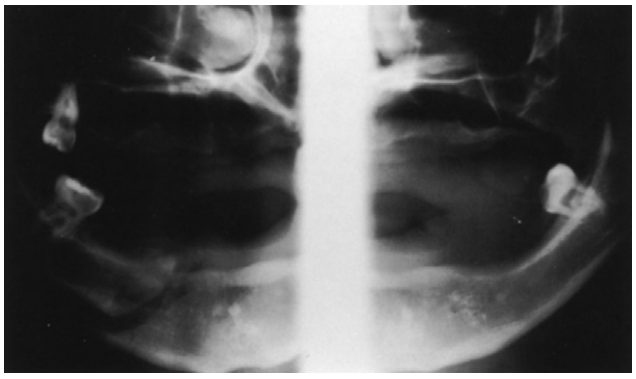


FIGURE 14-31 Edentulous or partially edentulous mandibles that are not atrophic but are severely displaced should be opened.

be stabilized with palatal screws or circumzygomatic wires, and routine IMF could be used to treat the fractured mandible.

Delay of Treatment and Interposition of Soft Tissue Between Noncontacting Displaced Fracture Fragments. When treatment has been delayed and soft tissue becomes interposed between noncontacting displaced fracture fragments, open reduction should be used. There are cases in which the treatment of mandibular fractures is delayed because of head injury or other serious medical problems so, with time, connective tissue grows between the bone fragments, inhibiting osteogenesis. When treatment is finally initiated, scar tissue must be removed and treatment completed via an open approach.

Malunion. When a poor result is obtained after mandibular fracture treatment, various types of osteotomies will have to be done via open surgical approaches to correct the deficiencies.

Special Systemic Conditions Contraindicating Intermaxillary Fixation. There are situations in which mandibular functional movement is necessary, and open rigid fixation techniques can provide that option. For example, patients with difficult to control seizures, psychiatric or neurologic problems, compromised pulmonary functions, and eating or gastrointestinal disorders could benefit from open rigid fixation techniques.

TREATMENT OF MANDIBULAR FRACTURES

Hundreds of techniques are available for treating mandibular fractures, as noted earlier in the section on history. However, state of the art treatment methods are currently limited and well defined. It is important to realize that establishment of occlusion is imperative for the successful treatment of mandibular fractures.

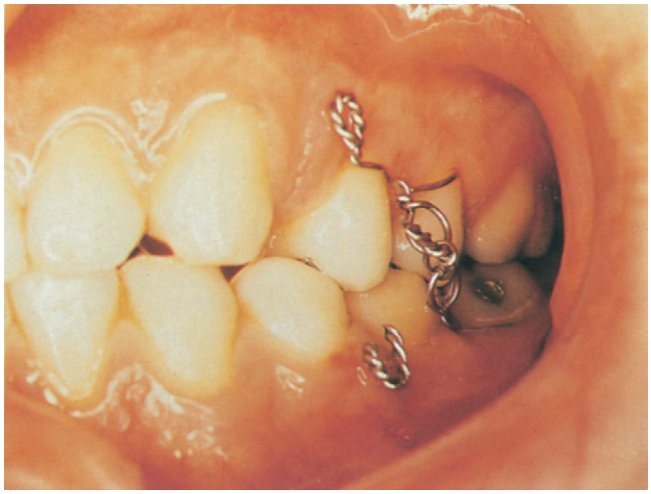


FIGURE 14-32 Ivy loops are effective for certain types of fractures.

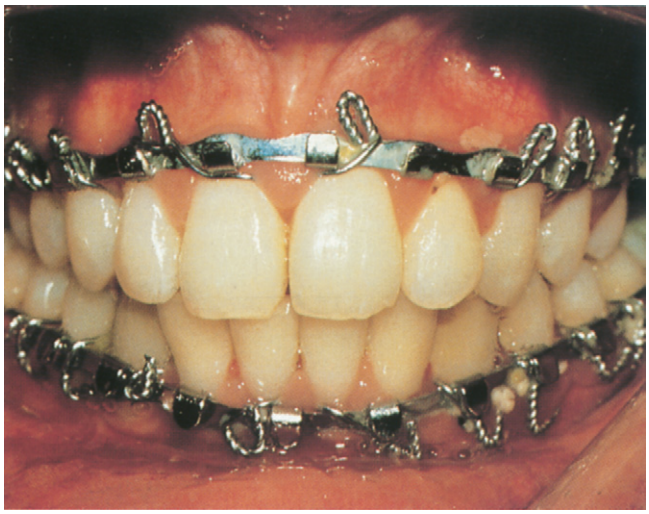


FIGURE 14-33 Arch bars are versatile and frequently used in the treatment of mandibular fractures.

Closed Reduction and Fixation of Dentulous Maxilla and Mandible

Bridle Wire. Hippocrates was the first to advocate the use of wires for the reduction of mandibular fractures. His technique of placing a single wire around the teeth adjacent to the fracture still has its place in modern oral surgery. A simple bridle wire placed around the adjacent teeth of a mandibular fracture can temporarily stabilize a flailed mandibular segment. This in itself helps prevent further soft tissue damage, aids in protecting the airway, helps alleviate pain from the two segments moving against each other, and assists in preventing the muscle cramping that is associated with unstable segments.

Equipment Needed

- Local anesthetic
- Needle driver or needle holder
- 24- or 26-gauge stainless steel wire

Procedure. After adequate local anesthetic has been administered, the two segments are manually reduced. The wire is passed around the necks of the teeth and the fracture loosely approximated. While manually stabilizing the fracture, the operator achieves further reduction by tightening the wire in a clockwise fashion. In the event that the adjacent teeth are loose, decayed, or avulsed, the operator can use the nearest stable teeth.

Ivy Loops. Ivy loops are a quick and easy way of obtaining maxillomandibular fixation. The loop is constructed of 24-gauge wire and passed interproximally to two stable teeth. The ends of the wire are first brought around to the mesial and distal sides of the teeth. The distal wire is then delivered under the loop and tightened to the mesial wire in an apical direction. The loop is then tightened to adapt it into the interproximal space.

Maxillomandibular fixation between Ivy loops can then be achieved by various methods. A smaller gauge wire can be passed through the loops and tightened. To obtain adequate fixation with this method, the loops should be short enough that they do not overlap and create an unstable fixation. Another method involves passing a smaller gauge wire around the lugs created by the loops (Fig. 14-32).

Arch Bars. There are a variety of arch bars available for achieving maxillomandibular fixation. Placement of arch bars can be a difficult task, depending on the dentition present and their stability in the traumatized mandible. Here we discuss placement of arch bars on a full intact dentition for reasons of simplicity and instruction. Later, we shall discuss the placement of arch bars on some different combinations of tooth loss (edentia).

Equipment Needed

- Local anesthetic
- Arch bars
- 24- and 26-gauge wire
- Needle drivers

Procedure. The first step in placement is measurement of the arch bar. The bar is usually placed two teeth proximal from the fracture. The bar is traditionally placed from a point distal to the first molar to a point distal to the first molar on the opposite side.

Wire is the next consideration; 24-gauge wire is recommended for the circumdental wires, and 26-gauge wire is used for the box wires that provide the maxillomandibular fixation. The first circumdental wires placed are usually on the second premolars. The measured arch bar is then placed in the loops of the wires and the wires are loosely secured. Wiring then takes place from midline to posterior to avoid excess arch bar in the anterior of the arch. After placement of the circumdental wires and gross reduction of the fractured segments, they are tightened in the same fashion—from midline to posterior (Fig. 14-33). Some have suggested that the mobile fracture segment should be tightened last, after maxillomandibular fixation. Adaptation of the arch bars to the interdental spaces helps maximize tooth to arch bar contact and helps prevent loosening of the arch bar. The box wires are placed and occlusion is obtained. The circumdental wires are tightened and the rosettes are formed. Box wires are then fully tightened and maxillomandibular fixation is achieved. The goal is to have proper occlusion before complete tightening of interdental wires.

The Erich arch bar and, occasionally, eyelet (Ivy loop) and continuous loop (Stout) wiring are used for

intermaxillary fixation. Other modalities more rarely used are the Risdon wire and intermaxillary fixation screws.[151]

Risdon Wire. Use of a Risdon wire can be advantageous when treating primary and early mixed dentition. Often, because of the anatomy of the teeth at this stage of development, the design of conventional arch bars does not allow for secure maxillomandibular fixation. There are two separate methods for using the Risdon wire, one continuous cable used on the entire arch or two separate cables joined at the midline of the arch.

A 24- or 26-gauge wire is passed around the distalmost strong tooth, leaving both ends on the buccal side. The wire is then twisted to create a cable, which is fastened in the same manner to the contralateral distalmost strong tooth.[152] Alternatively, a separate 24- or 26-gauge wire can be twisted around both distal strong teeth and the two separately twisted strands can then be crossed at the midline and twisted around each other.[119] Then, as noted earlier, a circumdental wire is passed above and below the twisted cable to ligate each tooth independently. A small rosette is formed and elastics are used to create maxillomandibular fixation (Fig. 14-34).

Intermaxillary Fixation Screws. The application of arch bars and insertion of wires in the interdental spaces increase the chance of accidental skin puncture, hence increasing the chance of transmission of HIV and viral hepatitis. The use of cortical bone screw fixation for the treatment of mandibular fractures was first described by Karlis and Glickman.[150] The advantages of these intermaxillary fixation screws are as follows:

1. Ease of application
2. Decreased operating time hence diminished overall cost.
3. Decreased risk of disease of transmission.
4. No impingement of cervical gingiva

Equipment Needed

- Local anesthetic
- 24-gauge wire
- IMF screws
- Screwdrivers
- Needle drivers

Procedure. This procedure can be performed under local anesthetic or, if the patient cannot tolerate it, under IV sedation or general anesthetic. The mucosa just medial to the canine region is injected with local anesthetic in the upper and lower dentitions. Next, the self-tapping IMF screw, 8 to 12 mm in length, is inserted in the bone in a transmucosal fashion. Alternatively, the mucosa overlying the site can be incised with a no. 15 blade and pilot holes can be drilled to facilitate insertion. Close attention should be paid to the location of the mental nerves and root apices to avoid inadvertent iatrogenic injury. Next, IMF is obtained using 24-gauge wires.

The disadvantages of IMF screws are the lack of tension band effect and interference with internal fixation plates. Therefore, we recommend their use only in minimally displaced fractures.

The screws can be removed under local anesthetic in the office. Usually, the mucosa around the screw will grow to cover the head of the screws, necessitating an incision and a minimal amount of dissection to retrieve them.

Partially Edentulous Mandible

If the patient is partially edentulous, a preexisting partial denture can be wired to either jaw using circummandibular or circumzygomatic wiring techniques. If no prosthesis is available, impressions can be taken and acrylic blocks can be fabricated, incorporated with an arch wire, and applied to the remaining teeth. The acrylic prosthesis is wired to the mandible with circummandibular wires.

Edentulous Mandible

If the patient is completely edentulous, dentures can be wired to the jaws using circummandibular and circumzygomatic wires or, in the case of a maxillary denture, palatal screw fixation can be done to hold the denture (Fig. 14-35). Another method of fixation consists of placing intermaxillary fixation screws through the

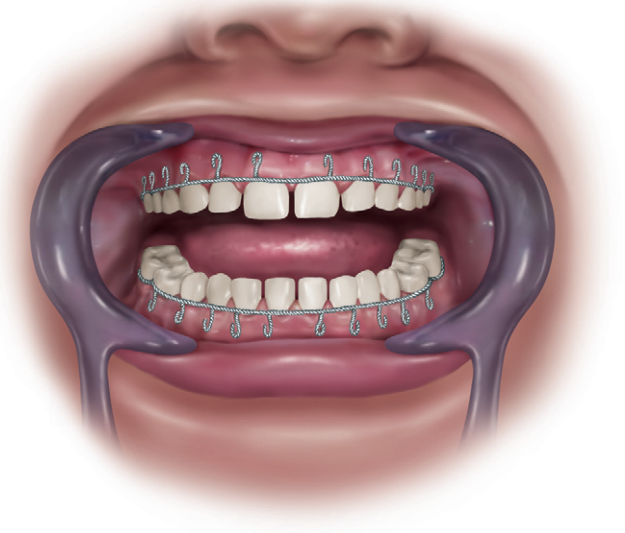


FIGURE 14-34 Risdon wire.

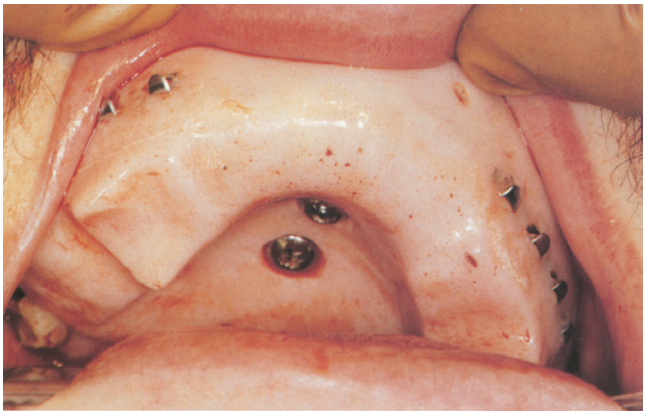


FIGURE 14-35 A palatal screw can be used to hold the splint or denture in place.

preexisting denture to stabilize it and then place the patient in intermaxillary fixations.

If dentures are not available, impressions are taken of the jaws and acrylic base plates are processed and used as dentures. An arch bar can also be processed into the dentures or holes can be placed into the flange of the denture for intermaxillary wires. Prosthetic incisor teeth can be removed for existing dentures, and space can be made in the acrylic to allow food intake (Gunning splint) (Figs. 14-36 to 14-38).

A technique that should probably be considered as semiclosed (or semiopen) is the biphasic pin fixation (Roger Anderson or Joe Hall Morris appliance) technique. In this approach, pins are placed on either side of the fracture site and, after fracture reduction, an external appliance, followed by an acrylic bar, is placed to maintain the bone fragments in their proper position (Figs. 14-39 and 14-40). A variation of this technique, in which a monophasic appliance is used, has been discussed.[153,154] Although this technique can be used for dentulous or edentulous patients, it is most valuable in the following situations: (1) in edentulous cases, to maintain space where bone is missing because of severe trauma or resection; (2) in severely comminuted fractures; and (3) when intermaxillary or rigid fixation

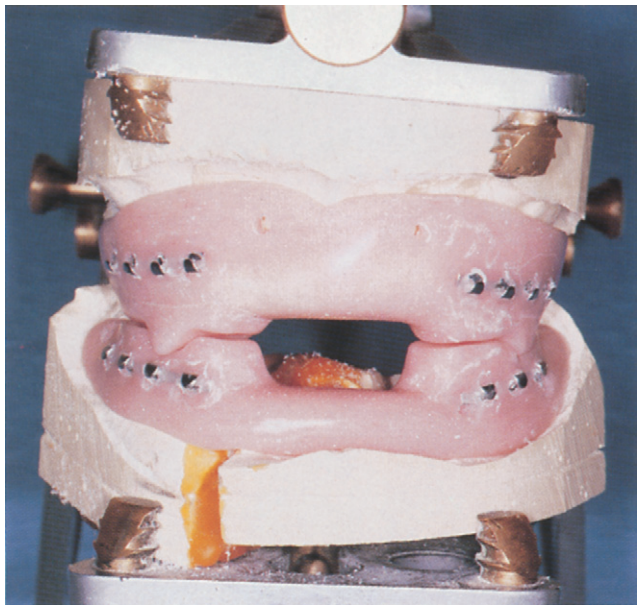


FIGURE 14-36 Gunning splint used for edentulous mandibular fractures.

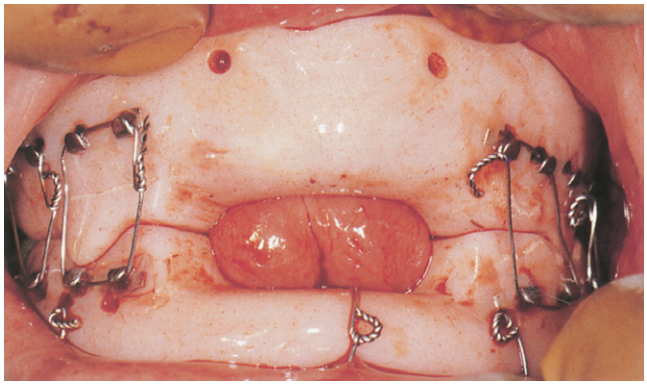


FIGURE 14-37 Gunning splint used for edentulous mandibular fractures.

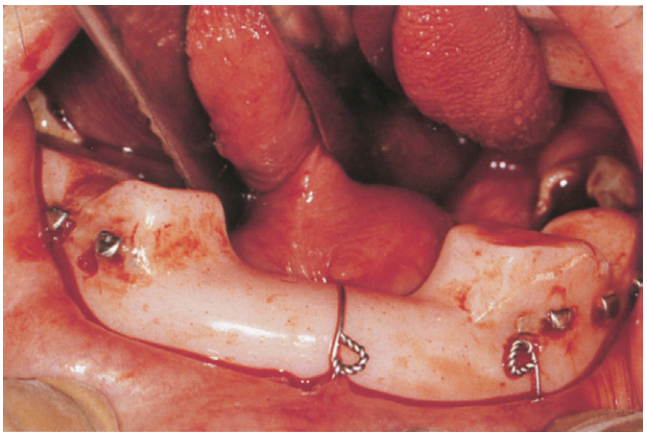


FIGURE 14-38 Gunning splint used for edentulous mandibular fractures.



FIGURE 14-39 The biphasic pin fixation technique is very useful in fractures for which there is comminution or infection.

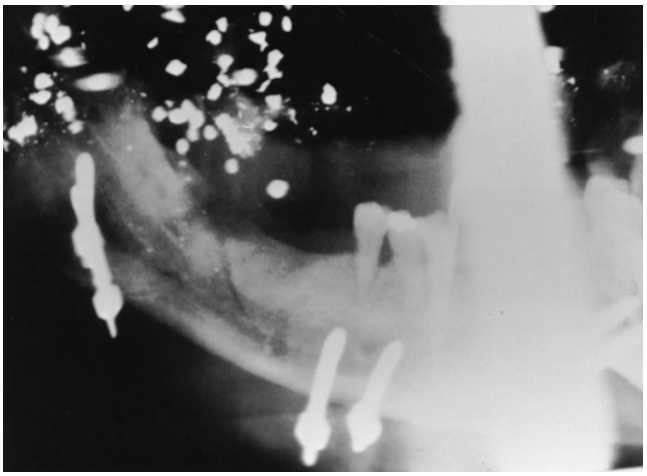


FIGURE 14-40 Biphasic pin fixation technique.

cannot be used. A more detailed discussion of managing edentulous patients with fractures is presented in Chapter 30.

OPEN REDUCTION AND FIXATION PROCEDURES

Just as there are a multitude of techniques for closed reduction and fixation, there are also many methods of open reduction and types of orthopedic hardware available for establishing bone approximation. As noted, open reduction should be used in specific situations because of the morbidity attendant with open procedures.

SURGICAL APPROACHES

Before operating on a mandibular fracture, the surgeon should include the angle of the mouth in the operating field to monitor facial nerve activity and ensure that the anesthesiologist has not paralyzed the patient for an extended period of time. Factors used to establish the location of incision include fracture location, skin lines, and nerve position.

Submandibular Approach

The submandibular approach was first described in 1934 by Risdon.[155] The skin incision is 4 to 5 cm in length, 2 cm below the angle of the mandible. Optimally, the skin incision should be positioned in an existing skin crease to hide the scar and should be made at right angles to the skin surface (Fig. 14-41). The subcutaneous fat and superficial fascia are dissected to reach the platysma muscle. The platysma is sharply dissected to reach the superficial layer of the deep cervical fascia. The marginal mandibular branch of the facial nerve lies just deep to this layer, so it is important to know its course. Dingman and Grabb[155] dissected out the nerve in 100 cases. In 81% of patients, the nerve passed above the inferior border of the mandible proximally to where the facial artery crossed the inferior border. In 19% of patients, the nerve took a downward course, with the lowest being 1 cm below the inferior border. Whether anterior or posterior to the facial artery, all branches of the nerve innervated the depressors of the lower lip, past the inferior border of the mandible. Those branches remaining below the inferior border distal to the facial artery innervated the platysma muscle. The marginal mandibular branch consisted of two branches in 62% and one branch in 21% of patients, 8% had three branches, and 3% had three or more branches.

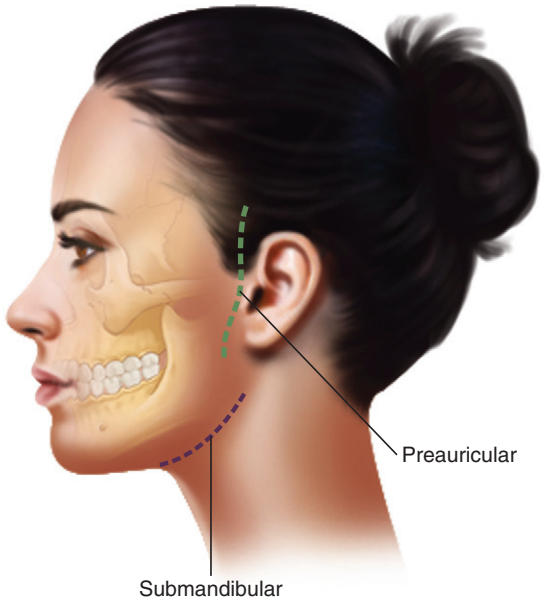


FIGURE 14-41 Submandibular and preauricular incisions.

The dissection to bone is carried through the deep cervical fascia by the surgeon, carefully using a nerve stimulator. The dissection is continued beneath the fascia to the inferior border of the mandible. The submandibular gland and its capsule will become evident, and the lower pole of the parotid may be encountered. The dissection is carried to the masseter muscle, with the surgeon taking care to retract the nerve fibers superiorly. Once the muscle is encountered, it is sharply divided at the inferior border to expose the bone. The muscle, periosteum, and soft tissue are retracted superiorly to expose the body, ramus, and fracture site. If facial vessels cannot be retracted successfully, they may be divided and ligated. Typically, the submandibular lymph node can be identified adjacent to the facial vessels. Exposure can be increased and closure enhanced by dissecting the medial pterygoid and stylomandibular ligaments from the inferior and posterior borders. Further exposure can be obtained by distracting the angle and inferior border with a wire or bone forceps. The submandibular gland and its capsule are usually located just inferior to the inferior border of the mandible. The parotid gland is generally posterior to the ramus but may wrap around the inferior angle. The capsules of both should be avoided during dissection. Disruption of gland parenchyma may lead to sialoceles or salivary fistulas.

Retromandibular Approach

Hinds and Girotti[156] first described the retromandibular approach in 1967. This approach was basically a variation of the submandibular approach except the incision was approximately 3 cm above the submandibular incision. The incision is also described by curving behind the angle of the mandible. The incision is made to encounter the parotid, masseteric, and deep cervical fascia (Fig. 14-42). The dissection is then extended anteriorly through the deep cervical fascia with the surgeon using nerve stimulation. The incision to bone through the masseter muscle is usually between the marginal mandibular and buccal branches of the facial nerve. The muscle and periosteum are incised over the angle instead of the inferior border. The soft tissue and nerve fibers are then retracted superiorly. This incision gives superior access to the ramus and subcondylar region of the mandible.

Preauricular Approach

This allows for exposure of the TMJ and is easily extended to allow access to temporal anatomy. The incision commonly encounters the superficial temporal vessels, which

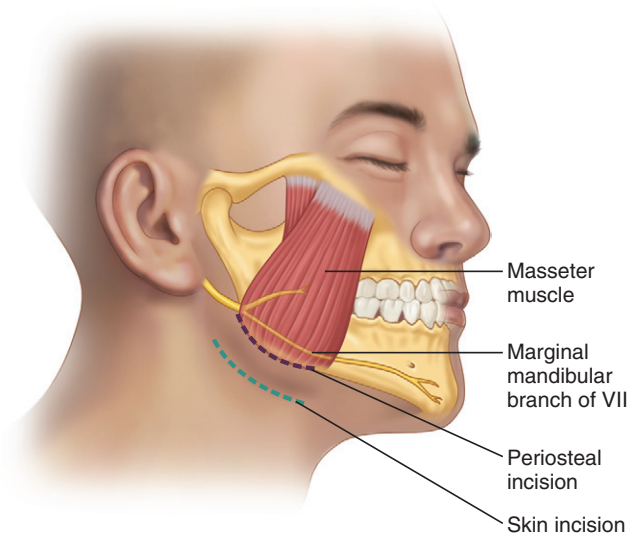


FIGURE 14-42 Retromandibular incision. The Risdon approach illustrates the relationship of the marginal mandibular branch of the facial nerve (VII) to the inferior border of the mandible and periosteal incision *(dotted line).*

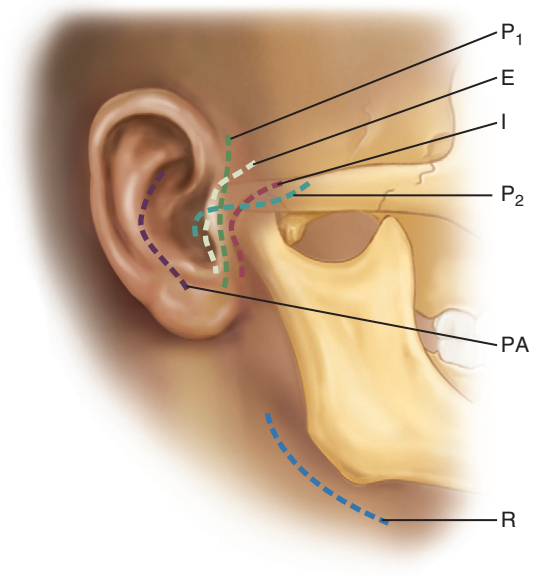


FIGURE 14-43 Preauricular incision. Surgical approaches to the posterior mandible and temporomandibular joint. *E,* Endaural approach; *I,* inverted hockey stick approach; P_1, P_2, preauricular approach; *PA,* postauricular approach; *R,* Risdon approach.

can be avoided by incising along the preauricular cartilage.

The incision is made sharply in the preauricular folds, approximately 2.5 to 3.5 cm in length, as described by Thoma[157] and Rowe[158] (Fig. 14-43; see Fig. 14-41). The preauricular fold is identified by pushing the ear and tragus forward. The incision extends at a 45-degree angle to the zygoma, from the superior aspect of the ear to the inferior attachment of the ear and cheek. Care should be taken not to extend the incision inferiorly because it might encounter the facial nerve as it enters the posterior border of the parotid gland. The incision and dissection should extend just superficial to the perichondrium. If this plane is followed, it is unlikely that the temporal vessels will be encountered and retracted anteriorly. The dissection is carried anteriorly until the temporal fascia is encountered at the superior portion of the incision. Care should be taken not to perforate into the internal auditory canal as it courses anteromedially. The superficial temporal fascia is continuous with the subcutaneous musculoaponeurotic system above the zygomatic arch and extends superiorly to the galea. The undersurface of this layer is where the superficial temporal vessels, auriculotemporal nerve, and facial nerve are found. All these layers should be retracted anteriorly with the soft tissue flap. The facial nerve has been described as crossing the zygomatic arch 0.8 to 3.5 cm (mean, 2 cm) anterior to the concavity of the auditory canal. A total of 50% have been found to cross at a point less than 2 cm. The distance from the auditory concavity and lateral articular tubercle is also 2 cm (1.6 to 2.5 cm) and should be the anteriormost point of dissection needed for access.

The temporal fascia divides 2 cm above the zygomatic arch and is an extension of the parotidomasseteric fascia. The lateral layer is contiguous with the lateral periosteum and the deep layer is contiguous with the medial periosteum. Between these two layers is fat, temporal vessels, and the zygomaticotemporal branch of the maxillary nerve. A sharp incision is made through the periosteum 2 to 3 cm anterior to the skin incision. The periosteum, vessels, and nerves are then retracted anteriorly. The temporal and zygomatic branches of the facial nerve lie within the lateral periosteum-fascia layer. This dissection is carried anteriorly to the lateral eminence, exposing the joint capsule. Fracture location dictates whether the capsule is opened.

Endaural Approach

The endaural incision is started in the skin crease between the anterior helical cartilage and tissue, extended downward in the cleft between the tragus and helix and inward approximately 5 mm along the roof of the auditory canal. As the incision deepens, it is carried anteriorly through the tragal cartilage. After the tragal cartilage is released, the incision is deepened along the auricular cartilage, similar to the preauricular approach described earlier.

Intraoral Access

Symphysis and Parasymphysis. Anterior mandibular fractures may be accessed via intraoral incisions. First, the incision region is infiltrated with local anesthetic and vasoconstrictors. The lip is retracted and a curvilinear incision is made perpendicular to the mucosal surface. It is important to carry the incision out into the lip, leaving at least 1 cm of mucosa attached to the gingiva. The mentalis muscle, now visible, should be incised perpendicular to bone, leaving a flap of muscle attached to bone for closure. The dissection is carried

subperiosteally to identify the mental neurovascular bundle, approximately midway between the alveolar ridge and inferior border, below the second premolar or slightly anterior. The fracture site is identified and reduced. The surgical site is closed in layers. The mentalis muscle is secured to the remaining pedicle with interrupted sutures while an assistant reduces the wound with finger pressure from below. The mucosa is then closed and an adhesive bandage is applied to the chin to support the mentalis muscle and thus prevent drooping.

Body, Angle, and Ramus. After administration of adequate local anesthetic and vasoconstrictors, the cheek is retracted laterally. The mucosa is incised to the bone with the blade positioned perpendicular to the bone to avoid the mental nerve. The incision is made approximately 5 mm from the mucogingival junction to allow adequate mobile tissue for closure. The proximal portion of the incision should be carried along the external oblique ridge, only as high as the mandibular occlusal plane. Extending the incision higher predisposes the buccal fat pad to prolapsing onto the surgical field. The anterior surface of the ramus can then be exposed by stripping the buccinator and temporal tendon with a notched angled retractor and periosteal elevator. Once the coronoid is exposed, a curved Kocher clamp can be applied, which will act as a self-retaining retractor. Dissection in this manner, subperiosteally, keeps the buccal fat pad out of the field. The masseter muscle can then be elevated with periosteal elevators and J strippers. The entire ramus and subcondylar region can now be exposed with the placement of Bauer retractors in the sigmoid and antegonial notches. The masseter can also be retracted with the placement of LeVasseur-Merrill retractors.

Wire Osteosynthesis

Historically, open techniques were performed through a skin incision or laceration, and wires were generally used to maintain the fracture fragments. With the advent of modern orthognathic surgery, intraoral open surgical approaches have become the standard and, although wire osteosynthesis is still widely used in the United States, techniques developed in Europe that use bone plates and screws for rigid fixation have become widely accepted here. Wires may be simpler to place and usually will maintain the bone fragments and prevent bone displacement by muscle pull until healing occurs. However, wires lack rigidity, directional control, and surface to bone surface contact area to maintain rigidity under function, so IMF must be used. On the other hand, with screw or plate rigid fixation, IMF is usually unnecessary.[159-162]

Wire osteosynthesis is most commonly used for angle fractures at the superior border of the mandible; the wire is placed via an intraoral approach. Concomitant removal of an impacted third molar allows excellent access and easy placement of the wire. Parasymphyseal or midsymphyseal fractures are also often reduced and fixed, with wire osteosynthesis placed via an intraoral approach. The wire is positioned beneath the teeth, inferior alveolar canal, and mental foramen. As noted, fractures in this area are often oblique, causing telescoping. In this case, the wire must be placed through both segments of bone, which may be millimeters away from the buccal fracture line. However, if the fracture is perpendicular to the buccal surface of the mandible, wire should be placed in a figure-eight fashion to cradle the lower border and to bring the two edges of the fracture together.

RIGID FIXATION

Although the subject of rigid fixation is discussed thoroughly elsewhere in this text, a short summary of the principles and techniques is included here for completeness. Jones and Van Sickels[162] reported on this subject and presented a thorough literature review.

The use of bone screws and compression plates for the treatment of mandibular fractures is derived from concepts developed in orthopedic surgery. Currently, titanium and bioabsorbable osteosynthesis systems are available for rigid fixation. Essentially, three goals are desired—anatomic reduction, fracture fragment compression, and rigid immobilization. Anatomic reduction is thought to promote primary bone repair, resulting in direct lamellar bone formation in medullary bone without a cartilaginous phase. In cortical bone, longitudinal growth of capillaries and osteogenic cells across the fracture line occurs by way of tunnels.

Compression plating requires absolute stability for bone healing. In contrast, locking plates function as internal fixators with multiple anchor points. Greiwe and Archdeacon have explained that "this type of fixed-angle device converts axial loads across the bone to compressive forces across fracture sites, minimizing gap length and strain."[163]

The primary goal of a bone plate should be to provide maximum stability in the bone fracture region with a minimum amount of implanted material. The eccentric dynamic compression plates with eccentrically positioned holes create compression across a fracture. The screws placed at an angle in the holes tend to converge as they are tightened, bringing the bone fragments together. Locking plates are designed to be slightly convex, so that when they are tightened on the buccal surface of the bone, compression occurs on the lingual surface. In addition, this method was designed to minimize biologic damage, thereby promoting earlier callus formation. The plates are held with monocortical screws (these present less danger to nerve vessels and tooth roots) or bicortical screws (enhanced stability). Tapped screw holes allow maximal contact between the screw and bone surface area. Self-tapping screws are more easily applied.

A disadvantage of traditional compression plates is that the plate must be perfectly adapted to the underlying bone to achieve the desired result. Hence, the compression of the undersurface of the plate to the cortical bone has been shown to disrupt the underlying cortical blood supply, which results in bone resorption.[164,165] To overcome this, the concept of a locking bone plate was introduced. In this technique, the screws are locked to the plate to allow stable fixation without compression of the plate against the bone.[166]

In an experimental study, Söderholm et al have shown greater potential for stability with locking screw-plate

systems.[167] This system allows for a minimum number of screws to be placed without the need for a perfect adaptation of the plate against the bony surface. Another advantage of this system is that the screw is unlikely to loosen from the bone because it is screwed into the plate.[165] Studies have shown infection rates varying from 3.6% to 28%.[168-174]

Lag screws have been described for use in oblique fractures. A lag screw has threads on half the shaft, with the portion below the head being smooth so that it will not engage. This allows the inner segment to be compressed against the outer segment. The lag screw technique is accomplished by preparing the outer segment's drill hole slightly larger than the thread diameter. This is called the gliding hole. The threads only engage the inner segment and compress it against the outer segment. This technique of bone fixation is stable enough so that maxillomandibular fixation is not needed. Typically, two to three screws are placed to prevent rotational forces, and the screws must diverge at a minimum of 7%.

Of paramount importance to many of these systems is the concept of a tension band across the mandible. Because of muscle actions, different areas of the mandible are under different forces at the same time (e.g., compression, tension, or neutral). Metal plates positioned over areas of maximal tension allow equal distribution of forces over the fracture surface, eliminating displacement by muscle action. As expected, these sophisticated techniques are not without difficulty, as discussed in the following section on complications.

Although anatomic reduction, compression, rigid fixation, tension bands, and various metal appliances are important in the treatment of difficult to manage long bone fractures, it has not been shown that these same approaches are vital to the treatment of mandibular fractures. Perhaps the one overriding value of these techniques is the elimination or decreased duration of IMF. Whether the advantages of this technology outweigh its disadvantages has yet to be proven.

ENDOSCOPIC REPAIR

Medical uses of endoscopy date back to the era of Hippocrates. With the advent of the flexible endoscope and fiberoptics, endoscopic techniques have revolutionized a number of approaches in the surgical field. The endoscopic approach to the treatment of abdominal and gynecologic disorders has been well established.[172] Initial applications in craniomaxillofacial surgery were for aesthetic procedures, such as the brow-lifting procedure and forehead recontouring.[175-177] Uses were expanded within the field to treat orbital wall fractures,[178-183] distraction osteogenesis,[180,181] zygoma fractures,[186,187] frontal sinus fractures,[188-190] cranial synostosis,[191,192] and subcondylar fractures.[193-197] By using the endoscopic technique, the risk of damage to the facial nerve and visible scarring is minimized. For our purposes here, the endoscopic technique is mostly used for the repair of subcondylar fractures (discussed elsewhere in this text), but it can also be used for the repair of high ramus fractures. The surgical approach is via a transoral sagittal splitlike incision or an extraoral submandibular incision.

COMPLICATIONS OF MANDIBULAR FRACTURE TREATMENT

Complications following the treatment of mandibular fractures are rare. Orthopedic evidence supports the avoidance of fracture contamination. Intraoral flora, however, offer a unique risk in the management of mandibular fractures. Any surgical intervention, open or closed, can theoretically lead to direct wound contamination. From a literature review and our experience, it appears that the most common complication is infection or osteomyelitis. Reported cases also mention hematoma or psuedoaneurysm formation and auditory canal hemorrhage.[198,199] Contributing factors seem to be preoperative, perioperative, and postoperative oral hygiene, presence of teeth in the line of fracture, alcoholic or metabolic disturbances, prolonged time before treatment, patient poor compliance or noncompliance, displacement of fracture fragments, and probably iatrogenic causes via open fixation procedures.[200-202] Edwards et al[161] have found a strong relationship between the severity of the mandibular fracture and the complication rate.

INFECTION AND TEETH IN THE LINE OF FRACTURE

With variations of incidence of mandibular fractures, evaluation of infections must also be evaluated on the basis of the region and socioeconomic conditions of the population served. Khan,[202] in a study from Harare, Zimbabwe, has reported a 22.5% preoperative incidence of infection associated with facial fractures and a postoperative infection rate of 18%. Abiose,[203] in a study from Ibadan, Nigeria, has reported a preoperative infection rate of 56.25%, but thought that the infection "did not necessarily accompany delay nor unduly complicate definitive treatment." From a historical standpoint, it is interesting to compare current results regarding infection with those reported by Chambers and Scully[204] in an article written about mandibular fracture treatment in India during World War II. Of 124 patients treated for mandibular fractures, 46% had teeth extracted from the line of fracture and 44% had postoperative infections and delayed union.

Kelly and Harrigan,[76] although admitting that "a certain percentage of patients treated were lost in follow-up," reported that 66 cases (2.3%) of a total of 3329 fractures (including ramus, coronoid, and condyle) required a surgical extraoral procedure, necessitating drainage, or sequestrectomy or saucerization. The most common organisms isolated were *Staphylococcus* (60%), *Streptococcus* (20%), and others (14%), including two cases of *Actinomyces* and one of *Klebsiella* infection. Cultures were apparently not done for anaerobes. In a limited study, Rajasuo et al found that most anaerobe specimens (*Actinomyces*, *Campylobacter*, and *Lactobacillus* spp.) were associated with third molar involvement.[205]

In a review of 909 cases of mandibular fractures from Cape Town, South Africa, Melmed and Koonin,[206] found 85 cases of sepsis and 4 cases in which osteomyelitis developed. Of the 85 sepsis cases, 4 occurred with open reduction, 5 were caused by a tooth in the line of fracture, 8 resulted from the application or removal of

Gunning-type splints, 25 resulted from very poor teeth, and 43 (50%) were caused, the authors believed, by a delay of more than 48 hours in seeking treatment after injury. Interestingly, Smith[207] has demonstrated that there was no increase in the incidence of wound dehiscence, wound infection, or delayed union in patients who had delayed osteosynthesis from 2 to 11 days after injury.

Olson et al,[84] in their analysis of 580 cases of fractured mandible, found that 156 (26.9%) had some type of complication, including infection, respiratory disorders, neurologic problems, delayed healing, and nonunion. They said that "Complications are the most common in the vehicular-accident victim who has sustained multiple injuries. The patient who sustains only mandibular fracture with or without facial laceration, seldom experiences complications."

In one of the few prospective studies in the literature, James et al[81] evaluated 253 consecutive patients with 422 mandibular fractures and found a postoperative infection rate of 6.95% (16 of 230 fractures with sufficient follow-up). There were 261 teeth directly associated with mandibular fractures, 39% of which were extracted at the time of treatment. The postoperative infection rate had no bearing on whether the tooth in the line of fracture was extracted or not. They used antibiotic therapy routinely for all patients with compound fractures. Of these patients, 46 did not continue taking antibiotics after hospital discharge and, in 4, postoperative infection developed. Of the 177 fractures that required an open reduction, 12 (6.78%) became infected. Of the 12 infections, 6 were associated with angle fractures in which the tooth in the line of fracture was extracted at the time of surgery. In contrast to the study of Olson et al,[84] most of their infected fractures were related to injuries involving IPV. The authors' data supported the concept that healthy teeth in the line of fracture do not increase the incidence of infection and, in many cases, their aid to stabilization outweighed the consideration of infection. It is interesting that aerobic and anaerobic organisms were obtained from cultures of specimens from the 16 patients with postoperative infections, with alpha-hemolytic streptococci and *Bacteroides* organisms found most often. Most of the organisms cultured were sensitive to penicillin.

Two studies have shown remarkably similar results. Amaratunga[135] found an infection rate of approximately 5%, with or without teeth in the line of fracture (191 patients, 226 fractures). Schneider and Stern[208] found complications, including delayed union, infection, and odontalgia, in 5% of patients who had teeth in the line of fracture (157 patients, 199 fractures). Bernstein and McClurg[41] studied 156 consecutive patients with fractures of the mandible and found that in 20 patients (12.82%), infections developed; 4 of the infections were related to teeth in the line of fracture. Prophylactic antibiotics did not seem to affect the incidence of infection.

Chuong and Donoff[209] found little difference in the incidence of infection when comparing the retention of teeth in the line of fracture with the extraction of teeth in the fracture site (14% versus 11%, respectively). However, they were careful to note that teeth in the line of injury that are significantly mobile, have root exposure in markedly distracted fractures, or interfere with reduction or fixation of the fracture were extracted. These selection criteria are perhaps more stringent than those to which most clinicians would subscribe. Marker et al[210] have demonstrated a 3.5% incidence of infection when closed reduction was used in patients in whom a completely or partially impacted third molar was in the line of fracture. They postulated that the infection rate was low compared with that with rigid fixation because patients were not allowed to move their jaws immediately.

In a study of 26 mandibular fractures, Chan et al[211] has reported that 8 developed complications, including soft tissue infections, osteomyelitis, malunion, and nonunion. A common feature in all these cases was infection of teeth in the line of fracture and poor compliance or noncompliance with the treatment plan. The results of their literature review and retrospective study are presented in Table 14-1.

In a study by Ellis in a sample of 402 patients,[173] a postoperative complication rate of 19% was found in the group containing teeth in the line of fracture and a complication rate of 15.8% in fractures not containing teeth in the line of fracture (p = NS [not significant]). For those fractures associated with a tooth, when the tooth was retained, the incidence of infection was 19.5%. When the tooth was removed, the incidence was 19.0%.[174] In a similar study, Malanchuk and Kopchak observed that noncarious teeth in the line of fracture could not be considered a predisposing factor for the development of infection.[212]

Roed-Petersen and Andreasen[213] found that there was much less pulp necrosis in teeth involved in the fracture site when the fracture was treated within 48 hours (15%

TABLE 14-1 Studies of Mandibular Fractures Involving Teeth

Study	No. of Patients	Teeth in Fracture Line	Infection, Delayed, Nonunion	Incidence of Complications (%)
Neal and Wagner	519	260	87	32
Kahnberg	—	185	14*	13
Wilkie	250	190	—	8
Zallen and Curry	643	64	36*	50 (without antibiotics) 6 (with antibiotics)
Ridell	84	—	20*	4.2
Roed-Peterson	1	110	27*	25
Kromer	113*	60	32	53
Chan et al	26	26	8	30

*Data modified from statistics presented in cited references.
Data from Chan DM, Demuth RJ, Miller SH, Jastak JT: Management of mandibular fracture in unreliable patient populations. *Ann Plast Surg* 13:298, 1984.

versus 37% for those treated later). A study by Kamboozia and Punnia-Moorthy[214] evaluated the dental complications associated with the teeth in the line of mandibular fractures. They demonstrated a statistically significant increase in the incidence of nonvitality of teeth adjacent to and in the line of a fracture when open reduction and internal fixation were performed as opposed to IMF.

Factors Affecting Incidence of Infection and Other Complications

Closed Versus Open Reduction. Factors regarding the location and degree of severity (see earlier) can dictate treatment modality. Earlier reports have noted that surgical experience, along with patient history (e.g., substance abuse, excessive alcohol consumption) can have a significant affect on treatment outcome with either approach. Passeri et al[215] have reported a 13% incidence of infection when using nonrigid fixation on 99 angle fractures in 96 patients. They thought that the high incidence of infection was secondary to the patient population studied and a 3- to 4-day delay in treatment.

Lamphier et al[216] have reviewed 594 mandibular fractures and compared the complications associated with open and closed treatment over a 4-year period. In an urban setting, they found that closed reduction offered a lower incidence of postoperative morbidity when compared with open reduction techniques. Their results showed a 3.2% infection rate in the closed reduction group versus 9.1% in the open reduction group.

In a retrospective review of 100 fractures in 56 patients, Moore et al[82] have found a postoperative infection rate of 9.8% in those managed with open reduction compared with a rate of 8.9% in those treated by closed reduction. There were too few cases to generalize on the effect of teeth in the line of fracture. Surprisingly, nine patients (16.1%) had soft tissue infection.

Terris et al[217] have retrospectively evaluated 183 fractures in 112 patients for the incidence of major complications, including infection, when comparing open reduction with plate osteosynthesis, open reduction with wire osteosynthesis, and closed reduction. Results of this study demonstrated a statistically significant difference in the rate of infection and other major complications between plate osteosynthesis and the other two forms of treatment. The incidence of infection was not significant when wire osteosynthesis was compared with closed reduction. However, a significant difference was found when the frequencies of other complications (malocclusion, delayed union, and inadequate fixation) were measured and compared between these two groups.

Leach and Truelson[218] have reported similar findings when comparing plate osteosynthesis with traditional methods (intermaxillary fixation, wire osteosynthesis, and external fixation). They reported a 30% incidence of infection in the plating group, as opposed to 13% in the traditional group.

Different Rigid Fixation Techniques and Systems. Rigid fixation techniques, as noted, can be categorized into two groups, locking and nonlocking. Ellis et al[219-223] have studied the effects of different plating systems and techniques on mandibular angle fractures; these studies provide us with information on the performance of individual systems and postoperative complication rates when the fracture area remains constant. These studies involved the use of two dynamic compression miniplates (31 fractures), the AO reconstruction plate (52 fractures),[221] two 2.4-mm dynamic compression plates (65 fractures),[222] and two noncompression plates (69 fractures)[223] in the treatment of mandibular angle fractures. All the fractures were treated with open reduction and internal fixation only, using AO-ASIF principles.

The lowest infection rate, 7.5%, was seen in the fractures treated with the AO reconstruction plate and the highest rate, 32%, was found in the 2.4-mm dynamic compression miniplates. The dynamic compression plates and the noncompression plates each had an infection rate around 25%. Interestingly, it was noted that in the group of fractures treated with the 2.4-mm dynamic compression plates, the mandibles that had fixation screw holes tapped had a lower incidence of infection than those that were untapped—29% and 40%, respectively. It can therefore be concluded that smaller plates have an increased rate of infection, possibly because of a lack of providing absolute rigidity. In this series of articles, only the AO reconstruction plate was found to provide a predictably low incidence of complications.

Ellis also examined the outcomes of two 1.0-mm miniplates versus a single larger, stronger 1.25-mm plate in treatment of mandibular symphysis and body fractures.[218] One of the most notable results showed an increased incidence of wound dehiscence and plate exposure in the two-plate group, 6%, versus the single-plate group with 4%. However, the single-plate technique showed an 11% rate of postoperative infection compared with a 7% infection rate with the two-plate technique.

Danda has compared postoperative complications in a randomized study of treatment with two noncompression miniplates versus a single noncompression miniplate in patients with mandibular angle fractures. No significant advantage was seen in the use of two miniplates over a single plate.[224]

With the advent of the locking screw-plate system, infection rates as low as 3.6% have been cited in the literature, confirming the advantages associated with this system.[225]

Antibiotics. Larsen and Nielsen[95] have cited studies showing infection in 36 of 104 patients with mandibular fractures before the use of antibiotics, a 19% incidence of infection in a partially preantibiotic period (1943 to 1953), and a current infection rate of 0%. Their study of 229 patients showed a 0.4% incidence of infection. They attributed their low infection rate to early treatment, thorough wound cleaning before treatment of compound fractures, and prophylactic antibiotics.

In a prospective randomized study (64 patients) of antibiotic usage for the treatment of fractures with teeth in the line of fracture, Zallen and Curry[139] have found a complication rate of 6.25% in patients who received antibiotic coverage compared with a complication rate of 50.33% in those who did not.

Limchayseng[138] has studied 158 mandibular angle fractures and found infection, despite postoperative antibiotics, in 25% of patients in whom third molars were

retained in the line of fracture. In this study, however, 86% of the third molars were removed before fracture reduction and fixation. A higher infection rate was also reported when fractures were moderately or grossly displaced. Fractures treated by rigid fixation showed a higher incidence of infection (28%) than those managed with open reduction with superior border wire fixation (6%); however, fractures with more displacement tended to be treated with rigid fixation.

Current studies, however, question the actual benefit of prophylactic antibiotics. Kyzas has reviewed 31 studies of more than 5000 patients, and ultimately argued that the "overall evidence for the use of prophylactic antibiotics is poor."[199] In a prospective, randomized, double-blind clinical study, Abubaker and Rollert have found no advantage in the use of postoperative oral antibiotics for the treatment of uncomplicated mandibular fractures.[225] Lovato and Wagner,[141] in a retrospective study of 150 patients, that approximately 10% of those in the extended antibiotic group developed an infection whereas 13% of patients became infected in the perioperative group.

Extraoral Versus Intraoral Surgery. With the advent of trocar and cannula systems, extended extraoral incisions have been minimized to small stab incisions. Over a 7-year period, Mehra and Murad treated 98 fractures with an intraoral or transoral approach and 65 fractures with an extraoral approach.[226] Interestingly, the results showed a similar post-perative complication rate of infection between the two modalities, 2% and 1.5% respectively.

Wagner et al[227] have studied the morbidity associated with extraoral open reduction (82 patients, 100 fractures) and found a complication rate of 13%, including a wound or bone infection rate of 10%. They found that 67 fractures were associated with teeth and, in 32 cases, the teeth were removed before reduction. The group with teeth in the line of fracture had 9 of the total of 13 postoperative complications, including three wound infections and six infected fractures that required sequestrectomies.

Kerr[228] has analyzed 755 facial fractures and noted post-treatment infection in three mid-face fractures and 13 mandibular fractures. Of these 13, nine involved extraoral open reduction and lower border wiring. In seven of the nine cases, the offending organism was *Staphylococcus pyogenes,* and the onset was within 4 to 10 days after treatment. The cases were treated with incision and drainage, without removal of the wire. It was suggested that this indicated that the infection had not involved the fracture itself but was a superficial wound infection introduced at the time of operation.

Chuong and Donoff[209] have evaluated their mandibular fracture cases treated via an intraoral approach. Of 372 fractures, 161 were treated by open reduction via an intraoral approach and 23 involved removal of an impacted or partially erupted third molar and placement of an intraoral upper border wire. Three body fractures in edentulous portions of the mandible and five symphyseal fractures were treated by intraoral wire fixation. Four complications were seen in the 31 fractures (12.9%) treated by the intraoral approach. Two cases of dehiscence of the angle, one case of infection of the symphysis, and one case of inadequate reduction and fixation of the angle were noted. In all cases, union progressed in 4 to 5 weeks.

Freihofer and Sailer[229] found a complication rate of 7% in 148 patients with 178 wiring sites approached intraorally, combined with 4 weeks of intermaxillary fixation. Complications included occlusal abnormalities (3%), delayed bone union (3%), pseudarthrosis (0.5%), and osteomyelitis of the fracture site (0.5%).

Wire Osteosynthesis Versus Rigid Fixation. In an extensive review, Theriot et al[230] found the reported infection rate to be variable when comparing wire osteosynthesis with bone plate osteosynthesis in the treatment of mandibular fractures. The range was between 0.5% and 14.7% for wire osteosynthesis, with European studies having the lowest rate of infection (1.1% via an extraoral approach and 0.5% via an intraoral approach). In the United States, the authors reported infection rates from 6.6% to a high of 14.7% when wire osteosynthesis was used in a limited number of patients. The data derived from this review have shown an initial high rate of infection associated with bone plate osteosynthesis, decreasing as the researchers became more proficient with the technique. They also cited studies on the incidence of infection with bone plates—3 of 11 patients infected (27%), 5 of 26 patients infected (19.2%), and 25 infections in 171 patients (14.6%).[156] They also reported on other results, a 3.8% infection rate in a series of 183 patients and a 3.5% infection rate using compression plate osteosynthesis. In their own prospective study of 75 patients with 126 mandibular fractures, they reported infections in 4 of 34 patients with 52 fractures treated by bone plate osteosynthesis and two infections in 41 patients with 74 fractures treated by wire osteosynthesis.

Using AO-ASIF and osteo systems, Tu and Tenhulzen[231] have reported removing the plates in 11.4% of patients because of complications when using the Champy system for mandibular fractures (183 cases). Becker[232] has reported infection (3.8%), malunion (0.5%), nonunion (0.5%), and the need for occlusal equilibration (4.8%) while using the Champy system. In 50 cases, Cawood[233] found infection in 6% and malocclusion in 8%.

Souyris et al[234] have reported a 12-year study using cobalt-chromium alloy (Vitallium) plates with bicortical self-tapping screws placed along the inferior border of the mandible. Postoperative infections occurred in 14.5% of patients (25 of 171), 20 additional patients required removal of the plates because of the late development (months to years) of an inflammatory reaction, and 15% of patients had occlusal discrepancies managed by occlusal equilibration. Using Vitallium cobalt-chromium plates with bicortical self-tapping screws, Luhr[64] has reported an infection rate of 5.7% (105 patients) for extraoral placement and 3.2% (255 patients) for intraoral placement.

In a retrospective analysis, Terris et al[217] have measured the frequency of major complications (e.g., infection, malocclusion, hematoma, inadequate fixation) between plate and wire osteosynthesis. Their study demonstrated a significant difference between the two groups,

30.6% and 10.3%, respectively. Infection was the most frequently reported complication. They failed to mention any incidence of postoperative nerve injury or deficit. In their retrospective study Leach and Truelson have noted that rigid internal fixation is associated with a significant difference in the rate of nerve injury, infection, operative time, and cost when compared with traditional methods.[218] They also noted that significantly fewer patients in the rigid fixation group returned for at least 6 weeks of follow-up.

Ellis has prospectively reviewed three treatment methods over a 12-year period[235]: (1) nonrigid fixation that included 5 to 6 weeks of maxillomandibular fixation; (2) nonrigid but functionally stable fixation using a single miniplate; and (3) rigid fixation using two miniplates. Of 60 patients treated with nonrigid fixation, 32 complained of neurosensory deficit and 9 had a wound problem (e.g., cellulitis, purulence, dehiscence). Of 62 patients treated with one miniplate (nonrigid), 37 complained of neurosensory deficit and 2 had a wound problem. Of 63 patients treated with two miniplates (rigid), 31 complained of neurosensory deficit and 14 had a wound problem. The outcomes showed that the use of a single miniplate generated fewer complications comparatively.

OTHER COMPLICATIONS

Delayed Healing and Nonunion

Delayed healing and nonunion of the mandible are rare and occur as a result of violation of the treatment goals outlined in the previous section. By definition, a nonunion is the lack of osseous union by two or more fracture segments after the usual healing period. Infection is the greatest factor, but severity of the injury, inadequate reduction, lack of fracture stability, uncooperative patients, alcoholism, and metabolic and nutritional deficiencies all play a major role in prolonged healing.

Kelly and Harrigan[76] have reported 34 nonunions of the mandible of 3338 mandibular fractures (1%). Infection accounted for the greatest number of nonunions, and lack of cooperation by the patient (e.g., removal of IMF) was second. The most common site of a nonunion was in the body of the mandible. Bui et al[236] have noted that one subject released maxillomandibular fixation multiple times during the course of treatment; this patient subsequently developed an infection and skewed their complication rate from 6.2% to 8.2%. In the same study, they reviewed 49 patients with mandibular fractures that were accessed through intraoral exposure and reduction using a 2.0-mm, eight-hole strut plate. Although the study had limited participants, an extraordinary result showed absolutely no nonunion or malunion.[237]

On the other hand, Melmed and Koonin[206] had 20 cases of nonunion resulting from 909 fractures of the mandible (2.2%). They believed that alcoholism, delay in seeking treatment, and carious teeth contribute to the problems of management and complications. The prospective study of James et al[81] showed delayed union (no clinically evident union after 8 weeks) in 9 of 253 patients (3.5%) and nonunion in 3 patients (1%). Of the delayed unions, four were in the angle, four were in the body, and one was in the symphysis. Seven patients had open reduction and four had preoperative or postoperative infection. Two nonunions occurred in the angle and one occurred in the body. Moore et al[82] found a nonunion rate of 1.8% in 56 patients with 100 mandibular fractures. The nonunion occurred in a 74-year-old patient with bilateral fractures of an atrophic edentulous mandible. Bernstein and McClurg[41] have reported delayed union in 5 of 156 patients with mandibular fractures. The diagnosis was made by eliciting pain when the fracture site was torqued; treatment consisted of additional IMF time. In a study from Sweden, Heimdahl and Nordenram[94] found two patients with osteitis and osteomyelitis, causing severe bone loss and pseudarthrosis, in 100 patients. They believed that abuse of alcohol or narcotics, or both, resulted in uncooperativeness on the part of patients (23%) and contributed to the nonunion. Chuong and Donoff[209] defined delayed union as mobility of the fracture site after 5 weeks of treatment with maxillomandibular fixation. Using this rather strict criterion, they found that delayed union occurred in 12 of 372 mandibular fractures (3.1%). No cases of nonunion were reported. Haug and Schwimmer[237] have reported 32 cases of fibrous union in 714 patients. They concluded that age, race, gender, mechanism of injury, and noncompliance with antibiotic therapy were not factors contributing to formation of a fibrous union. They determined that self-abusive habits (e.g., alcohol and drug abuse), inadequate immobilization, location of the fracture, teeth in the line of fracture, and postoperative infections all contributed to the development of a fibrous union.

Delayed Healing and Nonunion Related to Fixation Techniques

Bochlogyros[238] has reviewed German studies, compiled the nonunion cases, and categorized them by treatment method: (1) closed reduction and intermaxillary fixation; (2) interosseous wiring; and (3) stable compression plate fixation. The results are shown in Tables 14-2 to 14-4. It was found that the factors predisposing to nonunion are delay in treatment, inadequate immobilization, and osteomyelitis of the fracture site before and after surgery.

TABLE 14-2 Incidence of Nonunion of Fractures of the Mandible After Closed Reduction and Intermaxillary Fixation

Study	(year)	No. Of Patients	Nonunion
Wassmund	(1943)	1500	2 (0.1%)
Köle	(1956)	655	(0.6%)
Herrmann et al	(1960)	556	8 (1.4%)
Paschke and Berz	(1961)	385	5 (1.3%)
Müller	(1967)	2258	17 (0.8%)
Trauner	(1973)	530	4 (0.8%)
Claudi and Spiessl	(1975)	68	1 (1.5%)
Mathog and Boies	(1976)	577	14 (2.4%)
Krüger	(1982)	104	1 (0.96%)
Bochlogyros	(1985)	529	3 (0.6%)

From Bochlogyros PN: Non-union of fractures of the mandible. *J Maxillofac Surg* 13:189, 1985.

TABLE 14-3 Incidence of Nonunion of the Mandible After Interosseous Wiring

Study	(year)	No. of Patients	Nonunion
Edgerton and Hill	(1952)	434	18 (4.5%)
Plumpton and Crawford	(1955)	30	1 (3%)
Freihofer and Sailer	(1975)	148 (178 fr.)	1 (0.5%)
Schegg et al	(1975)	450	2 (0.5%)
Bochlogyros	(1985)	139	1 (0.7%)

From Bochlogyros PN: Non-union of fractures of the mandible. *J Maxillofac Surg* 13:189, 1985.

TABLE 14-4 Incidence of Nonunion of the Mandible After Stable Compression Plate Fixation

Study	(year)	No. of Patients	Nonunion
Schmitz et al	(1975)	220	2 (0.9%)
Scharf and Reuter	(1975)	82	1 (1.2%)
Bochlogyros	(1985)	78	3 (3.9%)

From Bochlogyros PN: Non-union of fractures of the mandible. *J Maxillofac Surg* 13:189, 1985.

Facial Widening

Ellis and Tharanon[239] have noted this complication in case reports. The use of rigid fixation in mandibular fractures can cause facial widening if the device is not correctly placed. The type of mandibular fracture that is most prone to this complication is a symphyseal fracture associated with condylar fractures and poor dentition. In symphyseal fractures, muscle pull from the tongue and suprahyoids can cause lateral flaring of the mandibular angles and lingual tipping of the buccal segments. The buccal fracture line at the symphysis remains intact, but the lingual cortex is separated. Increased flaring is seen when bilateral subcondylar fractures are also present. In this case, the buccal segments are flail segments, and precise reduction is paramount. Flaring is also caused by tightening of the maxillomandibular fixation wires. The authors have suggested a simple solution. Providing pressure in a medial direction to the gonial angles during reduction and fixation can help alleviate the chance of a facial width complication.

In a similar study, Cillo and Ellis have found that approximately 25% of patients with double unilateral mandibular fractures show gonial flaring on the affected side.[240] It was noted that patients with posterior occlusion had the most favorable outcome.

Alcoholism, Substance Abuse, Immunosuppression, and Delayed Healing

Alcohol abuse and mandibular fractures are closely interrelated in regard to cause and the increased incidence of complications.[207,241] Recovery is compromised in an alcoholic because of the suppression of T cell counts and a delayed response in cell migration, adhesion, and signal transduction.[242] Also, chronic alcohol consumption reduces protein production, which negatively affects platelet counts and aggregation, leading to improper hemostasis. Citing an extensive literature review on alcohol and its relationship to mandibular fractures, Adele et al[242] have carried out a retrospective study on 401 patients with fractures of the body of the mandible. The most notable variable related to delayed healing was the frequency of alcohol abuse. They stated that "… the frequency of patients with alcohol problems and psychosocial handicaps is reported high in most studies on jaw fractures." The patients in the delayed healing group were more frequently intoxicated by alcohol at the time of trauma and at the first visit. As a group, they were much less likely to keep appointments and were more apt to remove their own maxillomandibular fixations. Eid et al[243] have found that complications were more likely to occur in the presence of an alcoholic state, poor oral hygiene, and uncooperativeness. In an analysis of 352 patients with 589 fractures, Passeri et al[244] reported a statistically significant relationship between chronic substance abuse and postoperative complications. They reported a 6.2% rate of complications in patients with no history of substance abuse and a complication rate of 15.5% for chronic alcohol abusers, 19.2% for non-IV drug abusers, and 30% for IV drug abusers. In another study of 472 patients presenting with 699 mandibular fractures, Serena-Gomez and Passeri found an 8.5% complication rate in non–substance abusers, 17% for alcohol abusers, 14% for non-IV drug abusers, and 37.5% for IV drug abusers.[245] The correlation between substance abuse and postsurgical complications can be attributed to physiologic alterations and patient behavior.

Recognizing the delayed healing problem in alcoholics, Cannell and Boyd[246] set up a specific treatment protocol especially for the vagrant alcoholic patient. These patients were kept in the hospital for 1 month or longer. Open reduction was avoided, if possible, and additional fixation was used (metal cap splints, when possible). When open reduction was necessary, a minimal amount of periosteum was reflected and heavy antibiotic coverage was instituted. Despite all their precautions, infection of fractures or wounds occurred in 12 of 16 patients in the series. In 8 of these, rigid fixation was used after thorough cleansing of open fracture sites, removal of teeth in fracture lines, and strict attention to aftercare. The authors noted that the accumulation of hepatic fat may lead to diffusion of fatty emboli capable of impairing local blood supply. They believed that the quality of the mandibular bone was poor and that movement of the fracture sites because of noncompliance was the most likely precursor of infection. They also speculated about secondary osteoporosis, osteomalacia, and vitamin deficiency in the undernourished alcoholic. Perhaps the subject is best summed up by Heimdahl and Nordenram, who concluded that the "treatment of these patients is … not only surgical but very much social."[94]

Numerous studies have documented that substance abuse and medically compromised patients have an increased risk of developing a postoperative complications following treatment. Human immunodeficiency virus (HIV) infection causes a decline in the CD4 (T helper cell) count, resulting in impaired immunity. Martinez-Gimeno et al[247] and Schmidt et al[248] have reported a higher overall postoperative infection rate

with HIV-positive patients. However, controversy still remains as to whether the HIV-positive patient is at increased risk of developing a postoperative infection. With HIV, variables include antiretroviral medications, virulence of the HIV strain, and the fact that a relatively intact immune system may still be present. It is essential to obtain an accurate history of the patient's past opportunistic infections and CD4 count as a means of better assessing patient vulnerability for negative treatment outcomes.[201]

Nerve Disorders

The most commonly injured nerve associated with mandibular fractures is the inferior alveolar nerve and its branches—mylohyoid, dental branches, incisive branch and, especially, the mental nerve. Rarely, other branches of the mandibular nerve may be injured, such as the masseteric (condylar fractures), buccal (intraoral lacerations associated with angle or body fractures), auriculotemporal (condylar fractures), and lingual (intraoral lacerations) nerves. The prominent sign of inferior alveolar nerve deficit is numbness or other sensory changes in the lower lip and chin.

A rare but impressive nerve deficit is that associated with the marginal mandibular branch of the facial nerve. The telltale motor dysfunction of the musculature of the face or lips can result from trauma in the region of the condyle, ramus, and angle of the mandible and from lacerations affecting the facial nerve, particularly the distribution of the marginal mandibular branch. Most of the sensory and motor functions of these nerves will improve and return to normal with time because they are a consequence of compression or stretching; however, lacerations or nerve tissue loss produced by grossly displaced fracture fragments, soft tissue loss, or gunshot wounds can result in permanent deficits.

There are a few studies of sensory nerve deficit associated with mandibular fractures. Renzi et al have prospectively reviewed 97 patients with 103 facial fractures. Reportedly, presurgical trigmeninal nerve impairment was higher when fractures were associated with direct nerve involvement (100%) and dislocated fractures (84%) as opposed to nondisplaced fractures (47%). After 12 months, residual hypoesthesia was noted only with 6 mandibular fractures, which were associated with direct nerve involvement. Moore et al[82] (56 patients, 100 fractures) have reported an incidence of mental nerve paresthesia of 1.89% and facial nerve damage of 1.8%. It is not known whether the deficit was permanent. Larsen and Nielsen[95] have reported permanent sensory disturbances in the area of the mental nerve following mandibular fracture in 19 patients, corresponding to 8% of 229 patients evaluated.

Milford and Loizeaux[249] have reviewed the literature on false aneurysms and partial facial paralysis secondary to mandibular fractures. They also noted one case of facial paralysis following a fracture of the mandibular condyle, coronoid process, and symphysis. Paralysis was caused by edema in the fallopian canal, and function returned with time.

Rapids and Brock[250] have also reported the case of a 5-year-old patient with hemiparalysis of the face after a minimally displaced fracture of the condyle on the same side. Recovery was complete 2 months after surgery.

Schmidseder and Scheunemann[251] have reported on nerve injury following condylar neck fractures. They found post-traumatic neurologic complications in 8 of 237 fractures. One case involved the chorda tympani, with unilateral loss of taste in the tongue. One case involved the facial nerve, resulting in total paresis. Five cases involved the auriculotemporal nerve and, in two of the cases, Frey's syndrome developed. One case involved the buccal branch of cranial nerve VIII and occurred 7 days after a condyle was grossly medially displaced. Most of these cases improved over time.

SUMMARY

From this review, it is apparent that infection is the most common type of complication arising from the treatment of mandibular fractures. Although the incidence varies, it appears to range from 5% to 10% (average). Preoperative oral sepsis, with grossly carious and periodontally involved teeth, contributes to the problem and, unless diseased teeth are important for reduction and fixation of the fracture, they should be removed. Inadequate immobilization of the fracture segments and prolonged delay in treatment also contribute to infection.

Although it would be expected that open reduction would contribute to infection, this relationship is not evident in a review of the literature. In addition, there appears to be little difference between the infection rates of intraoral and extraoral open reduction procedures.

Rigid fixation techniques are initially seen to result in a higher complication rate, but as surgeons become more proficient with the procedures, complication rates fall. Delayed union results from infection, lack of fracture stability, noncompliance, and alcoholism. Nonunion is extremely rare.

It is evident that alcohol abuse plays a major role in the cause of mandibular fractures and results in a higher rate of complications following treatment. This higher complication rate can be attributed immunosuppression as well as noncompliance because of impaired judgement.

There are few studies on the incidence of permanent nerve damage associated with mandibular fractures to allow any definitive conclusions to be reached. More controlled prospective studies on the various methods of reduction and fixation of mandibular fractures are necessary to establish clinical protocols.

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CHAPTER 15

Traumatic Injuries to the Mandibular Condyle

David M. Yates | John R. Zuniga | Michael F. Zide

OUTLINE

Condylar Neck Region Biomechanics
Classification of Temporomandibular Joint Fractures
Strasbourg Osteosynthesis Research Group
Classification for Management of Subcondylar Fractures
Radiographic Evaluation Condylar Fractures
Mandibular Condylar Fractures
Indications for Reduction
Treatment
Reduction of the Condyle for Access
Complications of Condylar Fracture Treatment
Condylar Fractures in Children
Physical Therapy for Condylar Fractures

The literature suggests that condylar fractures account for 17.5% to 52 % of all mandibular fractures. In most condylar fractures, direct force to the distal mandible sequentially shocks the joint area proximally.[1-5]

CONDYLAR NECK REGION BIOMECHANICS

Multiple factors create condylar head displacement, including direction, magnitude, and the point of maximum force.[6] Stability of the occlusion and jaw position (open versus closed) during the traumatic insult affect condylar displacement. Little or no displacement occurs in occlusion with adequate molar support; significant condylar displacement may occur with the mouth open, exposing condylar vulnerability.[7]

Surgeons commonly overlook condylar fractures.[8] Subtle signs may mask diagnosis. On the other hand, patients may deform significantly. Patients may occlude prematurely on the ipsilateral side and open posteriorly on the contralateral side (Fig. 15-1). They will deviate toward the fracture side on opening because of disruption of lateral pterygoid forces.[9] Patients with bilateral condylar fractures characteristically occlude posteriorly and open the anterior occlusion symmetrically (Fig. 15-2). Subtle findings may include limited mouth opening, limited mandibular movements, and preauricular pain.

According to a review by Zachariades et al,[6] 72% of condylar fractures are associated with concomitant mandibular fractures, usually parasymphyseal (Fig. 15-3). Body fractures prevail with bilateral condylar fractures, except in children.

Concomitant facial fractures predominate with more proximal condylar fractures. The forces generated to yield coexistent facial fractures produce diacapitular (intracapsular) fractures (75%) and condylar base (subcondylar) fractures (25%).[10] In isolated condylar fractures, the degree of malocclusion increases as the fracture moves distally. Malocclusion was present in only 12% of intracapsular fractures, 31 % of condylar neck fractures, and 57% of subcondylar fractures.[9]

CLASSIFICATION OF TEMPOROMANDIBULAR JOINT FRACTURES

Temporomandibular joint (TMJ) classification schemes tend to be complex, difficult to use, and clinically irrelevant in treatment plan development.[11] There are many variables to consider, including intracapsular versus extracapsular fractures, condylar fracture location (high, medium, or low), age of the patient, age of the fracture, dislocation of the condyle, simple versus comminuted, type of joint capsule injury, amount of occlusal support, ramus height shortening, and degree of fracture angulation (displacement).[12,13]

Condylar displacement and ramus shortening are useful determinants. Patients with displaced condylar fractures of 10 degrees or more, together with ramus height shortening of 2 mm or more, benefit from open reduction internal fixation. This is true despite the level of condylar fracture.[10,13-17,24] The literature is conflicted concerning the indications for fracture repair based solely on condylar displacement; however, increased degrees of condylar displacement correspond to increased joint dysfunction and pain.[19,20]

Two helpful classification systems are reviewed here. The first is an anatomic classification scheme that evaluates condylar displacement, condylar fracture location (diacapitular, neck, or base), and ramus height shortening. The second is a treatment algorithm aimed at subcondylar fractures.

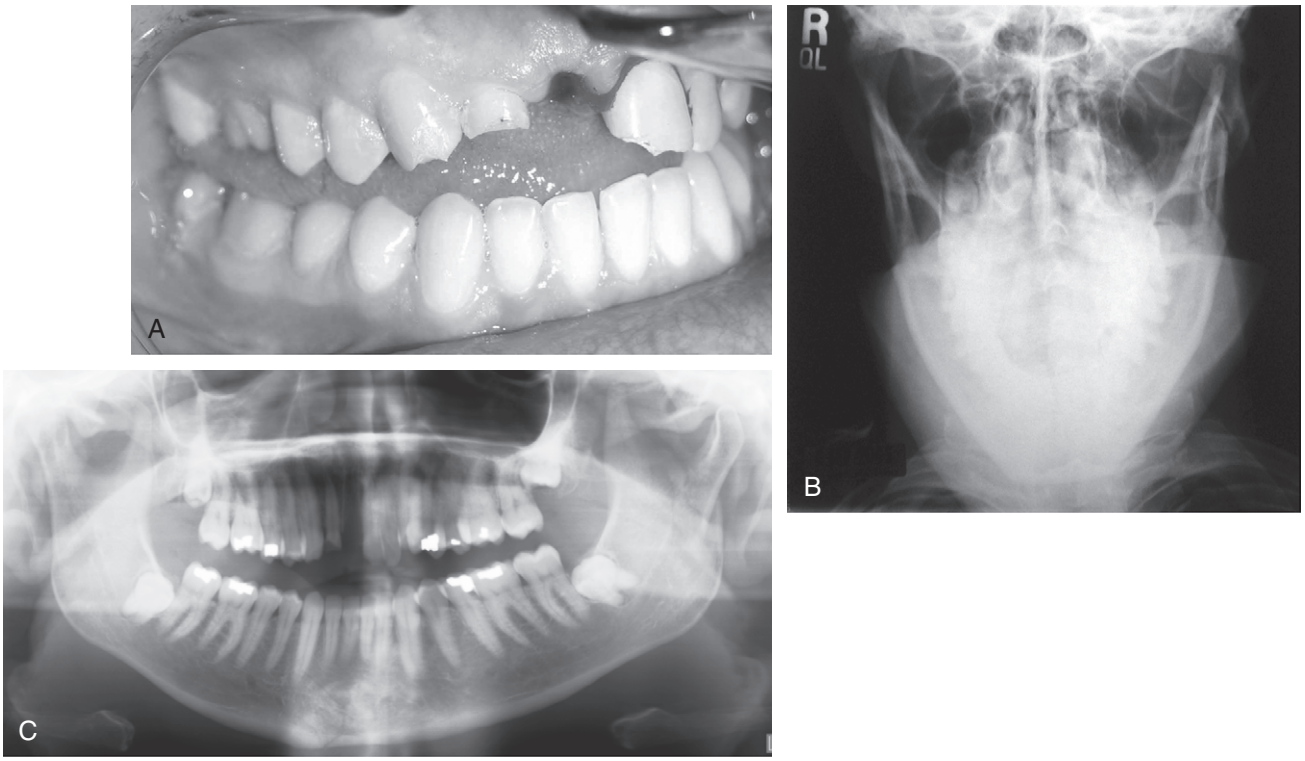


FIGURE 15-1 Classic condylar fracture occlusion—premature occlusion of dentition on ipsilateral side of fracture, posterior open bite on contralateral side of fracture. (From Fonseca RJ, Turvey TA, Marciani RD: *Oral and maxillofacial surgery*, ed 2, St. Louis, 2009, Saunders)

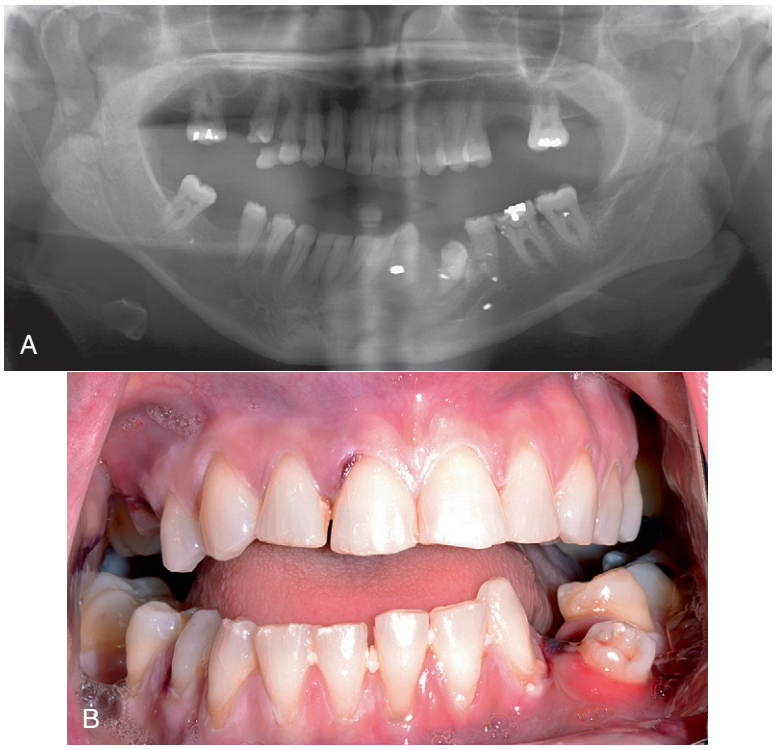


FIGURE 15-2 Bilateral condylar fracture. (From Zuniga Zide Bilateral condylar fractures.)

STRASBOURG OSTEOSYNTHESIS RESEARCH GROUP

This system was developed to simplify the categorization of condylar fractures based on anatomic and clinical significance (Fig. 15-4).[10,18,21,22] It may be stated as follows:

- *Diacapitular fracture* (through the head of the condyle). The fracture line starts in the condylar head and may extend outside of the capsule.
- *Fracture of the condylar neck.* More than half of the fracture is superior to an imaginary line extending from

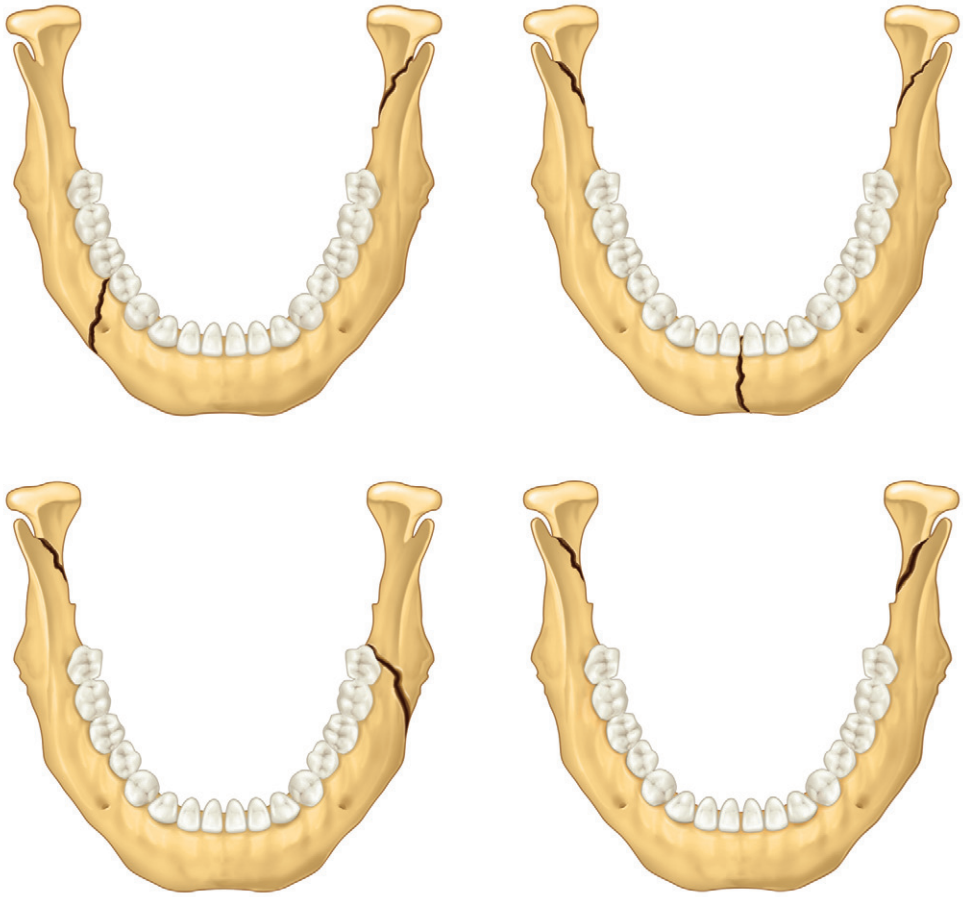


FIGURE 15-3 Condylar fracture patterns.

the most inferior portion of the sigmoid notch perpendicular to the tangent of the ramus (line A).

- *Fracture of the condylar base.* More than half of the fracture is inferior to line A.
- *Displacement.* Minimal displacement is defined as displacement less than 10 degrees or ramus height shortening 2 mm or less. Moderate displacement is defined as displacement of 10 to 45 degrees. Severe displacement is defined as displacement of 45 degrees or more.

Displacement refers to fracture line status. Dislocation refers to the luxation grade of the condylar head.[21]

CLASSIFICATION FOR MANAGEMENT OF SUBCONDYLAR FRACTURES

This system uses two proven indications, ramus height shortening and condylar displacement (coronal or sagittal plane), for open reduction and internal fixation (ORIF) and attempts to provide corresponding treatment guidelines.[14,23,24]

- *Class 1 fractures.* Minimally displaced leads to closed reduction; ramus height shortening less than 2 mm; degree of fracture displacement less than 10 degrees.
- *Class 2 fractures.* Moderately displaced leads to ORIF; ramus height shortening 2 to 15 mm; degree of fracture displacement 10 to 45 degrees.
- *Class 3 fractures.* Severely displaced leads to ORIF; ramus height shortening more than 15 mm; degree of fracture displacement more than 45 degrees.

RADIOGRAPHIC EVALUATION OF CONDYLAR FRACTURES

Various views may be used to evaluate these fracture.[18,23,25] These include the standardized Towne's radiograph, anterior posterior dimension (coronal position), Panorex, and medial lateral dimension (sagittal position).

For the measurement of condylar process displacement,[23,25] coronal displacement is evaluated with Towne's radiograph (Fig 15-5*A*) and sagittal displacement with a panoramic radiograph.

To evaluate the loss of ramus height,[18,23,25] a panoramic radiograph is used. The measurement technique is as follows (see Fig. 15-5*B*):

- Line drawn between gonial angles across Panorex
- Perpendicular lines to most superior aspect of condylar heads
- The difference between the nonfractured and fractured side equals the change in ramus height.

MANDIBULAR CONDYLAR FRACTURES

INDICATIONS FOR REDUCTION

There has been much disagreement about whether fractures should be treated open or closed. Social inequities, reimbursement quandaries, and surgical schedules guide choices when objective beneficial conclusions are hazy.

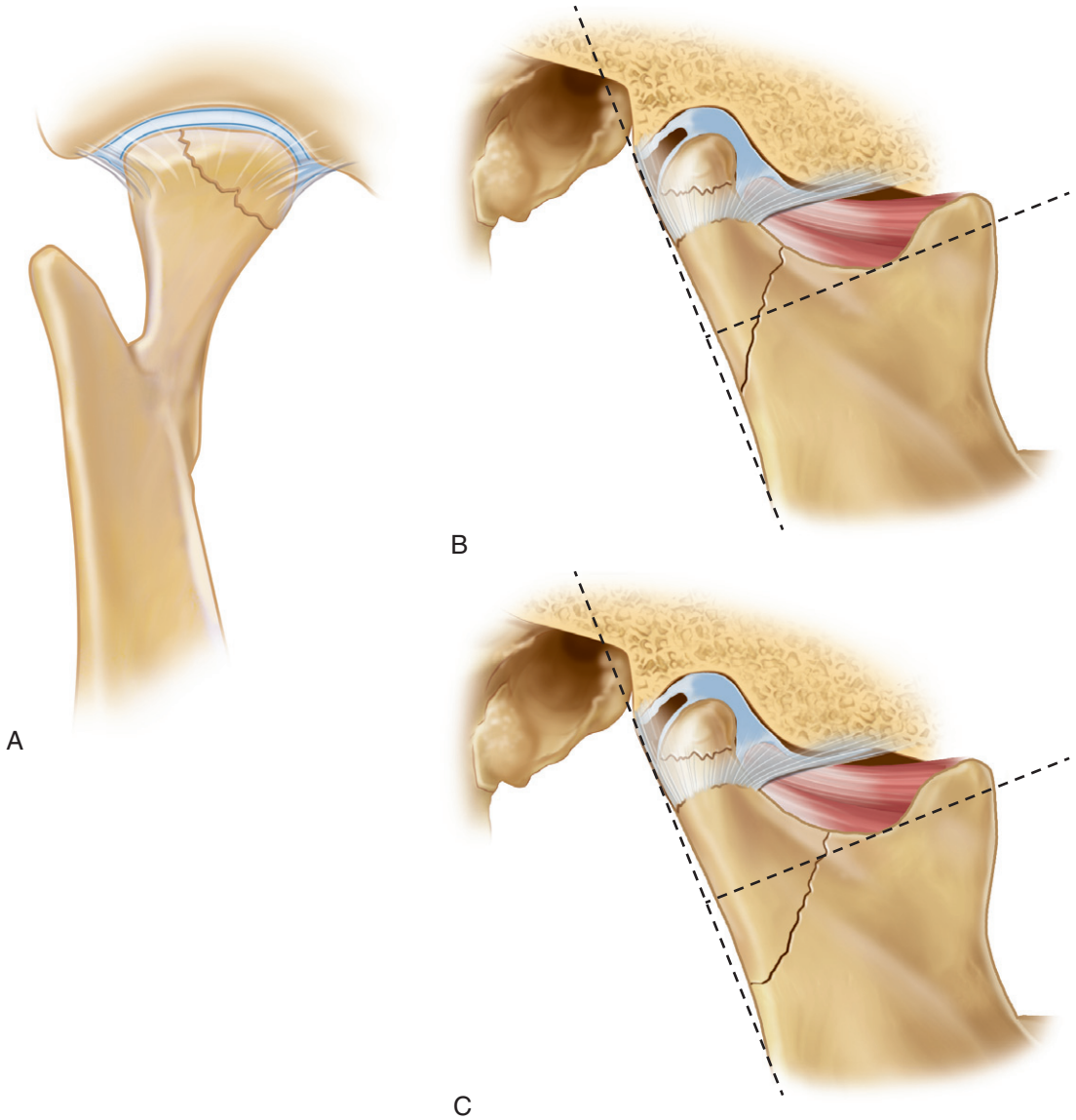


FIGURE 15-4 **A,** Diacapitular fracture. **B,** Condylar neck fracture. **C,** Condylar base fracture.

Opening a condyle fracture is no simple task for the inexperienced or unaided; hence, a surgeon will acquiesce to an acceptable result, acquired with minimal surgical intervention. Reflecting their history and experience, there are centers where all condylar fractures are treated closed and a few where the vast majority are opened.[26] Currently, without universal standards and indications, fractures are mostly opened on a case by case basis.

There are certain situations that are almost always perceived as absolute indications for ORIF of condylar fractures. Conversely, there are also clear indications for treating some condylar fractures with closed reduction. These are listed in Box 15-1. The quandary, then, is how to treat the remaining condylar fractures that fall in between the absolute indications for ORIF and the clear indications for closed reduction (CR).

Recent prospective randomized studies have compared CR with ORIF. Both CR and ORIF have been shown to be acceptable. Conclusive evidence, however, reveals that patients with ramus height shortening of 2 mm or more and condylar displacement of more than 45 degrees (severely displaced) benefit from ORIF regardless of fracture level or type of fixation performed.[27,28] Patients in the open treatment group had less pain, discomfort, malocclusions, and greater range of motion in all parameters.[14,18,26] The data are inconclusive concerning indications for fracture repair based solely on moderate condylar displacement (10 to 45 degrees); however, the degree of condylar displacement correlates directly with condylar dysfunction and long-term pain.[19]

Ellis, in 2009,[29] retrospectively reviewed 332 patient cases with unilateral extracapsular condylar fractures to determine when open treatment of condylar process fractures would be meritorious. This study did not isolate fracture type or displacement of the condylar head. It was concluded that only a patient who develops a malocclusion after release from maxillomandibular fixation

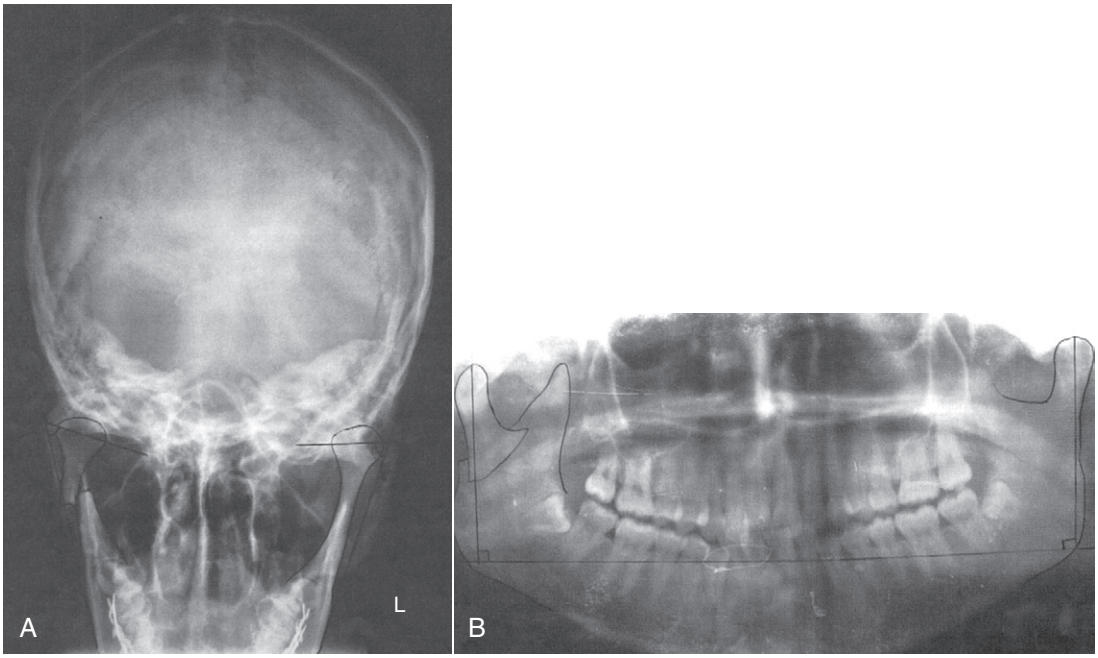


FIGURE 15-5 **A,** Condylar displacement. **B,** Loss of ramus height. (From Bhagol A, Singh V, Kumar I, Verma A: Prospective evaluation of a new classification system for the management of mandibular subcondylar fractures. *J Oral Maxillofac Surg* 69:1159–1165, 2011.)

BOX 15-1 Indications for Open Reduction

ABSOLUTE INDICATIONS
- Condylar displacement into middle cranial fossa
- Invasion by foreign body
- Lateral extracapsular displacement of condyle
- Malocclusion not amenable to closed reduction (e.g., functional reduction of ramus height)

STRONG EVIDENCE FOR OPEN REDUCTION
- Bilateral condylar fractures
- Gross condylar displacement > 45 degrees (severely displaced)
- Anatomic reduction of ramus height ≥ 2 mm
- Condylar fractures with an unstable base (associated midface fractures)
- Unstable occlusion (e.g., periodontal disease, less than three teeth per quadrant)
- Condylar fractures for which adequate physiotherapy is impossible

MIXED EVIDENCE FOR OPEN REDUCTION
- Moderate condylar displacement, 10 to 45 degrees

WHEN TO TREAT WITH CLOSED REDUCTION
- Nondisplaced or incomplete fractures
- Isolated intracapsular fractures
- Condylar fractures in children (except for absolute indications)
- Reproducible occlusion without dropback or with dropback that returns to midline on release of posterior force
- Medical illness or injury that inhibits ability to receive extended general anesthesia

Material adapted from references 14, 18, 19, 29, 30, 31, and 32.

(MMF) need be considered for open reduction. In this study, only 1 of 332 patients developed a postoperative malocclusion.

A recent prospective, double-center study by Kokemueller et al[26] has compared endoscope-assisted transoral reduction and internal fixation with CR. Inclusion criteria included unilateral condyle fractures exhibiting at least one of the following characteristics: more than 30 degrees of displacement, severe functional impairment (open bite or malocclusion), severe pain on movement, or vertical ramus shortening; high condylar fractures were excluded. Results revealed that the ORIF group was more symptom-free, with less malocclusion. The duration of MMF was shorter in the ORIF group compared with the CR group.

Regardless of whether a surgical or nonsurgical treatment plan is pursued, postoperative rehabilitation is an essential component to orthopedic success. Restoration of maximal incisal opening (MIO) of at least 40 mm, full excursive movements, and proper occlusion are laudable goals, achievable only with persistent rehabilitation and monitoring.

TREATMENT

Surgical Versus Nonsurgical Treatment

The surgical goals enumerated decades ago by Walker include the following five features[20,33]:

1. Pain-free mouth opening, with an interincisal distance beyond 40 mm
2. Good movement of the jaw in all excursive movements
3. Preinjury occlusion of teeth
4. Stable TMJs
5. Good facial and jaw symmetry

ORIF and CR patients benefit from early mobilization and extensive rehabilitation, generally over a period of 6 to 12 weeks.[34] Immediately after the traumatic injury, tissues are not yet tethered down by scar formation. Early mobilization of the joint after injury reduces permanent joint hypomobility.[35,36] Rehabilitation with a target maximal incisal opening reaching more than 40 mm before 12 weeks is fundamental and ideal before 6 weeks. Once an MIO of more than 40 mm is reached, it should be maintained during the next few months while the soft and bony tissues are maturing.

In clinical practice, joint rehabilitation consists of scheduled patient exercises to increase opening and range of motion. Early on, opening exercises alone are probably sufficient. Patients should be given arbitrary achievable goals. For example, ibuprofen or aspirin, heat and massage for 1 minute, and then opening three times daily before meals, as wide as possible, 30 times. Measurements encourage progress.

Active (the patient does the work) and passive (bite sticks, external forces) all have their place. Usually, we recommend active physiotherapy during the first 6 weeks, especially if other fractures are present, and only passive forms when obstacles are encountered (Box 15-2).

BOX 15-2 Rehabilitation in Patients Treated by Closed or Open Reduction

OCCLUSAL GUIDANCE*
- Place arch bars
- Class 2 elastics on ipsilateral side of fracture
  - Have patient discontinue guiding elastics 24 hours prior to clinic appointment. If occlusion stable and opens symmetrically, there is no need to continue with elastic guidance.
  - Remove arch bars during 6- to 12-wk period postoperatively after rehabilitation goals are achieved.

FUNCTIONAL EXERCISE: REHABILITATION TARGETS
- Maximal incisal opening > 40 mm
- Lateral excursive movements > 10 mm
- Protrusive excursive movements > 12 mm
- Full use of TMJ throughout the day (diet as indicated by pain and coexisting injuries)
- Close supervision by surgeon to evaluate occlusion, adjust regimen as necessary

*If malocclusion is present.
Adapted from Palmieri C, Ellis E, Throckmorton G: Mandibular motion after closed and open treatment of unilateral mandibular condylar process fractures. *J Oral Maxillofac Surg* 57:764–775, 1999.

Surgical Approaches to the Condylar and Subcondylar Regions

All open approaches have three common aspects to their success:

1. The ramus must be distracted.
2. The proximal condyle must be controlled and manipulated.
3. The fracture must be anatomically reduced and plated with more than one screw on the proximal segment.

Submandibular Approach.

This is also known as the Risdon approach.[12,37-42]

Indications. These include axial anchor screw fixation.

Advantages. These include the ability to distract the mandibular ramus and direct access of the gonial angle.

Disadvantages. These include limited surgical site exposure (the incision is distant from the fracture), difficult to reduce medially displaced condyles, and plate and screw fixation restricted without a transfacial trocar.

Pertinent Anatomy

Marginal Mandibular Branch of the Facial Nerve. Posterior to the facial artery, the marginal branch passes inferior to the border of the mandible in 19% to 56% of specimens studied (Fig. 15-6).[37,38] The maximum inferior extent of the mandibular branch is 1.2 cm below the mandible. Anterior to the facial artery, the marginal branch passes inferior to the border of the mandible in 0% to 6% of specimens studied. It passes immediately deep to the superficial layer of the deep cervical fascia, which is immediately deep to the platysma, and superficial to the facial vessels.

Facial Artery and Vein. This passes through or along the superficial surface of the submandibular gland and approximates the inferior border of the mandible just anterior to the pterygomasseteric sling (Fig. 15-7). The artery then rounds the inferior border of the mandible and becomes superficial to the mandible. Generally, the anterior facial vein runs posterior to the facial artery above the inferior border of the mandible. The facial vein is just deep to the platysma.

Surgical Technique

Exposure. The corners of the mouth and lower lip are exposed anteriorly and the entire ear, posteriorly.

Incision. Using arch bars and elastics or wires prior to facial incision, place the patient in MMF. These are 1.5 to 2 cm below the inferior border of the mandible in or parallel to a skin crease. In patients with ramus height shortening, place the incision 1.5 to 2 cm below where the anticipated reduced mandible would be. The initial incision is placed to the depth of the platysma, with extensive undermining in all directions.

Dissection. See Figure 15-8.

- *Through the platysma.* Undermine and sharply dissect, being careful to stay superficial to the superficial layer of the deep cervical fascia.
- *From the platysma to pterygomasseteric sling.* Make a small incision through the superficial layer of the deep cervical fascia at the level of the skin incision (1.5 to 2 cm inferior to the mandible). The facial artery and vein may be retracted anteriorly or divided and ligated if necessary. Continue the dissection superiorly until the pterygomasseteric sling is encountered.
- *Division of the pterygomasseteric sling.* The use of a nerve stimulator is necessary through this portion of the dissection, with the absence of muscle relaxants, to identify the branches of the facial nerve. Sharply incise the pterygomasseteric sling with a scalpel along the inferior border of the mandible. Use a periosteal elevator to expose the ramus up to the level of the TMJ capsule and coronoid process. A sigmoid notch retractor is helpful in fully exposing the ramus.

Osteosynthesis: Axial Anchor Screw

Visibility and Reduction. Distract the distal segment using ligature wire at the gonial angle (see Fig. 15-9). Use a

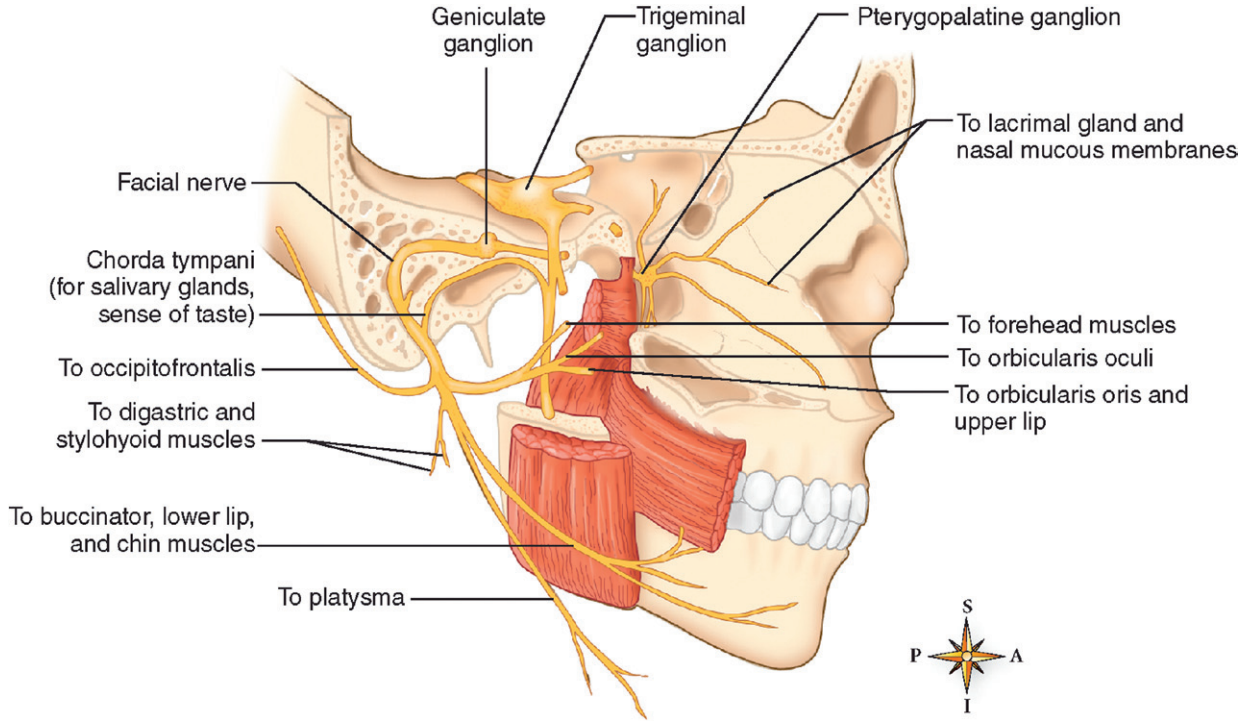


FIGURE 15-6 Facial nerve. (From Patton K, Thibodeau G: *Anatomy and physiology*, ed 7, St. Louis, 2012, Mosby.)

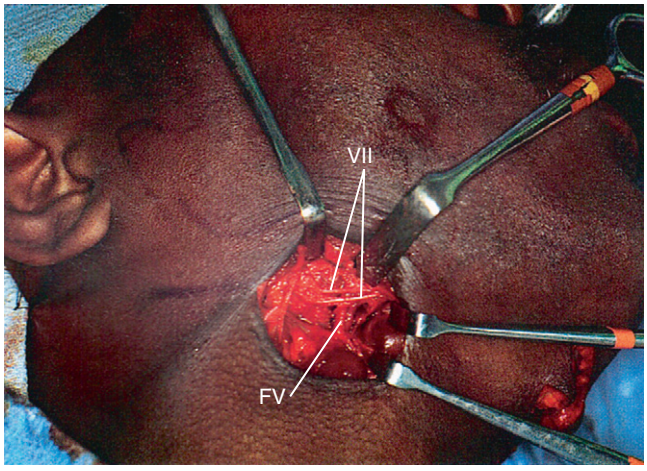


FIGURE 15-7 Exposure of the facial artery vein and cranial nerve VII. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, p 169.)

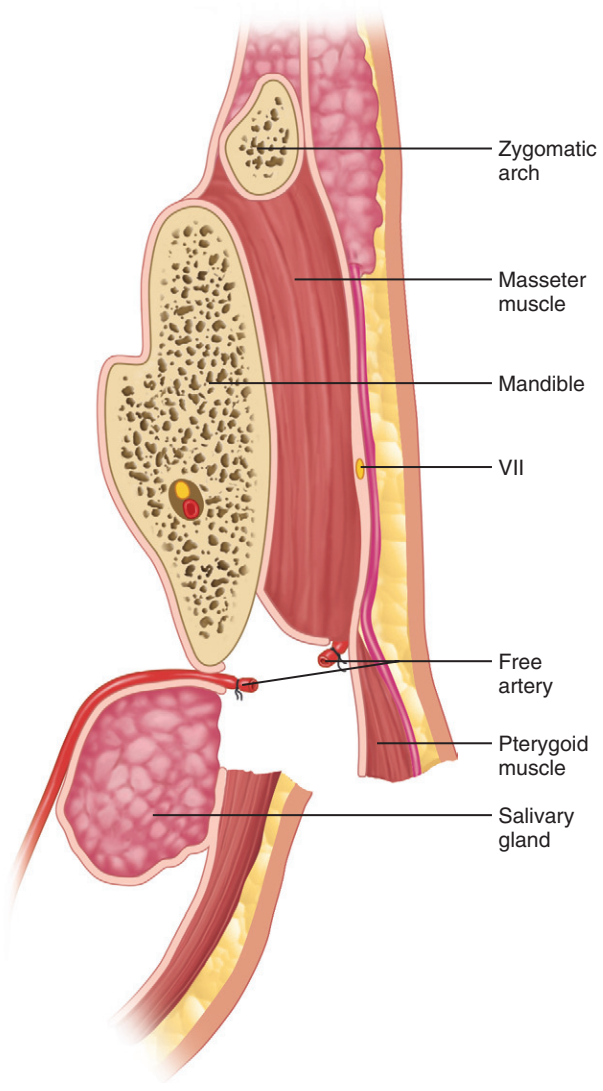


FIGURE 15-8 Anatomic landmarks of submandibular dissection.

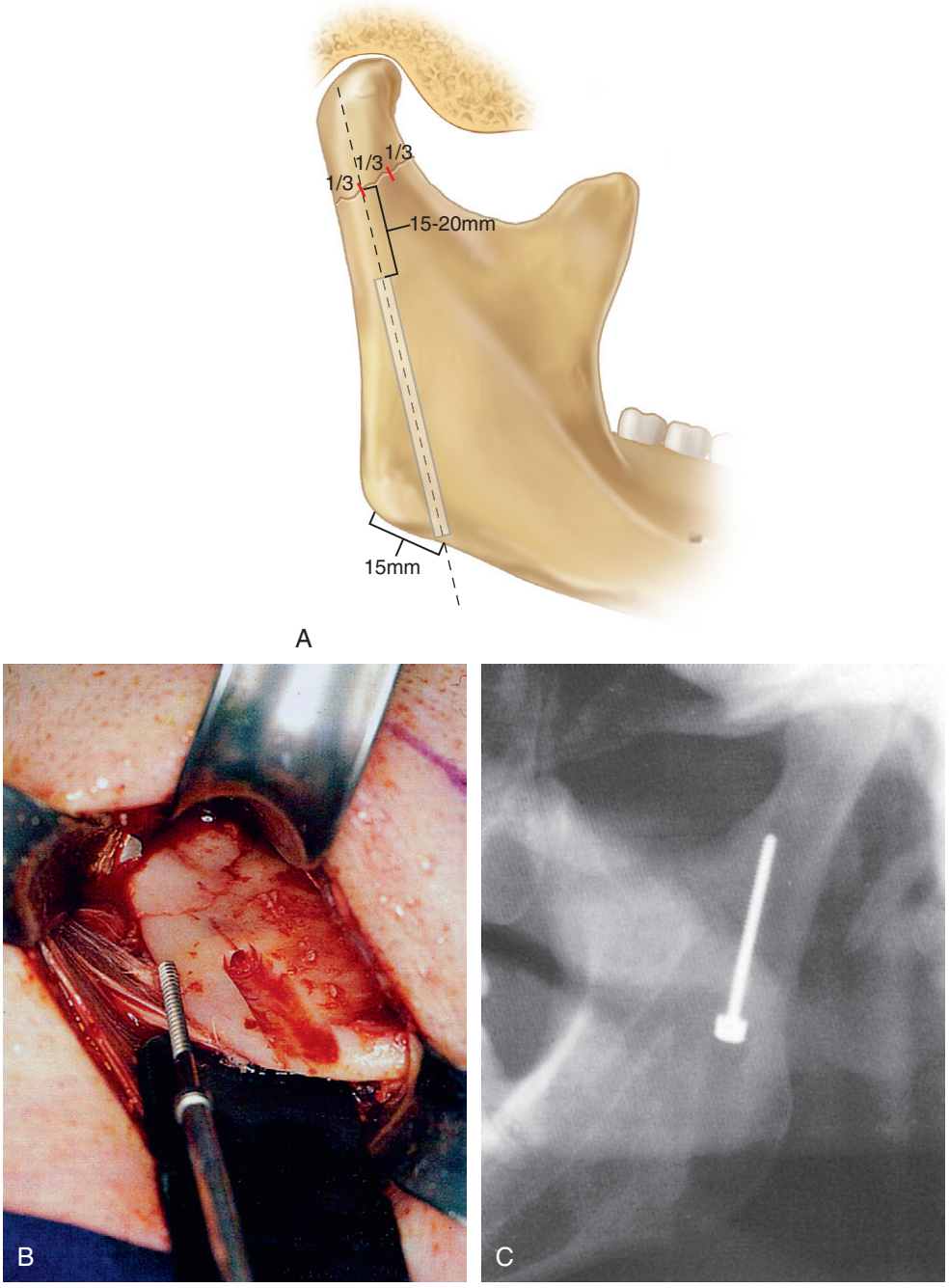


FIGURE 15-9 Direct fixation using lag screw technique. (**B** from Krenkel C: *Biomechanics and osteosynthesis of condylar neck fractures of the mandible*. Carol Stream, Ill, 1994, Quintessence.)

sigmoid notch retractor and condylar neck retractor to reflect soft tissues. Stabilize and reduce the proximal segment using a curved hemostat.

Direct Fixation. Place a groove in the lateral cortex approximately 1 cm anterior to the posterior border and 1.5 to 2 cm inferior to the fracture line (see Fig. 15-9). A centering instrument is used to place the screw hole; a 2-mm drill is used to drill the pilot hole to the fracture line. The drill guide is placed and a 1.5-mm drill is used to penetrate beyond the fracture line into the proximal segment an additional 1 to 2 cm. Measure length and place and tighten titanium screw with biconcave washer.

Indirect Fixation. This alternative method is indicated when the proximal condylar fragment is difficult to reduce (Fig. 15-10). First, place the positioning screw into the proximal segment. Then reduce the proximal segment using a biomechanically advantageous screw. Place a groove through the lateral cortex to the fracture line. Lock the screw into place using a two-hole miniplate locked against the proximal screw shaft.

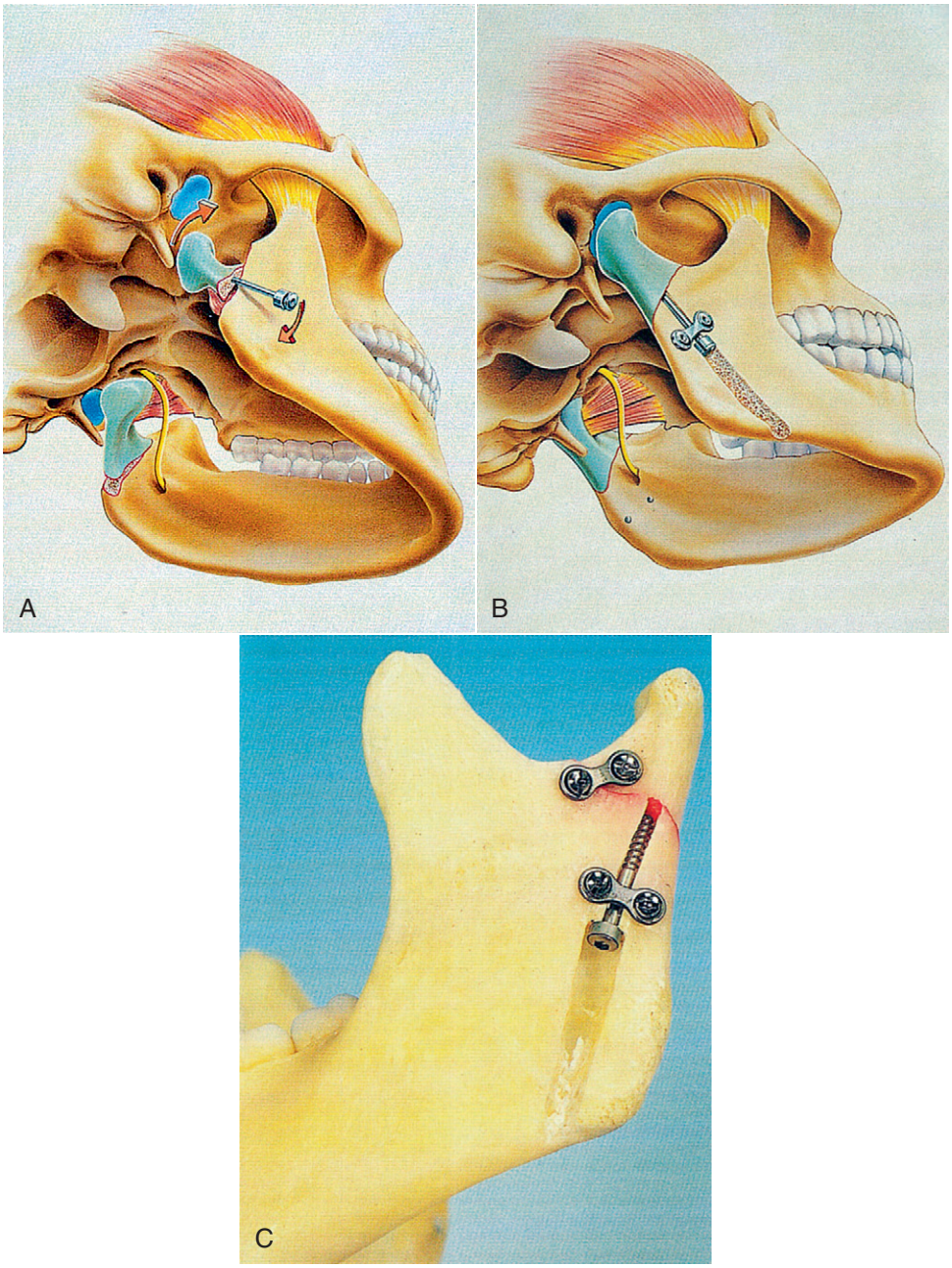


FIGURE 15-10 Indirect fixation using lag screw technique. (From Krenkel C: *Biomechanics and osteosynthesis of condylar neck fractures of the mandible*. Carol Stream, Ill, 1994, Quintessence, pp 104–105.)

Closure

- *Pterygomasseteric sling.* Suture together the masseter and medial pterygoid using interrupted resorbable sutures.
- *Platysma.* Suture using resorbable sutures in a running fashion.
- *Subcutaneous tissue.* Use resorbable sutures.
- *Skin.* Use nonresorbable suture material.

Retromandibular Approach

Indications. This is used for any fracture that is large enough to be reduced and stabilized by ORIF using plates and screws.[37,38]

Advantages. These include the following: there is a short distance between the incision and the fracture site; there is best access to the fracture site; there is no need for a transfacial trocar; the facial scar is less noticeable than with a submandibular incision; it is effective in patients with edema; and there is access for an osteotomy if required to reach the condyle.

Disadvantages. The facial scar is more noticeable than with a preauricular incision.

Pertinent Anatomy

Facial Nerve. See the Preauricular section for further discussion of the facial nerve. However, between the superior and inferior divisions of the facial nerve, the posterior ramus of the mandible can be safely accessed (Fig. 15-11).

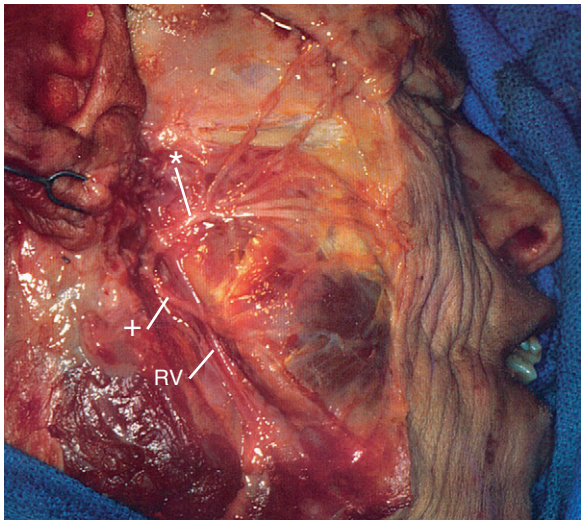


FIGURE 15-11 Superior and inferior divisions of facial nerve. Facial nerves—superior division (*) and inferior division (+). *RV,* Retromandibular vein. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, p 171.)

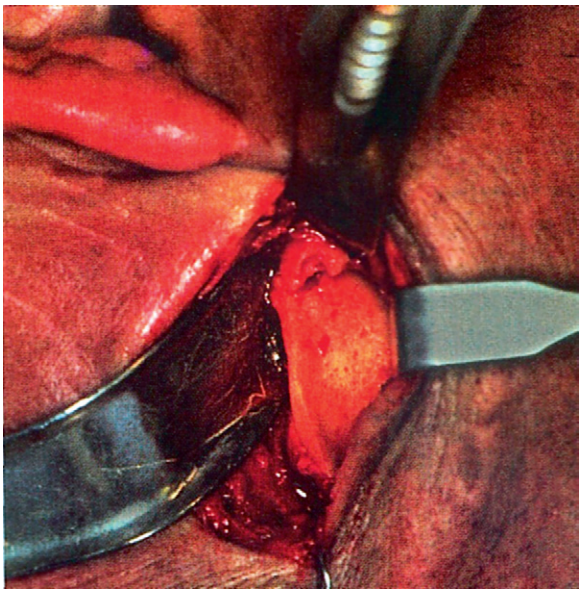


FIGURE 15-12 Exposure of ramus. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, p 179.)

Retromandibular Vein. This is formed deep to the neck of the mandible from the superficial temporal vein and maxillary vein (see Fig. 15-11), superficial to the external carotid and crossed by the facial nerve.

Surgical Technique

Exposure. Anteriorly, this should be at the corner of the mouth and lower lip and posteriorly, at the entire ear.

Incision. Place the incision 0.5 cm below the earlobe and carry inferiorly for 3 to 3.5 cm. Place it just posterior to the posterior border of the mandible. Incise to the depth of the scant platysma overlying the superficial musculoaponeurotic system (SMAS) and the parotid capsule. Undermine extensively in all directions for maximal exposure.

Dissection

- *Dissection to the pterygomasseteric sling.* Sharply incise the platysma, underlying SMAS, and parotid capsule (Figs. 15-12 and 15-13). Continue the dissection bluntly in the substance of the parotid parallel to the expected direction of the facial nerve. Carry the dissection to the periosteum of the posterior border of the mandible, avoiding injury to the branches of the facial nerve and retromandibular vein. The use of a nerve stimulator is necessary through this portion of the dissection, with the absence of muscle relaxants, to identify the branches of the facial nerve.
- *Division of the pterygomasseteric sling.* To divide the pterygomasseteric sling, sharply incise, starting as superiorly and posteriorly on the ramus as possible. Carry the incision around the gonial angle as far anteriorly and inferiorly as possible. Use a periosteal elevator to expose the ramus and condylar area. Use a sigmoid notch retractor to maintain exposure of the ramus. It is helpful to distract the mandible inferiorly to expose the condylar process fracture fully.

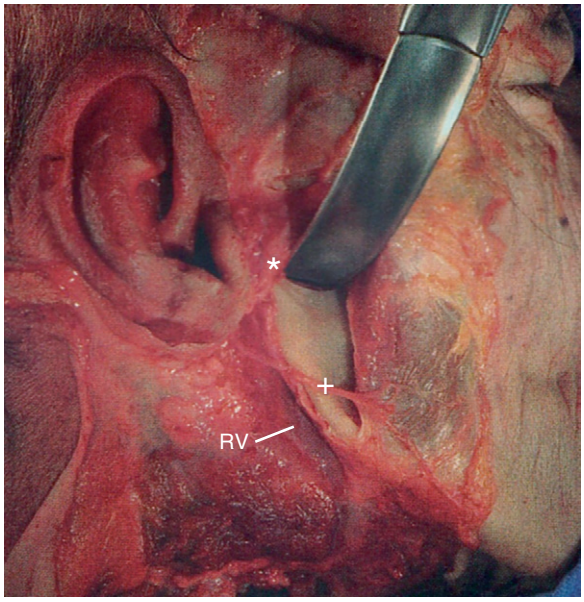


FIGURE 15-13 Position of facial nerve. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, p 179.)

Osteosynthesis

- Distract the proximal fragment inferiorly using an Allis clamp (Fig. 15-14). Place the hole superior to fracture line in the second screw hole from the top of the plate.[37]
- Place 24-gauge wire through the inferior hole of the plate. Distract the condylar head inferiorly to enable the placement of a drill hole in the superior hole of the plate, and apply a screw.
- Drill and place the screws in the inferior holes in the distal segment.

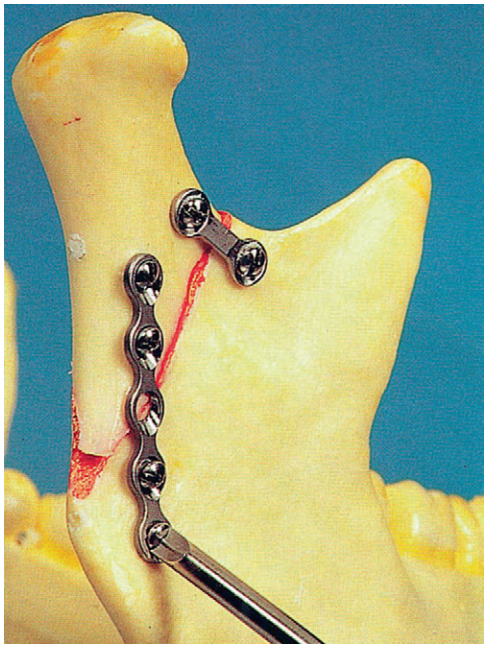


FIGURE 15-14 Double miniplate technique. (From Krenkel C: *Biomechanics and osteosynthesis of condylar neck fractures of the mandible*. Carol Stream, Ill, 1994, Quintessence, p 57.)

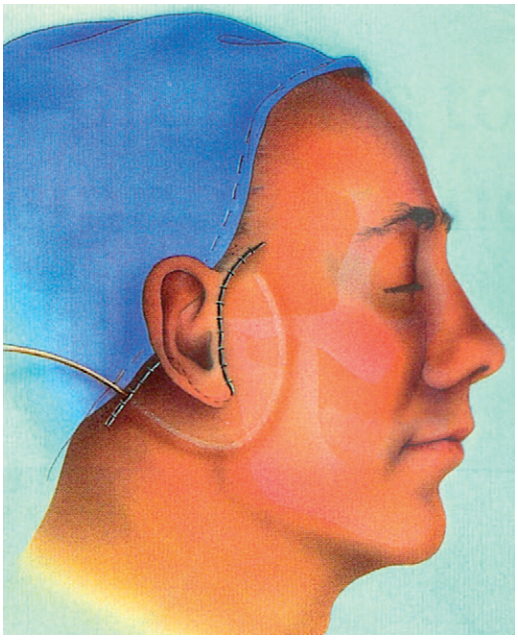


FIGURE 15-15 Rhytidectomy incision. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, p 189.)

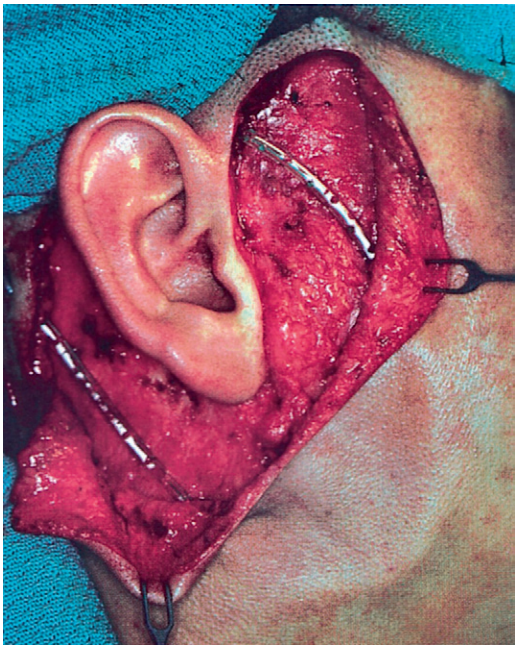


FIGURE 15-16 Drain placement. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, 189)

Closure. For the pterygomasseteric sling, suture together the masseter and medial pterygoid using interrupted resorbable sutures. For the parotid capsule, platysma, and SMAS layer, use slowly resorbable horizontal mattress sutures in a running fashion. This step is vital for avoiding a salivary fistula. For subcutaneous tissue, use resorbable sutures and, for the skin, use nonresorbable sutures.

Rhytidectomy Approach. This uses the same access as that of the retromandibular approach, with better cosmesis.[30,38] This approach must be drained with closed suction drainage postoperatively (Figs. 15-15 and 15-16).

Transmasseteric-Anteroparotid Approach.

Indications. This is done to provide access to high and low subcondylar and ramus fractures.[43-46]

Advantages. These include quick and direct access to fracture sites for direct plating and screw fixation, with excellent exposure and the ability to distract mandibular ramus because of access to the gonial angle, and has the best access of all the approaches.

Disadvantages. There is a visible scar that is more noticeable than with the other approaches and there is potential damage to the facial nerve.

Pertinent Anatomy

Marginal Mandibular and Buccal Branches of the Facial Nerve. Posterior to the facial artery, the marginal mandibular branch is above the inferior border of the mandible in 44% to 81% of specimens studied.[43-45] There is a single buccal branch of the facial nerve in 85% of specimens studied, which is inferior to the parotid duct. In 15% of cases, there are two branches, one above and one below the parotid duct. Both are immediately deep to the SMAS layer of the parotidomasseteric fascia.

Layers of the Parotidomasseteric Region. These are the skin, subcutaneous fat, parotidomasseteric fascia–SMAS layer, masseter superficial and deep bellies, and periosteum of the mandible.

Surgical Technique:

Exposure. Anteriorly, this is at the border of the mouth and lower lip and lateral nose and cheek; posteriorly, the entire ear. The patient is in wire MMF for the entire procedure.

Incision. Draw a line from the bottom of the earlobe at the posterior border of the ramus to the gonial angle.



FIGURE 15-17 Transmasseteric incision. (From Zuniga Unilateral condylar fracture: Transmasseteric approach)

Divide the distance of this line into thirds. Draw a line the same distance as above, anteriorly along the inferior border of the mandible from the gonial angle forward. Divide this distance into thirds.

Make the incision at the line connecting the intersections of the last third on the posterior ramus line and the first third on the inferior border line, the intersection closest to the gonial angle in each direction (Fig. 15-17).

The initial incision is to the depth of the parotidomasseteric fascia (SMAS). Then, there is extensive subcutaneous undermining in all directions to allow for maximal exposure.

Dissection to the Pterygomasseteric Sling. Undermine the parotidomasseteric fascia (SMAS) in the horizontal direction (parallel to the facial nerves). The use of a nerve stimulator is necessary through this portion of the dissection, with the absence of muscle relaxants, to identify the branches of the facial nerve.

If no facial nerve branches are identified by testing or visualization, then sharply incise and expose the pterygomasseteric sling. If the facial nerve is identified by testing or visualization and unavoidable in the horizontal approach, bluntly dissect through the parotidomasseteric fascia (SMAS) in the vertical direction to displace the facial nerve(s) away in the superior or inferior direction.

Division of the Masseteric Sling. Bluntly dissect (using hemostats or scissors) in a vertical plane, with access through the masseter muscle until the mandible is identified (Fig. 15-18). The direction of dissection is parallel to the masseter muscle fibers. Avoid sharp dissection to reduce bleeding and injury to the facial nerve. Once the mandible is identified, place two or three Obwegeser (towed-in) retractors on the mandible and retract to enhance the exposure of the lateral mandible at the ramus–midbody section.

Exposure of the Mandible and Fracture. Use a periosteal elevator to strip the masseter muscle and periosteum to expose the angle of the mandible first and then the lateral mandible along the ascending ramus. Identify the sigmoid notch and place a retractor (e.g., channel retractor) in the sigmoid notch for orientation. Finally, expose the posterior ramus of the mandible superiorly to the fracture site.

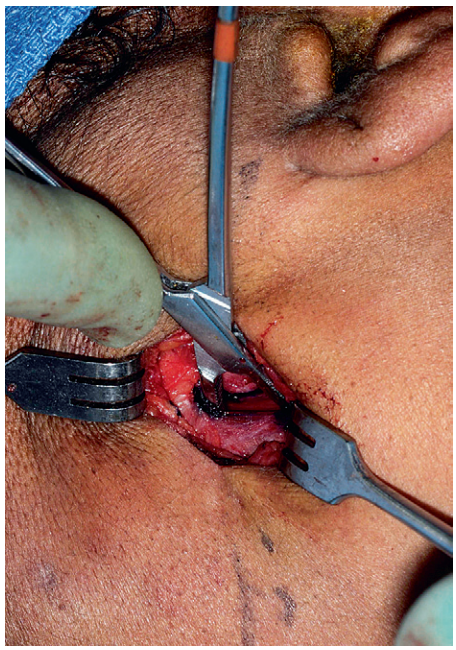


FIGURE 15-18 Division of the masseter. (From Zuniga Unilateral Condylar Fracture: Transmasseteric Approach)

Identify the proximal fracture or margin by visualization, palpation, or distraction of the mandible inferiorly using a Kocher clamp. Directly engaging the angle of the mandible through the access provided earlier can be helpful. Strip the lateral and inferior portions of the proximal segment of the fracture and place an Obwegeser retractor on the proximal lateral surface above the fracture junction.

Osteosynthesis. Engage the angle of the mandible through the access provided earlier using a Kocher clamp. One individual should displace the mandible inferiorly while the operating surgeon positions the proximal segment to the correct position.

Once the proximal segment is positioned properly, release the inferiorly displaced mandible. Repeat until the fracture is reduced. Generally, the reduction is self-retaining, without the need for lateral or superior compression or manipulation during the stabilization and osteosynthesis phase so the Kocher clamp can be removed to assist in further visualization of the osteosynthesis.

Place the plates (variable, straight, H, U, X, modified Y-shaped) (Fig. 15-19) and screws with direct visualization. This rarely requires a trocar except in a very high subcondylar fracture or limited access to the proximal segment's most superior hole(s).

Closure. No closure of the periosteum or masseter muscle sling is necessary. Suture the parotidomasseteric fascia (SMAS) using slowly resorbable sutures in running fashion (necessary to avoid salivary fistula). Suture the subcutaneous tissue using resorbable sutures. Suture the skin using nonresorbable sutures.

Preauricular Approach.

Indications. These include wire fixation of a high, anteromedially displaced proximal fragment.[37,38]

Advantages. This provides access to the superiormost portion of the joint.



FIGURE 15-19 Fixation with Y plate. (From Zuniga Unilateral Condylar Fracture: Transmasseteric Approach.)

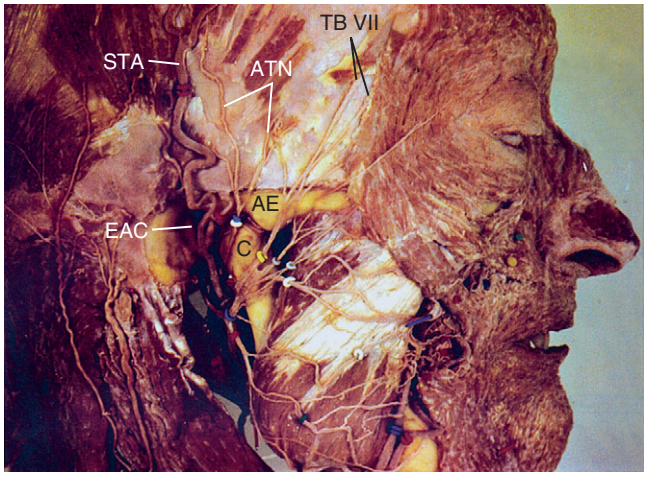


FIGURE 15-20 Anatomic landmarks. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins.)

Disadvantages. This is not indicated for placement of the plate and screw fixation. There is no access to the angle of the mandible to distract the ramus inferiorly; towel clip placed transcutaneously may be a reasonable substitute. Limited ramus exposure makes osteosynthesis plate placement extremely difficult. This results in more proximal segment stripping, leading to an increased risk of necrosis.

Pertinent Anatomy. See Figure 15-20.

Ginglymoarthrodial Joint. This allows for rotation and translation. The capsular ligament surrounds the condylar head, superiorly attaches to the temporal bone, and inferiorly attaches to the condylar neck. For the collateral discal ligaments, attach the articular disc to the condylar head.

Blood Vessels. These include the superficial temporal artery, lateral and posterior to the condylar head and neck. It runs within the temporoparietal fascia accompanied by the auriculotemporal nerve. As it crosses over the zygomatic arch, a temporal branch is given off, which frequently causes bleeding when using the preauricular approach. The retromandibular vein is formed from the maxillary and superficial temporal veins. It travels just posterior to the mandibular ramus and is superficial to the external carotid and deep to the facial nerve.

Nerves. The main trunk of the facial nerve is 2 cm deep to the skin at the middle of the anterior border of the mastoid process. The temporal branches are the most susceptible to injury; 8 to 35 mm (average, 20 mm) is the range of distances from the anteriormost portion of the bony external auditory canal (EAC) to where the upper trunk crosses zygomatic arch, under the surface of the temporoparietal (superficial layer of temporalis fascia) fascia. Avoid by incising within 8 mm of the EAC and dissecting beyond the temporoparietal fascia and periosteum of the zygomatic arch.

Trigeminal Nerve: Auriculotemporal Nerve. This courses laterally behind the condylar neck and supplies skin in the temporal and preauricular regions, EAC, and tympanic membrane. It is almost invariably injured when using the preauricular approach. Avoid by dissecting close to the cartilaginous portion of the external auditory meatus.

Layers of the Temporal Region. These include the skin, subcutaneous fat, temporoparietal fascia (i.e., superficial temporal fascia or suprazygomatic SMAS). Extension of the galea is continuous with the SMAS. The superficial temporal vessels run along the superficial surface. The facial nerve branches run along the deep surface. The superficial and deep layers of the temporalis fascia invest the fascia of the temporalis muscle. The superficial layer attaches to the lateral aspect of the zygomatic arch. The deep layer attaches to the medial aspect of zygomatic arch. The superficial temporal fat pad is between the superficial and deep layers of the temporalis fascia and usually contains a large vein.

Surgical Technique

Exposure. Expose the entire ear and lateral canthus of the eye.

Incision. This is made in the skin fold along the entire length of the ear. It extends superiorly to the top of the helix and may include the anterior extension. Incise to the depth of the superficial layer of the temporalis fascia.

Dissection to the Joint Capsule. Dissect along the anterior portion of the external auditory cartilage to avoid damage to the superficial temporal vessels and auriculotemporal nerve above the zygomatic arch and the parotid below the zygomatic arch (Figs. 15-21 and 15-22). Above the zygomatic arch, bluntly dissect approximately 1.5 to 2 cm anteriorly at the level of the superficial layer of the temporalis fascia. Below the zygomatic arch, bluntly dissect parallel to and along the external auditory cartilage.

Incise the superficial layer of the temporalis fascia just anterior to the tragus at the zygoma, continuing in the anterior superior direction. Carry the dissection along the posterior side of the superficial layer of the

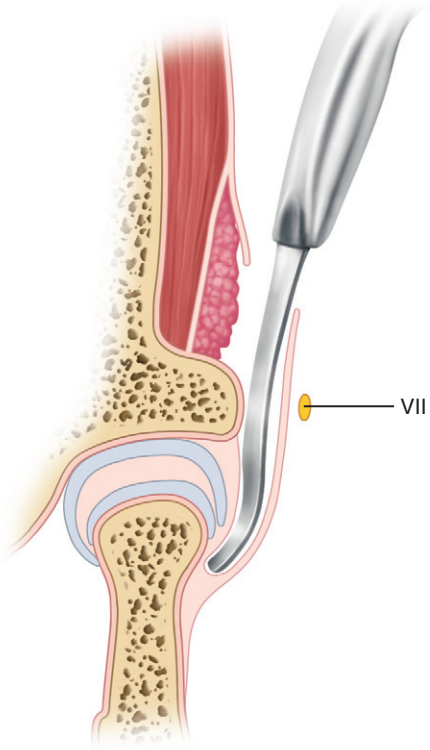


FIGURE 15-21 Dissection to joint capsule.

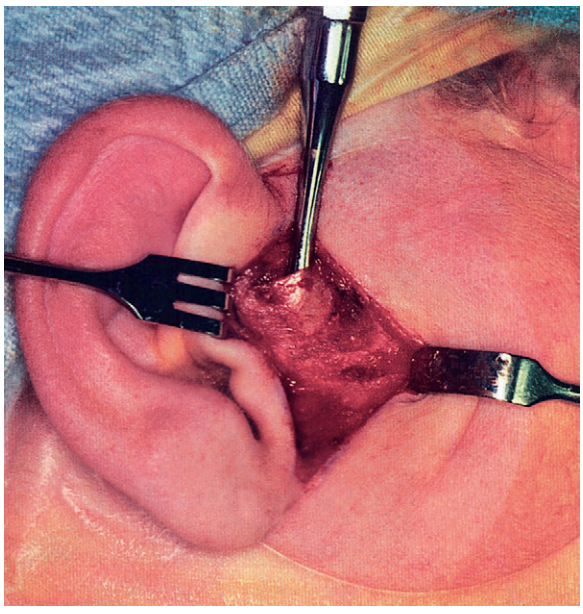


FIGURE 15-22 Exposing the joint. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, p 203.)

temporalis fascia to the zygoma; then incise and reflect the periosteum laterally. Carry the incision of the posteriormost portion of the dissected superficial layer of the temporalis fascia inferiorly, parallel to the original skin incision. Reflect and further dissect the subperiosteal flap anteriorly until the entire TMJ capsule is revealed.

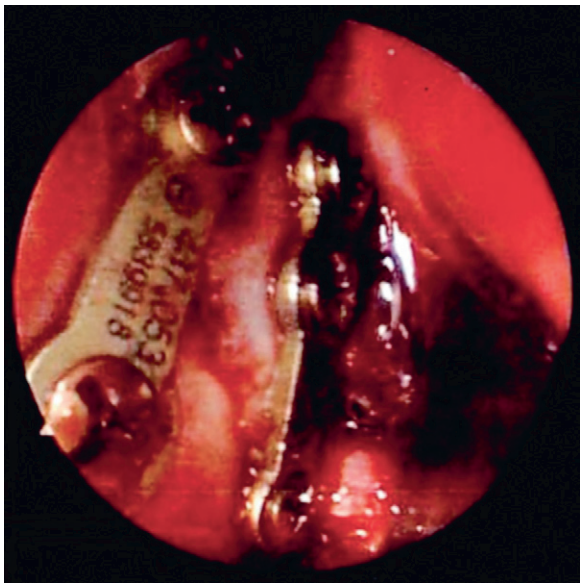


FIGURE 15-23 Disadvantages of endoscope-assisted intraoral approach. (From Zuniga Endoscope repair)

The use of a nerve stimulator is necessary through this portion of the dissection, with the absence of muscle relaxants, to identify the branches of the facial nerve.

Exposing the Superior Joint Space. Distract the condyle inferiorly and sharply dissect along the posterior slope of the articular eminence until the joint space unites with the retrodiscal tissues.

Exposing the Inferior Joint Space. Incise the disc along the lateral recess of the superior joint space. This allows exposure to the inferior joint space.

Closure

- *Inferior joint space.* Use slow resorbing or permanent suture material to reconnect the lateral disc attachments (medial) to the joint capsule (lateral).
- *Superior joint space.* Use slow resorbing sutures to attach the remnants of the TMJ capsule from the zygomatic arch to the remainder of the joint capsule.
- *Subcutaneous tissues.* Close using resorbable sutures.
- *Skin.* We recommend monocryl subcuticular sutures with a pressure dressing.

Intraoral Approach

Indications. This approach is indicated for low subcondylar fractures.[26,35,38,42,47-50] Axial anchor screws or miniplate fixation may be used.

Advantages. A visible scar avoided and damage to the facial nerve is minimized.

Disadvantages

Intraoral Approach without Endoscope. There is limited access, the poorest access of all the approaches, it is difficult to ascertain the adequacy of reduction and fixation, and there is a high rate of complications.

Endoscope-Assisted Intraoral Approach. This is more time-consuming, with a steep learning curve, poor visibility of the posterior ramus, and difficulty in reducing certain fracture types (Fig. 15-23).

Pertinent Anatomy. This is an anatomically safe approach, with minimal risks.

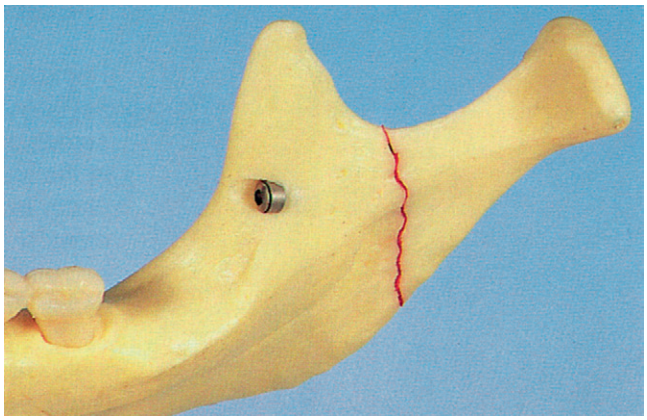


FIGURE 15-24 Intraoral axial anchor screw. (From Krenkel C: *Biomechanics and osteosynthesis of condylar neck fractures of the mandible*. Carol Stream, Ill, 1994, Quintessence, p 113.)

Surgical Technique

Incision. Incise at the anterior border of the ramus, extending to the lower buccal sulcus. This is similar to the surgical approach for a sagittal split osteotomy.[51] The incision is made through the periosteum.

Dissection and Exposure. Use a notch retractor to reflect soft tissues so the sigmoid notch can be visualized. Strip the masseter and temporalis muscles using a subperiosteal dissection. If inferior distraction of the ramus is needed, perform transcutaneously with a towel clamp.

Osteosynthesis: Miniplate and Screw Fixation

- *Preauricular transcutaneous trocar insertion.* Insert the drill and screwdriver through a trocar incision with the first drill hole placed in the proximal fragment. Insert the screw and plate via a transoral incision.
- Other screws and drill holes are placed through a trocar incision.

Osteosynthesis: Axial Anchor Screw. Position the screw just superior and parallel to the occlusal plane, superior to the mandibular foramen. Place the groove in the medial cortex approximately 1.5 to 2 cm anterior to the fracture line (Figs. 15-24 and 15-25).

A centering instrument is used to place the screw hole. A 2-mm drill is used to drill the pilot hole to the fracture line. A drill guide is placed and a 1.5-mm drill is used to penetrate beyond the fracture line into the proximal segment an additional 1 to 2 mm. Measure length and place and tighten a titanium screw with a biconcave washer.

Endoscope-Assisted Osteosynthesis. Advantages over traditional screw plate intraoral approaches include being able to access high condylar fractures, no extraoral trocar site needed, there is much better visualization and assurance of proper reduction, and fewer reported complications.[26,49,50] Equipment needed includes angled drills, a 30-degree angled 4-mm endoscope, screwdrivers, and special illuminating hooks and retractors (Figs. 15-26 and 15-27).

Insert the endoscope through a transoral or submandibular incision and identify the fracture fragments. Use one or two four-hole miniplates to fixate the fracture, depending on dislocation grade of fracture, fragment stability after reduction, and amount of space available.

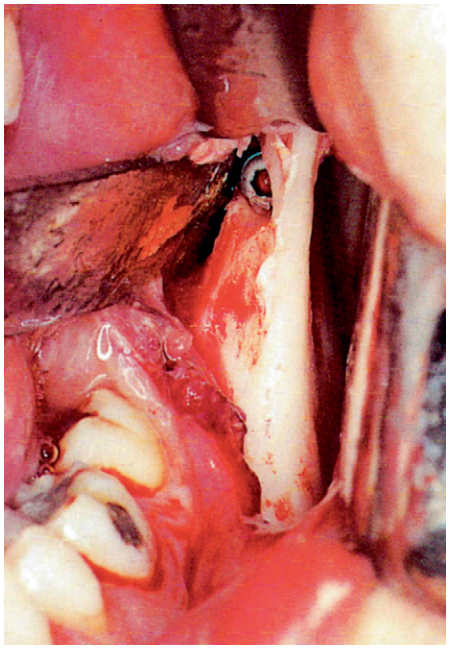


FIGURE 15-25 Axial anchor screw. (From Krenkel C: *Biomechanics and osteosynthesis of condylar neck fractures of the mandible*. Carol Stream, Ill, 1994, Quintessence, p 114.)



FIGURE 15-26 Endoscope through intraoral incision.

Closure

- *Oral mucosa.* Resorbable sutures
- *Skin.* If trocar site is present, nonresorbable sutures

Osteosynthesis of the Mandibular Condyle. The primary goals of fracture reduction are to restore ramus height and correct angular displacement. A number of approaches and dedicated instruments have been



FIGURE 15-27 Endoscope through submandibular incision.

suggested for ORIF. A comparative study evaluating osteosynthesis via the lag screw, miniplate, or Kirschner wire has noted that all techniques are acceptable, with precise repositioning of the condylar head at 90%. However, shortening of the ramus more than 5 mm was found to occur more frequently in the miniplate group than in the lag screw group ($p < .05$).[36]

- *Kirschner wires.* Thick wires and pins, used before the advent of miniplates, are required for at least 14 days of MMF. This method is obsolete because decreased range of motion and dysfunction commonly occurred.[42]
- *Miniplate systems.* This is a common osteosynthesis technique, with significant drawbacks, such as plate fractures (up to 35 %), screw loosening, and plate bending.[36,52,53]

  An elegant study by Throckmorton and Dechow has determined that the greatest amount of tensile strain exists in the anterior and lateral portions of the condyle.[54] Therefore, a double miniplate method is more successful (no plate fracture, bending, or screw loosening) and can withstand significantly greater loads than other methods, such as a single miniplate or minidynamic compression plate system.[52,55] Obviously, patients with more oblique, comminuted, and smaller fracture segments and those with unsupported posterior occlusion are most at risk for ORIF plate failures. In this group of patients, prolonging the nonchewing diet and restoring molar occlusion is encouraged.
- *Axial anchor screw.* This is generally approached through a submandibular or intraoral incision. The technique restores vertical ramus height and may result in less resorption than miniplate systems.[36] This affordable indirect technique reduces butt to butt condylar neck fractures with biconcave washers and 2-mm lag screws[12,39,42] (see Figs. 15-9, 15-10, 15-24, and 15-25.)

Finally, endoscopic exposure and fixation may reduce facial scars, but so far is more time-consuming than direct fracture approaches for any plating method.[50]

REDUCTION OF THE CONDYLE FOR ACCESS

Access and reduction of medially dislocated condylar heads is difficult and stressful to the uninitiated and inexperienced. Distraction of the mandibular ramus inferiorly on the fractured side must forcefully counteract unopposed muscle pull of the pterygomasseteric sling superiorly. After the superior ramus is opened, guiding instruments position the proximally dislocated fracture segment (Fig. 15-28). Ideally, periosteum should be preserved on the anterior and medial condylar segments, which retains lateral pterygoid vascularity.[12]

Inferior Distraction of the Ramus

A 0.5-mm retaining wire may be threaded through a bicortical drill hole and fed into a 6-mm Silastic tube to protect soft tissue during traction. This technique is effective in the submandibular approach.[12]

Ellis and Zide insert a bicortical screw in the gonial angle and wrap a 24-gauge traction wire securely around the head of the bone screw. An 18-gauge needle is introduced retrograde through the skin below the mandibular angle into the surgical field. The ends of the traction wire are pushed into the 18-gauge needle and then pulled atraumatically out the needle hole. After needle removal, inferior force on the wire twister distracts the ramus inferiorly[37] (Fig. 15-29).

Transcutaneous ramus distraction with a towel clamp may also be performed. It is usually indicated when performing an intraoral, preauricular, or transmasseter approach.

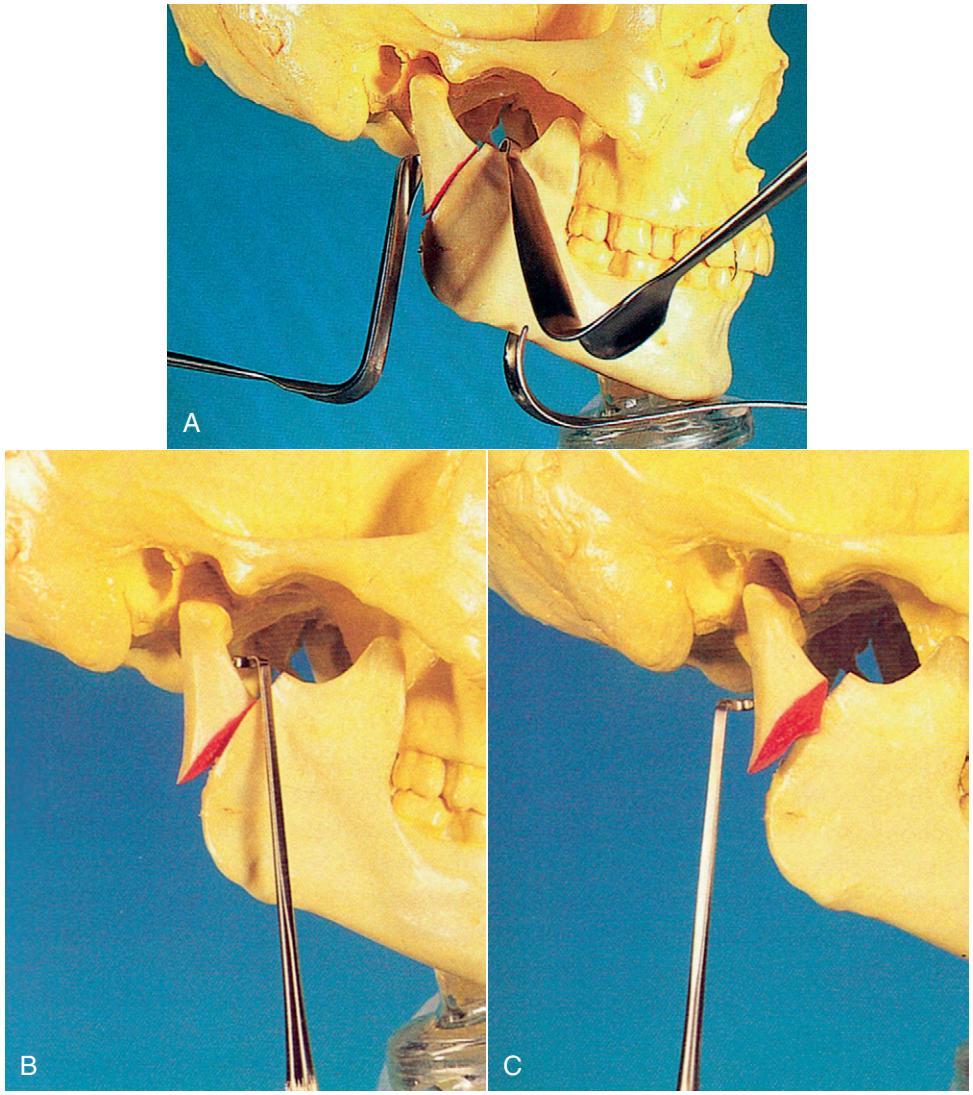


FIGURE 15-28 A-C, Guiding instruments position the proximally dislocated fracture segment. (From Krenkel C: *Biomechanics and osteosynthesis of condylar neck fractures of the mandible*. Carol Stream, Ill, 1994, Quintessence, pp 84-85.)

Difficult Proximal Condylar Dislocations

To reposition with a wire extension (Fig. 15-30), use a fine twist drill at a low-speed handpiece (at 1000 rpm) to drill a hole 2 mm anterior to the distal border of the medially dislocated proximal segment. A 0.3-mm wire approximately 50 cm long is threaded through the hole. A vascular clamp secures the wire. The condyle is then reduced with inferior traction and a repositioning fork.

Alternatively, an axial repositioning screw (≈27 to 30 mm) may be inserted within the exposed fractured surface of the proximal fragment at an angle of 1 to 3 degrees to the dorsal border of the fragment. After reduction, the repositioning screw may be removed or retained secondarily anchored in the ramus (see Fig 15-10).

Our preferred technique is to drill a screw hole. A screw is inserted in the hole through the second hole of the plate and the plate and screw are used to manipulate the condylar head into position. When that is not enough, another traction location is selected or a preauricular incision is opened.[12]

COMPLICATIONS OF CONDYLAR FRACTURE TREATMENT

The most frequent complications after ORIF or CR are malocclusion, hypomobility (8% to 10%), ankylosis (0.2% to 0.4 %), asymmetry, dysfunction or degeneration, and iatrogenic injury.[13]

Malocclusion. This is often implied secondary to improper treatment, such as inadequate use of occlusal guidance or closed reduction when open reduction is indicated.[13] However, this is disingenuous because malocclusion or centric relation discrepancies are common secondary to displaced fractures treated with CR methods. Older patients (>50 years) with preexisting locked-in occlusion and definitive wear facets often complain of occlusal disharmony, even after proper physiotherapy. Patients presenting with displaced fractures, ramus shortening, and early objective malocclusion will often have persistent centric relation discrepancies when treated in a closed fashion. Malocclusion is also common when patients have prolonged hospitalizations, other

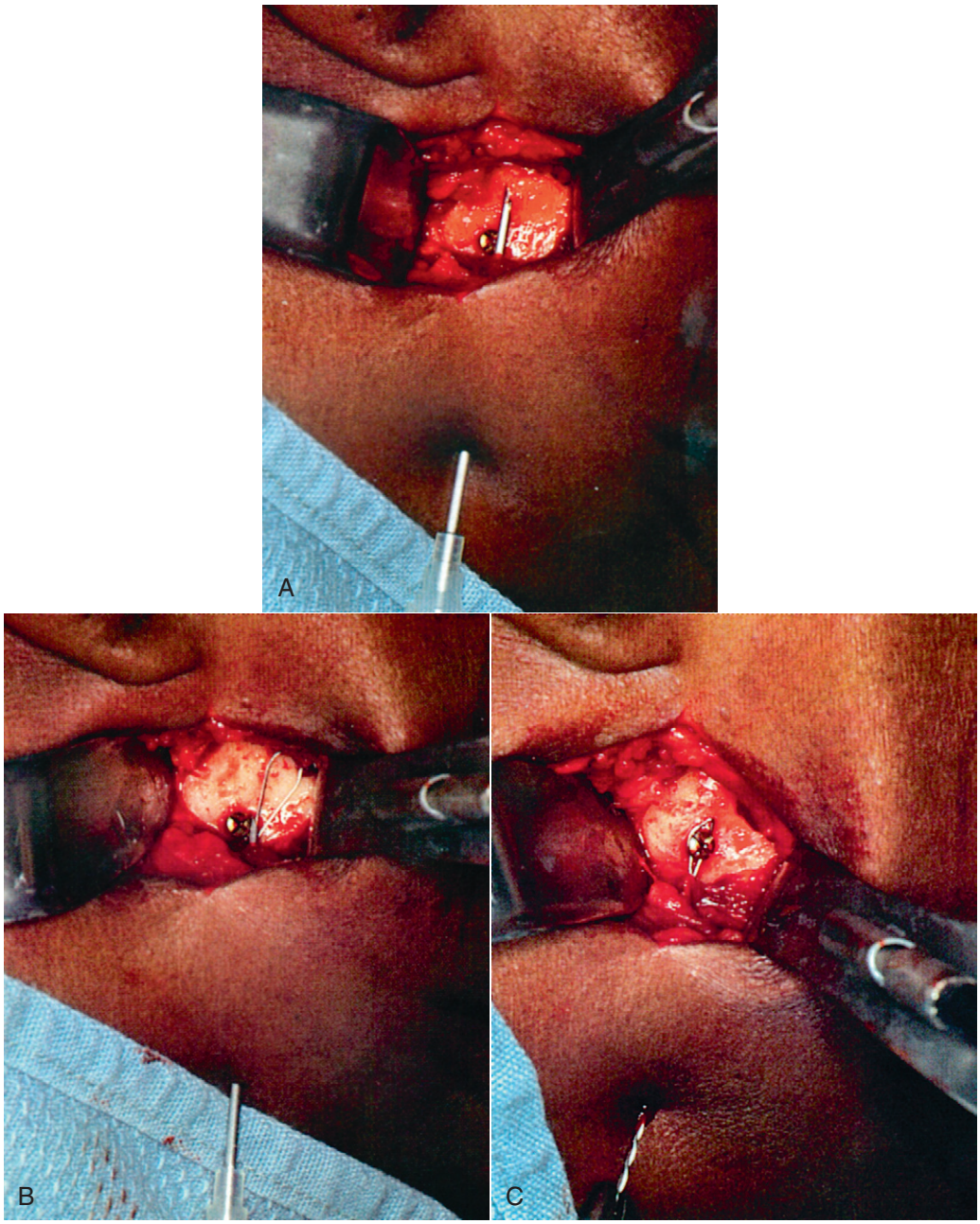


FIGURE 15-29 Inferior distraction using bicortical screw and 24-gauge wire. (From Ellis E III, Zide MF: *Surgical approaches to the facial skeleton*, ed 2, Philadelphia, 2006, Lippincott Williams & Wilkins, p 180.)

complex injuries, or any medical limitation to adaptive physiotherapy.

Mandibular Hypomobility. This is related to delayed physiotherapy of the joint and has been shown to increase the longer the patient is subjected to MMF.[56] Children are more susceptible to hypomobility, as are those subjected to high-energy injuries with capsular disruption.

Ankylosis. In children, ankylosis is related to severe meniscal disruption with inappropriate physiotherapy.[57] In adults, ankylosis usually results from a widened mandible, which leads to superior lateral displacement of the condyle. This may be mitigated by proper reduction of the fractures restricting mandibular widening.[58]

Asymmetry. In children, approximately 25% of condylar fractures will produce some facial asymmetry, whether it is hypoplasia or hyperplasia.[57] In adults, deviation on opening has been noted in up to 50% of individuals following fracture of the condyle.[13]

Dysfunction or Degeneration. All injured joints are more susceptible to arthritis, and the TMJ is no different. Risk factors include increased age, displaced condyle, longer periods of MMF, and hypomobility secondary to capsular or meniscal injuries.[13]

Condylar Resorption. This is somewhat avoidable by limiting the total denudation of the blood supply and proper anatomic reduction. However, difficult cases may

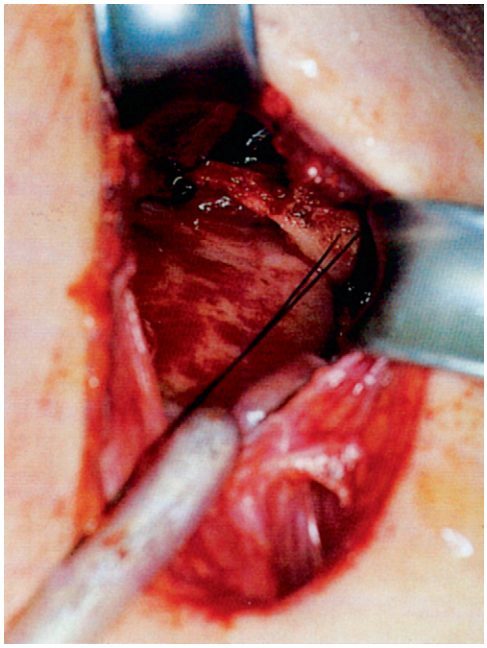


FIGURE 15-30 Repositioning with wire extension. (From Krenkel C: *Biomechanics and osteosynthesis of condylar neck fractures of the mandible*. Carol Stream, Ill, 1994, Quintessence)

demand replacement of the condylar head as a free bone graft.

Iatrogenic Injury. Following surgical repair of condylar fractures, up to 15% of patients may experience transient facial nerve weakness; however, permanent injury is rare.[13]

Chronic Pain. This occurs more commonly when condylar fractures are treated with closed reduction.*

More patients will have superior results when the surgeon correctly judges a proper path through the complexity of condylar fracture variables. Without proper physiotherapy, marginal results will be the norm. Even with proper physiotherapy, there will be patients with late malocclusions and arthritis. These patients may lead to us questioning whether the results might have improved with a different surgical or nonsurgical plan.

Open reduction is now an accepted modality that should be considered during the first 3 weeks after fracture. Patients should be informed that a closed reduction might not be fully therapeutic. Reduced swelling and reassessment present opportunities.

Nevertheless, use of open reduction techniques implies that no single technique is applicable to all patients. A surgeon may become aware of this during the delayed open reduction of a small condylar fragment in an older patient with a malocclusion. We have noted worse complications in patients who have been properly planned with open reduction than with closed reduction. However, execution of the selected process became corrupted through patient noncompliance or surgical errors.

*References 14, 16, 18, 31, and 59.

In summary, with closed reduction, early postsurgical malocclusions may not be completely corrected. If the easy open reduction period of 3 weeks is over, full range of mobility is a better goal. Re-creation of presurgical proper occlusion may be wishful. Occlusal disharmonies may merit late correction with orthognathic surgery. They are not correctable by physiotherapy alone after 8 weeks.

Frustration during the execution of open techniques must be avoided. The surgeon should plan proper choices beforehand if, perhaps, the mental picture of his or her finished surgery must be adjusted to the reality of the situation. It is worse to place a single screw in an obliquely fractured proximal segment than to let the segment go free, and it is worse to remove a displaced condylar head than to leave it in situ or fixate it properly as a free graft.

Arthritic noises and painful sequelae may develop after trauma to the joint and capsular components anywhere in the body. The TMJ is no different.

CONDYLAR FRACTURES IN CHILDREN

Condylar fractures are the most common facial fracture in children.[60] Condylar fractures may also result in significant functional deformities and facial asymmetry when improperly diagnosed or treated in children who are still undergoing significant facial growth.[3] Intracapsular crush injuries in children younger than 3 years have been demonstrated to retard growth on the affected side.[61-63] Lund has found that 30% of children with condylar fractures are more likely to have excessive growth on the fractured side.[64]

Because mandibular growth continues throughout the teenage years, most appropriately managed fractures remodel the condylar stump closer to its original form inversely with increasing age at the time of injury.[51] The younger the child, the more complete the remodeling.[6,65,66] As skeletal growth ceases, so does its regenerative capacity; only functional remodeling is possible in adults. Therefore, condylar fractures in children are managed conservatively and are rarely opened.[30]

Rehabilitation and restoration of MIO, excursive movements, and occlusion are the most important factors for proper fracture management in children. No studies have demonstrated any benefit of ORIF over CR of condylar fractures in children. In recent reviews, over 53% to 56% of respondents reported significant long-term TMJ complaints (most were minor). Results were independent of whether surgical or conservative treatment was carried out.[51,67]

Children younger than 15 years should be mobilized early and managed with a soft painless diet.[51,61,68] Guiding maxillomandibular elastics may aid in reestablishing proper occlusion when an open bite is present. There is no confirmed benefit to any period of MMF.

The absolute indications for open reduction in children are the same as those for adults and include the following: (1) displacement into the middle cranial fossa; (2) impossibility of obtaining adequate occlusion by CR; (3) lateral extracapsular displacement of the condyle; and (4) invasion by a foreign body.[30]

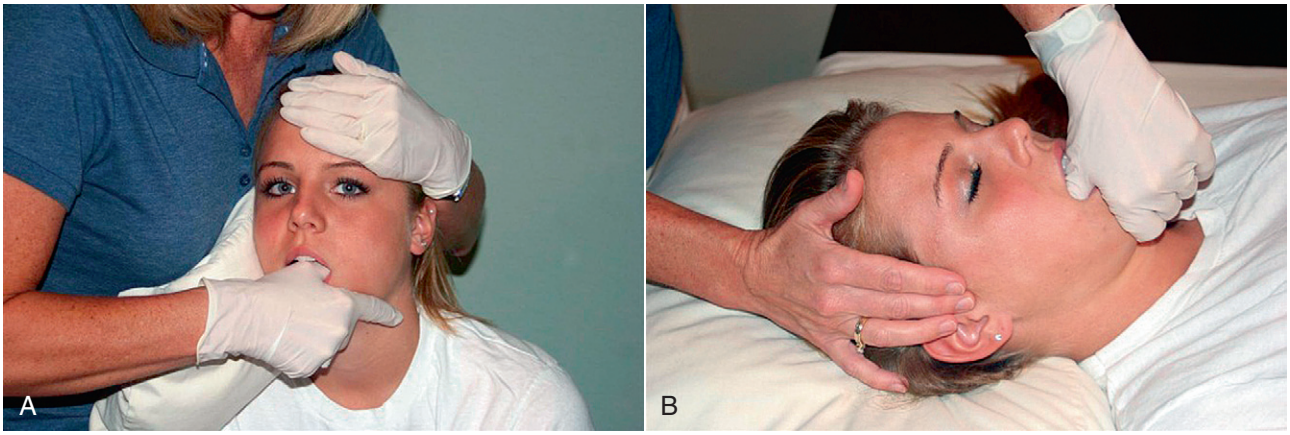


FIGURE 15-31 Manual therapy techniques. (Courtesy Julie DeVahl.)

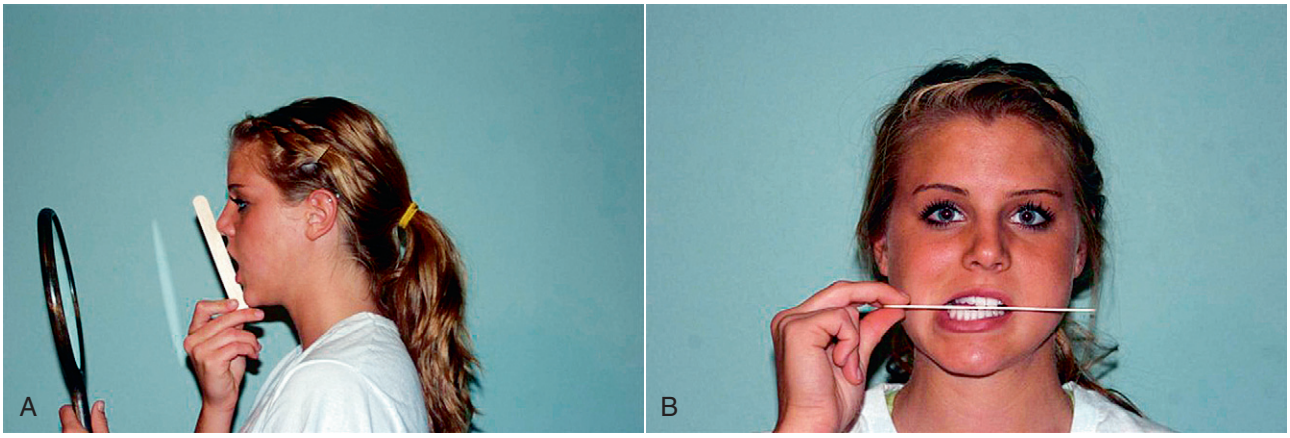


FIGURE 15-32 Active range of motion exercises. (Courtesy Julie DeVahl.)

PHYSICAL THERAPY FOR CONDYLAR FRACTURES

When the fracture site is stable and the patient is cleared for active motion, the physical therapist will examine the patient to quantify impairments and identify functional limitations. Typical postimmobilization findings include pain with mandibular movement, decreased range of motion in all planes, soft tissue adhesions, and decreased muscle strength and endurance. In addition to mandibular mobility, cervical mobility and posture are examined and deficits addressed in the rehabilitation program. Patients are frequently anxious and fearful of movement following traumatic injury and surgery, so patient education and cooperative goal setting are key to a successful recovery.

Pain experienced during initial attempts at active and passive movement normally decreases with successive attempts at motion. However, pain may inhibit the patient's willingness to move, so the application of adjunctive modalities is helpful during the initial stages of rehabilitation. Moist heat prior to range of motion exercises, followed by cold packs after exercise, can be demonstrated in the clinic and continued as a home intervention. In the presence of capsular fibrosis, ultrasound can provide deep heating of the connective tissue to prepare it for successive stretching with passive and/or active movements. Persistent pain in some patients may be managed with transcutaneous electrical nerve stimulation.

To restore mandibular range of motion, accessory motions of the TMJ must be addressed. Manual therapy techniques include joint distraction for general mobility, anterior glide to restore translation during mandibular depression greater than 20 to 25 mm and protrusion, and lateral or medial glides to restore lateral deviation required for chewing (Fig. 15-31). Soft tissue massage and myofascial release are used for soft tissue adhesions.

Following manual therapy, the patient is taught to perform active range of motion exercises. Visual feedback with a mirror is critical to enable the patient to perform early exercises properly. A tongue depressor can be used to facilitate straight opening and symmetrical lateral deviation (Fig. 15-32). When the fracture site is healed enough to handle stress, active-assisted opening is performed by the patient by placing her or his thumbs on the maxillary canines and/or premolars and the middle fingers on the central mandibular incisors (Fig. 15-33). The patient uses a prying open motion to facilitate movement. The same technique can be used with sustained passive stretching at end range of motion during later stages of rehabilitation.



FIGURE 15-33 Active-assisted opening by patient. (Courtesy Julie DeVahl.)

Muscle strengthening and endurance can be facilitated with progressive diet instructions. The patient is encouraged to chew on both sides of the mouth equally. In addition, isometric exercises can be performed in a progressive manner. The physical therapist demonstrates the amount of pressure to be applied manually to restrict mandibular movement in various planes and the patient performs the exercises as a home program (Fig. 15-34*A-C*).

When the patient demonstrates good control without pain, the exercises are repeated with 1 to 2 cm of mandibular depression (see Fig. 15-34*D*). Dynamic strengthening of the mandibular elevators can be performed by biting on surgical tubing with progressive wall thicknesses repetitively or with sustained holds.

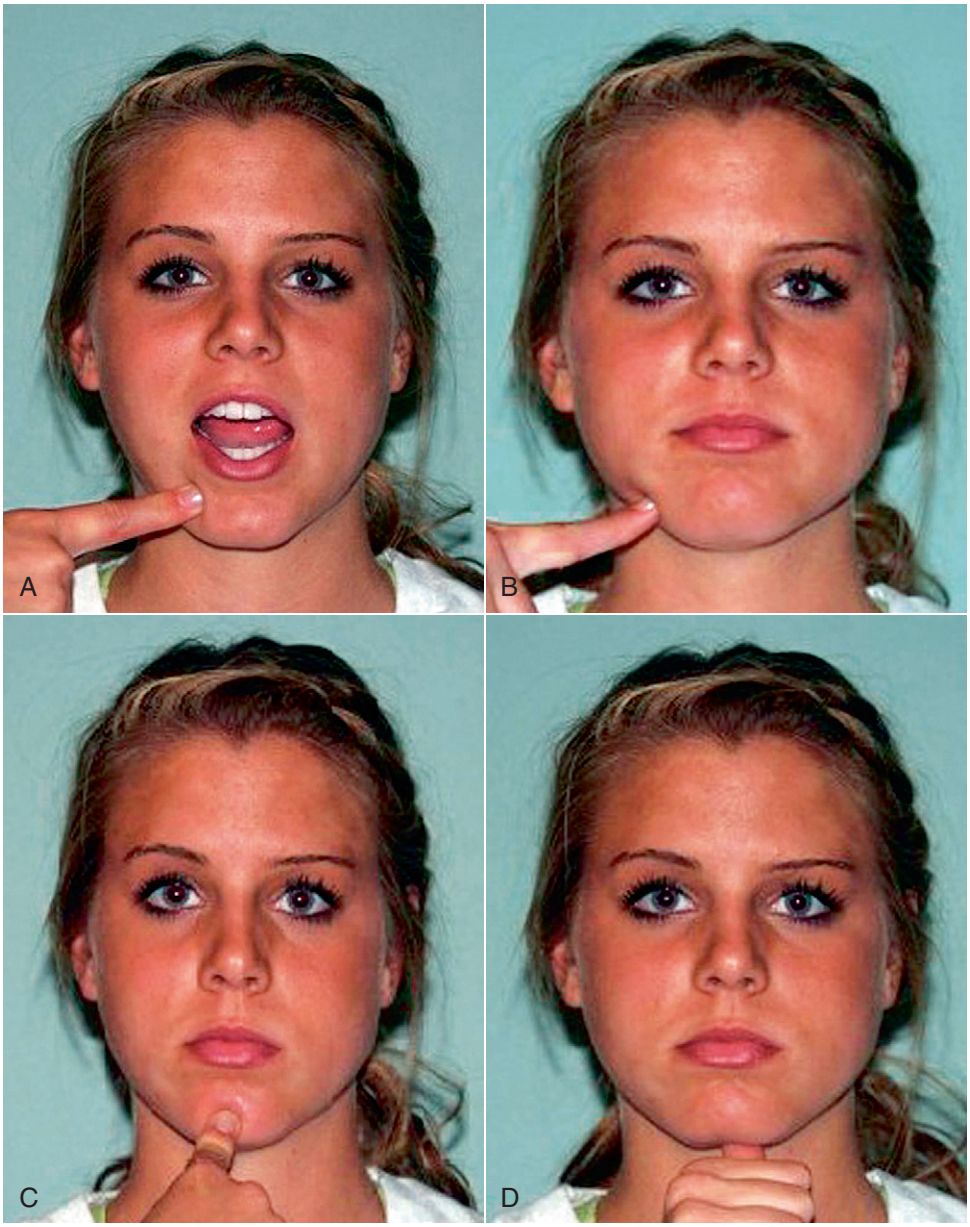


FIGURE 15-34 Muscle strengthening and endurance therapy. (Courtesy Julie DeVahl.)

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CHAPTER

16

Fractures of the Zygomatic Complex and Arch

Edward Ellis, III

OUTLINE

Anatomy
Terminology and Fracture Patterns
Classification of Zygomaticomaxillary Complex Fractures
Diagnosis of Zygomaticomaxillary Complex Fractures
Clinical Examination
Radiologic Evaluation
Treatment of Zygomaticomaxillary Complex Fractures
Determining Whether the Zygoma Has Been Properly Reduced
Need for Fixation
Need for Internal Orbital Reconstruction
Principles in the Treatment of Zygomaticomaxillary Complex Fractures
Surgical Approaches to Zygomaticomaxillary Complex Fractures
Reduction Techniques
Fixation Techniques
Internal Orbital Reconstruction
Intrasinus Approach to the Orbital Floor
Patients Treated for Zygomaticomaxillary Complex Fractures
Zygomatic Arch Fractures
Complications
Periorbital Incision Problems
Infraorbital Nerve Disorders
Implant Extrusion, Displacement, and Infection
Persistent Diplopia
Enophthalmos
Blindness
Retrobulbar and Intraorbital Hemorrhage
Malunion of the Zygoma

If excuses are needed for the writing of the present paper, they are to be found in the comparatively common occurrence of the fracture discussed, in the extreme scarcity of mention of it or its treatment in surgical literature, and in the fact that even well-known pathologic museums do not contain a single example. Modern textbooks of surgery and fractures deal with fractures of the malar-zygomatic compound so sparingly that one must be content with a few stray references or a paragraph on maxillary fractures or be guided by a terse sentence or two covering this subject.

H.D. Gillies, T.P. Kilner, and D. Stone, 1927[1]

Zygomatic fractures are common facial injuries, representing the most common facial fracture[2-14] or the second in frequency after nasal fractures.[3,15-17] The high incidence of these fractures probably relates to the zygoma's prominent position within the facial skeleton, which frequently exposes it to traumatic forces. The incidence, cause, age, and gender predilection of zygomatic injuries vary, depending largely on the social, economic, political, and educational status of the population studied. Most studies indicate a male predilection, with a ratio of approximately 4:1 over females.[18-27] Most authors also agree that the peak incidence of such injuries occurs around the second and third decades of life.[28,29] The causes of zygomatic injury in some studies are mostly altercations, whereas in others, motor vehicle accidents (MVAs) account for a more substantial number.[30,30a] The cause of the injuries sustained is greatly affected by the nature of the population in these studies; in the former studies, the populations were from industrialized areas with high rates of unemployment, in which interpersonal violence is very high.

In zygomatic fractures caused by altercations, the left zygoma is most commonly affected,* presumably because of the greater incidence of right-handed individuals. This predilection disappears in unilateral fractures caused by MVAs. Bilateral fractures of the zygoma are uncommon and account for approximately 4% of 2067 cases of zygomatic fracture in a 10-year review by Ellis et al.[26] Bilateral fractures in that study were more commonly the result of MVAs than altercations, indicating that the trauma inflicted in MVAs is more severe than that inflicted in altercations.

Because the gross shape of the face is influenced largely by the underlying osseous structure, the zygoma plays an important role in facial contour. Disruption of zygomatic position also has great functional significance because it causes impairment of ocular and mandibular function. Therefore, for cosmetic and functional reasons, it is imperative that zygomatic injuries be properly and fully diagnosed and adequately treated.

*References 12, 18, 20, 21, 24-26, and 30.

ANATOMY

The zygoma, a major buttress of the facial skeleton, is the principal structure of the lateral midface. A thick strong bone, the zygoma is roughly quadrilateral in shape, with an outer convex (cheek) surface and an inner concave (temporal) surface. The convexity on the outer surface of the zygomatic body forms the point of greatest prominence of the cheek. Therefore, the zygoma plays a major role in facial contour.

The zygoma is roughly the equivalent of a four-sided pyramid (Fig. 16-1). It has temporal, orbital, maxillary, and frontal processes, and articulates with four bones—the frontal, sphenoid, maxillary, and temporal (Fig. 16-2). The body of the zygoma extensively articulates with the maxilla along the anterior maxilla and along the orbital floor (see Fig. 16-2*B*). The suture between these two bones lies just lateral to the infraorbital foramen and runs laterally from the infraorbital rim to the undersurface of the zygomaticomaxillary buttress. It forms the superolateral aspect and part of the superoanterior aspect of the maxillary sinus. The zygoma also has a narrow weak articulation with the zygomatic crest of the greater wing of the sphenoid bone at the lateral aspect of the inferior orbital fissure (Fig. 16-3*A*). It forms a major portion of the lateral aspect and floor of the orbit. The frontal process is thick and triangular in cross section, with facial, orbital, and temporal surfaces. Because of its thickness, it is a frequent site for wire or bone-plate fixation following fracture. The temporal process is flat and projects posteriorly to articulate with the zygomatic process of the temporal bone; the combination of the two makes up the zygomatic arch. The zygomaticotemporal articulation is a thin delicate connection, which fractures frequently and with minimal force.

The zygoma provides an origin to a major portion of the masseter muscle along the body and temporal process. In addition, the temporal fascia attaches along the arch and posterolateral edge of the temporal process. The zygoma also provides attachments for the temporal and zygomatic muscles. The strong infraorbital and lateral orbital rims provide protection to the orbital contents.



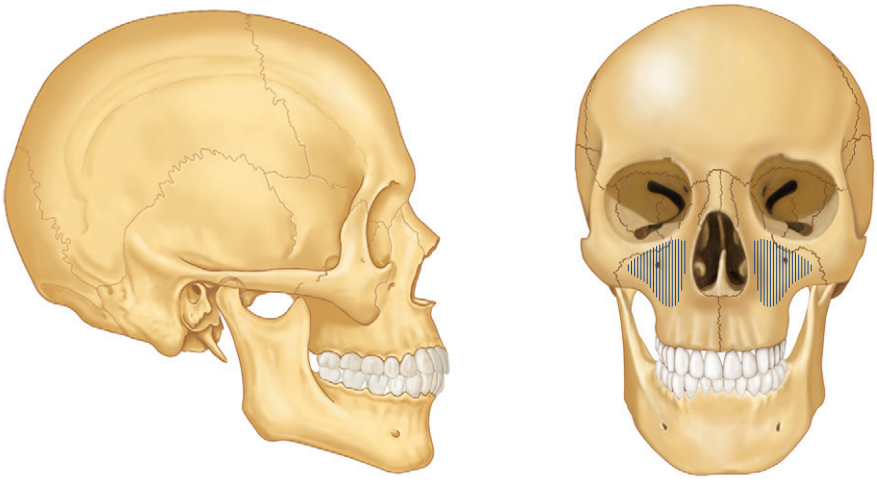
FIGURE 16-1 The disarticulated zygoma has four processes—the frontal, temporal, orbital, and maxillary—and constitutes the lateral portion of the orbit.

TERMINOLOGY AND FRACTURE PATTERNS

The malar bone represents a strong bone on fragile supports, and it is for this reason that, though the body of the bone is rarely broken, the four processes—frontal, orbital, maxillary, and zygomatic—are frequent sites of fracture.

H.D. Gillies, T.P. Kilner, and D. Stone, 1927[1]

The fracture pattern of any bone depends on several factors, including the direction and magnitude of the force. Fracture lines thus created pass through the areas of greatest weakness of a bone or between bones. Because of the strong buttressing nature of the zygoma and the thin bones surrounding it, most injuries involving the zygoma are accompanied by disruption of adjacent



A B

FIGURE 16-2 Anatomic position of the zygoma. **A,** Lateral skull demonstrating its articulation with the temporal, frontal, and maxillary bones. **B,** Frontal skull demonstrating its articulation with the maxillary, frontal, and sphenoid bones. The *hatch marks* demonstrate the extent of the maxillary sinus. Note that the zygoma forms the superolateral aspect of the sinus.

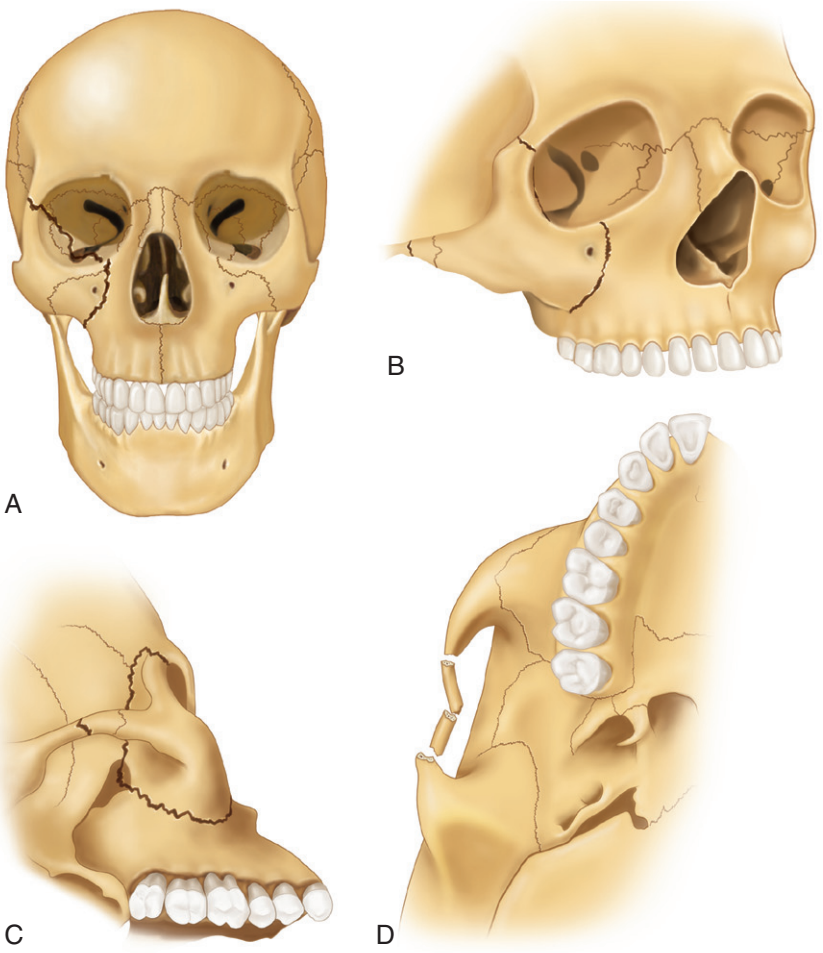


FIGURE 16-3 Common fracture pattern in ZMC injury. **A,** Frontal view of skull showing fracture medial to a zygomaticomaxillary suture and along a zygomaticosphenoid suture within orbit. **B,** Oblique frontal view of skull showing fractures through a frontozygomatic suture and posterior to a zygomaticotemporal suture. **C,** Temporal view of skull showing fractures extending from the inferior orbital fissure superiorly through the zygomaticosphenoid suture and inferiorly through the zygomatic buttress of the maxilla. **D,** Inferior skull showing a triple fracture through the zygomatic arch. Note that the orbital floor, medial orbital wall, and zygomaticomaxillary buttress are frequently comminuted in addition to the fracture patterns described.

articulating bones. This disruption occurs because when a force is applied to the body of the zygoma, it is distributed through its four processes to the adjacent articulating bones, many of which are weaker than the zygoma. Although the zygomatic bone is involved, it is rare to have an isolated fracture of the zygoma in which the fracture lines are completely within this bone or through only the sutures surrounding it.

Zygomatic or *malar fractures* are the terms commonly used to describe fractures that involve the lateral third of the middle face. Because of the impure nature of zygomatic fractures, other terms have been adopted in describing such fractures. *Zygomaticomaxillary complex, zygomaticomaxillary compound,*[31] *zygomatico-orbital,*[26] *zygomatic complex,*[32,33] *malar, trimalar,* and *tripod fractures* are terms that have been used to describe the clinical entity of fractures involving the zygoma and adjacent bones. The latter two terms are misnomers because the zygoma has not three but four processes, and their use should be condemned. Zygomatic, zygomatic complex, or zygomaticomaxillary complex (ZMC) are perhaps the most commonly used. They are used throughout this chapter because the zygoma is the major bone involved in such fractures and for the sake of simplicity. The term *zygomatic* or *ZMC* helps distinguish fractures that involve the zygoma and adjacent bones from isolated zygomatic arch fractures, and they are used when this distinction is necessary.

The inferior orbital fissure is the key to remembering the usual lines of ZMC fractures. Three lines of fracture extend from the inferior orbital fissure in an anteromedial, superolateral, and inferior direction (see Fig. 16-3). One fracture extends from the inferior orbital fissure anteromedially along the orbital floor, mostly through the orbital process of the maxilla toward the infraorbital rim. The orbital floor and medial wall are often comminuted, creating multiple lines of fracture within the internal orbit. The infraorbital canal is usually crossed by the fracture line(s) because the fracture frequently extends through the infraorbital rim to the facial surface of the maxilla, above or even slightly medial to the infraorbital foramen. The fracture extends from the infraorbital rim in the maxilla laterally and inferiorly under the zygomatic buttress of the maxilla. Comminution of the infraorbital rim and bone along the anterior and lateral maxilla is common, with frequent involvement of the infraorbital foramen. Therefore, the fracture rarely involves the zygomatic bone along the orbital floor and the anterior and lateral aspects of the face. The fracture lines are mostly within the maxilla.

A second line of fracture from the inferior orbital fissure runs inferiorly through the posterior (infratemporal) aspect of the maxilla and joins the fracture from the anterior aspect of the maxilla, under the zygomaticomaxillary buttress (see Fig. 16-3*C*).

The third line of fracture extends superiorly from the inferior orbital fissure along the lateral orbital wall posterior to the rim, usually separating the zygomaticosphenoid suture (see Fig. 16-3*A* and *C*). Extending superiorly, laterally, and anteriorly toward the lateral orbital rim, the fracture frequently separates the frontozygomatic suture at the lateral orbital rim. However, the fracture through the lateral orbital rim is occasionally superior or inferior to the frontozygomatic suture.

A ZMC fracture that follows this pattern usually has one additional fracture line through the zygomatic arch. Because the point of least resistance to fracture is not at the zygomaticotemporal suture, but approximately 1.5 cm more posteriorly, the point of fracture when a single fracture exists is usually in the approximate middle of the zygomatic arch, in the zygomatic process of the temporal bone. Frequently, however, three fracture lines exist through the arch, producing two free segments when the fractures are complete (see Fig. 16-3*D*). These segments can be displaced by associated muscle pull or may be pushed medially into the infratemporal fossa. Often, the fractures are incomplete, or greenstick, fractures, producing a medial or lateral warping of the zygomatic arch without notable upward or downward displacement.

This description is for the common or usual ZMC fracture. However, the variability of these fractures is great because of the differences in magnitude and direction of force, amount of soft tissue covering the zygoma, and density of the adjacent bones. Frequently, the lines of fracture are in locations different from those described earlier. Using radiographs to summarize the course of fracture lines in 100 isolated zygomatic injuries, Meyer et al[11] have found fractures in the body of the zygoma in almost 40% of cases, compared with the more common medial location along the anterior maxillary surface. Single or multiple lines of fracture (i.e., comminution) may exist. Gross displacement may occur, or no displacement at all. Because of the infinite number of possible variations, one must assess each zygomatic fracture independently and determine the extent and location of the fractures present.

CLASSIFICATION OF ZYGOMATICOMAXILLARY COMPLEX FRACTURES

It is probably fair to say that classification of zygomatic fractures according to the individual who tries to describe them. The result has been a confusing array of classification systems that try to describe the anatomic position of the displaced bone or to classify fractures using position and criteria for postreduction stability.[6,22,33-39] Whether a patient receives better treatment from being classified into one system or another is doubtful, and one should not dwell on the many classification systems available. As is true for many other aspects of surgery, it is extremely rare to find two patients who have exactly the same condition.

In 1990, Manson et al[40] published a classification of midfacial fractures that was based on the amount of energy dissipated by the facial bones secondary to the traumatic force. Their classification of high-, moderate-, or low-energy fractures was based on findings on computed tomography (CT) scans. High-energy fractures had extreme displacement, comminution of the articulations, and segmentation of the bones. They noted that these required extensive exposure and fixation for a satisfactory outcome. On the other hand, lower energy fractures were characterized by displacement but without comminution of bony articulations. They noted that these could be treated by less aggressive means. Using preoperative CT findings may be the most useful way to decide how much intervention may be required before surgery.

It behooves clinicians to evaluate each case individually. Whether they choose to prescribe treatment based on the experience of others for a given class of fracture is their choice; however, with proper surgical management, the nature of the treatment should depend more on the preoperative imaging analysis and surgical findings than on statistical prescription.

DIAGNOSIS OF ZYGOMATICOMAXILLARY COMPLEX FRACTURES

In a typical case, diagnosis may be made at sight once the characteristic appearance has been fully recognized. A peculiar facies is present, due chiefly to a certain flatness of contour and an absence of expression on the affected side.

H.D. Gillies, T.P. Kilner, and D. Stone, 1927[1]

The diagnosis of zygomatic fractures is primarily based on clinical and radiologic examination, although the history frequently raises a strong suggestion of the possibility that a fracture may exist and gives an indication about the nature, direction, and force of the blow. It should be stressed that the clinical examination is frequently difficult to perform adequately because of the nature of the patient's mental state and/or the amount of facial edema and pain. The swelling may conceal facial deformity that appears only after the swelling has subsided. If the examination can be performed immediately following the injury and before the onset of edema, more information can be obtained from the clinical examinations. Because there are no sensitive indicators of zygomatic fractures (e.g., those that the teeth provide in maxillary or mandibular fractures), and because the concomitant soft tissue edema and contusion that frequently accompany zygomatic injuries can obscure clinical examination, the use of imaging and clinical findings is important in the diagnosis of ZMC fractures.

CLINICAL EXAMINATION

After the clinician has ascertained the neurologic status of a patient with suspected ZMC fracture, the first priority is determination of the visual status of the involved

eye. A thorough ocular and funduscopic examination should be performed, with complete documentation of the findings. Ocular injuries, such as vitreous hemorrhage, hyphema, globe laceration, severance of the optic nerve, and corneal abrasions, were found in 4% of patients with midfacial trauma by Turvey[12] and in 5% of zygomaticoorbital fractures by Livingston et al.[41] Ophthalmologic consultation was deemed necessary in approximately 5% of 2067 cases of zygomaticoorbital injuries reported by Ellis et al.[26] Ioannides et al[42] found significant ocular and adnexal injuries in 26% of orbital fractures. Al-Qurainy et al[43] prospectively performed ophthalmologic examinations in 363 patients who had sustained midfacial fractures. Minor or transient eye injuries, such as corneal abrasion, chemosis, mild impairment of accommodation and visual acuity, and orbital emphysema, were found in 63% of patients. Moderate injuries, such as enophthalmos, conjunctival abrasion, traumatic pupillary changes, iridodialysis, lens damage, macular edema, and moderate to severe impairment of accommodation and visual acuity, were noted in 16% of patients. Severe ophthalmic disorders, such as gross proptosis, retrobulbar hemorrhage, corneal laceration, hyphema, angle recession, severe reduction or loss of vision, visual field loss, choroidal tear involving the macula, and optic nerve injuries, were found in 12% of patients. One third of all patients with comminuted ZMC fractures suffered a severe ocular disorder. Therefore, if the clinician discovers any significant or questionable findings in patients with midfacial fractures, ophthalmologic consultation should be obtained.

Examination of the zygoma involves inspection and palpation. Inspection is performed from the frontal, lateral, superior, and inferior views. One should note symmetry, pupillary levels, presence of orbital edema and subconjunctival ecchymosis, and anterior and lateral projection of the zygomatic bodies. The most useful method for evaluating the position of the body of the zygoma is from the superior view. The patient can be placed in a recumbent position or recline in a chair. The surgeon inspects from a superior position, evaluating how the zygomatic bodies project anteriorly and laterally to the supraorbital rims, comparing one side with the other. The surgeon should lay his or her index finger below the infraorbital margins, along the zygomatic bodies, pressing into the edematous tissue to palpate and reduce the visual effect of edema simultaneously when performing this examination (Fig. 16-4).[44] The superior view is also helpful for evaluating possible depression of the zygomatic arches. One should not forget to perform an intraoral examination, because zygomatic fractures are often accompanied by ecchymosis in the superior buccal sulcus and maxillary dentoalveolar fractures.

Palpation should be systematic and thorough, and one side should be compared with the other. The orbital rims are palpated first. The surgeon palpates the infraorbital rims with the index finger, moving the finger rhythmically from side to side along the rim. The lateral orbital rims are palpated with the index finger and thumb. One should also use the index finger along the inner aspect of the lateral orbital rim because fractures may frequently be detected by palpating inside the orbital rim, as opposed to palpating along the lateral aspect. When fractures are present, palpation frequently is accompanied by exquisite tenderness. The body of the zygoma and zygomatic arch are best palpated with two or three fingers in a circular motion, with the surgeon comparing this palpation with that of the opposite side. The zygomatic buttress of the maxilla is palpated intraorally with one finger, and hematoma or irregularities are sought.

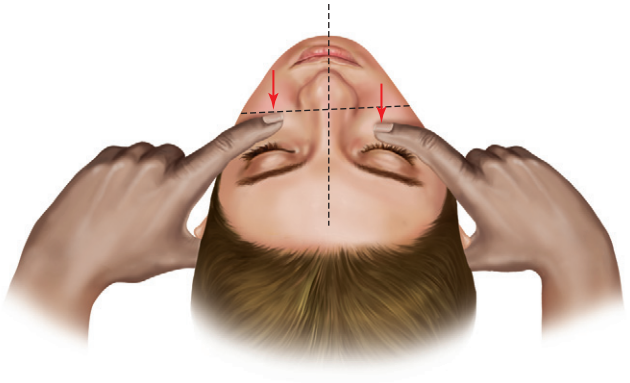


FIGURE 16-4 Method of assessing posterior displacement of the ZMC from behind the patient. The clinician should firmly depress the fingers into the edematous soft tissue while palpating along infraorbital areas.

Signs and Symptoms

Several signs and symptoms accompany zygomatic fractures. The presence and magnitude of their severity greatly depend on the extent and type of zygomatic injury. For example, facial flattening is more pronounced in injuries in which the zygomatic body has been greatly displaced, as opposed to those in which the body has not been displaced. Similarly, zygomatic arch fractures may be expected to produce less ocular disruption than ZMC fractures. The following signs and symptoms can accompany zygomatic fractures and therefore should be evaluated.

Periorbital Ecchymosis and Edema. Edema and bleeding into the loose connective tissue of the eyelids and periorbital areas is the most common sign following fracture of the orbital rim.[45,46] Swelling, often massive, may be present and is most dramatic in the periorbital tissue, where the eyelids may be swollen closed. The ecchymosis may be in the inferior lid and infraorbital area only or around the entire orbital rim.

Flattening of the Malar Prominence. A characteristic sign and striking feature of zygomatic injury is a flattening of the normal prominence in the malar area. An especially common finding in ZMC injuries, this flattening is reported in 70% to 86% of cases,[22,26,29] especially those in which distraction of the frontozygomatic suture and medial rotation and/or comminution have occurred. If edema is present, flattening may be difficult to discern soon after injury; however, one can usually gain an appreciation of this sign by depressing the index fingers into the soft tissue of the zygomatic areas and comparing one side with the other from above the patient (see Fig. 16-4).

Flattening over the Zygomatic Arch. A characteristic indentation or loss of the normal convex curvature in the temporal area accompanies fractures of the zygomatic arch. Visual and digital comparison with the opposite side is extremely helpful for detection of depressions of the zygomatic arch.

Pain. Severe pain is normally not a feature of zygomatic injuries unless the fractured segment is mobile. Patients do, however, complain of discomfort associated with the attendant bruising. Palpation of the fracture sites also elicits a painful response.

Ecchymosis of the Maxillary Buccal Sulcus. An important sign of zygomatic or maxillary fracture is ecchymosis in the maxillary buccal sulcus. Ecchymosis may occur even with a small disruption of the anterior or lateral maxilla and should be sought in patients with suspected zygomatic fractures.

Deformity at the Zygomatic Buttress of the Maxilla. Intraoral palpation of the anterior and lateral aspects of the maxilla frequently reveals irregularities of the normally smooth contour, especially in the area of the zygomatic buttress of the maxilla. Crepitation from comminuted fragments of bone is also frequently palpable. If no tenderness is experienced during this maneuver, the chances are that no fracture exists. The absence of pain makes a zygomatic fracture unlikely, but its presence does not establish one because the pain can be a result of soft tissue injury and/or maxillary fracture.

Deformity of the Orbital Margin. Fractures running through the orbital rim often result in a gap, or step deformity, if displacement has occurred. This finding is frequently noted at the infraorbital and lateral orbital rims when zygomatic fractures are present.[45,46] These areas may also be tender to touch.

Trismus. Limitation of mouth opening frequently accompanies zygomatic injuries and is present in approximately one third of cases.[26,46,47] This condition occurs with an even higher incidence in isolated fractures of the zygomatic arch (45%). The reason often cited for postfracture trismus is impingement of the translating coronoid process of the mandible on the displaced zygomatic fragments. Whether this contact actually occurs in most cases is doubtful, because the amount of displacement necessary for producing actual mechanical interference is great. A more likely explanation is muscle spasm secondary to impingement by the displaced fragments, especially on the temporal muscle (Fig. 16-5). An associated finding is deviation of the mandible toward the fractured side when the mouth is opened.

Abnormal Nerve Sensibility. An important symptom, present in approximately 50% to 90% of ZMC injuries, is impaired sensation of the infraorbital nerve.[22,26,45-52] Infraorbital nerve paresthesia is more common in fractures that are displaced than those that are not. It is difficult to differentiate true anesthesia from the altered sensation of swollen edematous tissue but, as the swelling decreases, infraorbital nerve anesthesia becomes apparent. Infraorbital anesthesia occurs when the fracture through the orbital floor and/or the anterior maxilla causes tearing, shearing, or compression of the infraorbital nerve along its canal or foramen (Fig. 16-6). Frequently, the entire orbital floor is comminuted, which results in multiple fragments of bone strung together by the infraorbital neurovascular bundle. When the line of fracture is lateral to the infraorbital groove and foramen (less common), the infraorbital nerve is spared. Disruption of the infraorbital nerve causes anesthesia of the lower eyelid, upper lip, and lateral aspect of the nose.

A related symptom may be altered sensitivity of the maxillary teeth and gingiva.[53] When this altered sensitivity is present, the clinician should suspect a disruption of the infraorbital nerve within its canal, where the middle and anterior superior alveolar nerves take origin.

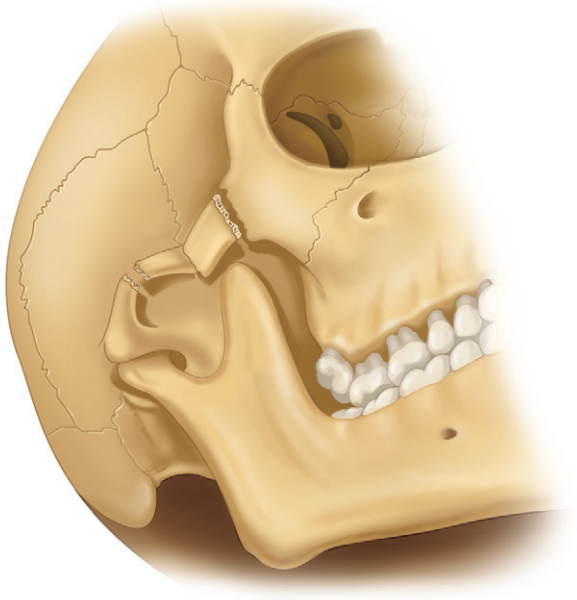


FIGURE 16-5 Depressed zygomatic arch impinging on the temporal muscle and/or coronoid process, limiting mandibular excursions.

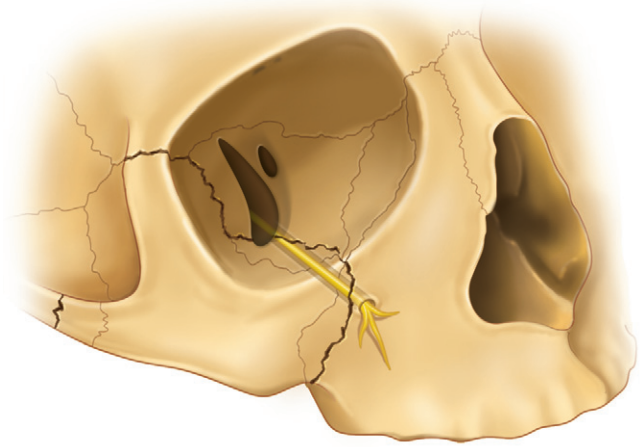


FIGURE 16-6 Typical fracture extends along or through the infraorbital groove or canal along the orbital floor and frequently across the infraorbital foramen on the facial surface of the maxilla. The orbital floor medial wall is frequently comminuted, with multiple lines of fracture crossing the infraorbital neurovascular bundle.

Epistaxis. Whenever the sinus mucosa is disrupted, hemorrhage into the sinus is possible. Most fractures through the sinus wall that have had even a minor amount of displacement tear the lining mucosa, producing internal bleeding. Because the maxillary sinus drains into the nose via the middle meatus, unilateral hemorrhage from the nose is possible and occurs in approximately 30% to 50% of ZMC injuries.[26,46]

Subconjunctival Ecchymoses. Subconjunctival hemorrhage, a frequent finding in zygomatic fractures, is present in 50% to 70% of cases.[26,46] It may accompany even a hairline crack through the orbital rim if the periosteum has been torn. Its absence does not exclude an orbital rim fracture because if no disruption of the periosteum has occurred, bleeding can accumulate in a subperiosteal location and may not be visible under the conjunctiva. When present, subconjunctival ecchymoses usually have no posterior limit and will be bright red because of the ability of oxygen to diffuse through the conjunctiva to the collection of blood.

Crepitation from Air Emphysema. Fracture through a sinus wall with tearing of the lining mucosa allows air to escape into the facial soft tissue if the pressure within the sinus is greater than that within the tissue. The soft tissue of the periorbital area, especially the eyelids, is prone to inflation with air because of its loose areolar nature. When inflation occurs, one can palpate crepitation, indicating subcutaneous emphysema. Crepitation is most easily appreciated by alternatively rolling two fingers gently over the tissue, which produces a characteristic crackling sensation. It is an uncommon finding following zygomatic fractures, but the potential for air emphysema is constant. When present, however, crepitation can be alarming to the patient. The emphysema disappears spontaneously in 2 to 4 days without treatment. The significance of emphysema is the potential for infection through the communication between the sinus and the soft tissue.

Displacement of the Palpebral Fissure. The lateral palpebral ligament is attached to the zygomatic portion of the orbital rim. Displacement of the zygoma carries the palpebral attachment with it and thus produces a dramatic visual deformity. When the zygoma is displaced in an inferior direction, the lateral palpebral ligament is also depressed, causing a downward slope to the fissure (antimongoloid slant) (Fig. 16-7). Because the orbital septum is attached to the infraorbital rim, inferior or posterior displacement of the inferior orbital rim causes depression of the lower eyelid, giving it a shortened appearance.[54] This depression may cause more sclera to be exposed below the iris and an apparent ectropion.

Unequal Pupillary Levels. With the disruption of the orbital floor and lateral aspect of the orbit that frequently accompanies zygomatic fractures, loss of osseous support for the orbital contents and displacement of Tenon's capsule and the suspensory ligaments of the globe permit depression of the globe.[55] This displacement is manifested clinically as unequal pupillary levels, with the involved pupil at a level lower than that of the normal side (see Fig. 16-7).

Diplopia. Diplopia is the name given to the symptom of blurred vision. Two varieties of diplopia exist; it is important to distinguish between them. Monocular diplopia, or blurring of vision through one eye with the other closed, requires the immediate attention of an ophthalmologist, because it usually indicates a detached lens, hyphema, or other traumatic injury to the globe. Binocular diplopia, in which the blurring of vision occurs only when the patient looks through both eyes simultaneously, is common and occurs in approximately 10% to 40% of zygomatic injuries.* Al-Qurainy et al[65] have found that the severity of diplopia is associated with the severity of midfacial injuries. Almost 30% of patients with comminuted fractures of the ZMC experienced diplopia, 22% of patients with noncomminuted displaced ZMC fractures had diplopia, and only 8% of patients with minimally displaced or nondisplaced ZMC fractures had diplopia. Binocular diplopia that develops following trauma can be the result of soft tissue (muscle or periorbital) entrapment, neuromuscular injury, intraorbital or intramuscular hematoma or edema, or a change in orbital shape, with displacement of the globe causing a muscle imbalance. Enophthalmos and globe ptosis associated with marked displacement of the globe can also cause diplopia.

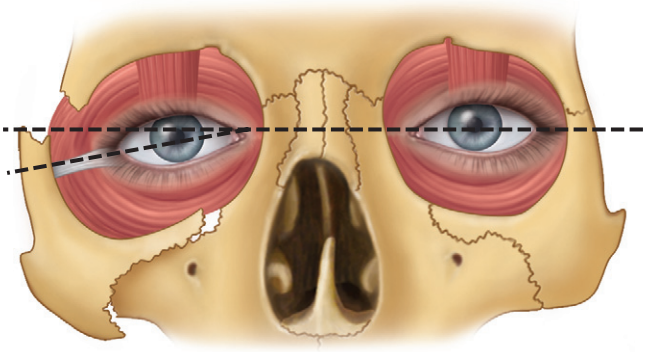


FIGURE 16-7 Inferior displacement of the zygoma results in depression of the lateral canthus and pupil because of depression of the suspensory ligaments that attach to the lateral orbital (Whitnall's) tubercle.

A useful point in differentiating the cause of diplopia is the finding that general edema of the orbit usually causes diplopia in the extremes of upward and downward gaze. Almost complete lack of eye movement in one direction is present with mechanical interference or neuromuscular injury, most commonly muscle entrapment. The diagnosis of diplopia can be difficult in the early stages of an injury, when severe edema of the orbit and eyelids is present. Diplopia of edema or hemorrhagic origin should resolve in a few days, whereas diplopia caused by entrapment of orbital tissue does not.

One can determine the presence of entrapment of orbital contents by the fracture through the orbital floor with a forced duction test. Small forceps are used to grasp the tendon of the inferior rectus through the conjunctiva of the inferior fornix and the globe is manipulated through its entire range of motion (Fig. 16-8). Inability to rotate the globe superiorly signifies entrapment of the muscles in the orbital floor. This test should differentiate

*References 15, 22, 26, 34, 35, 46, and 56-64.

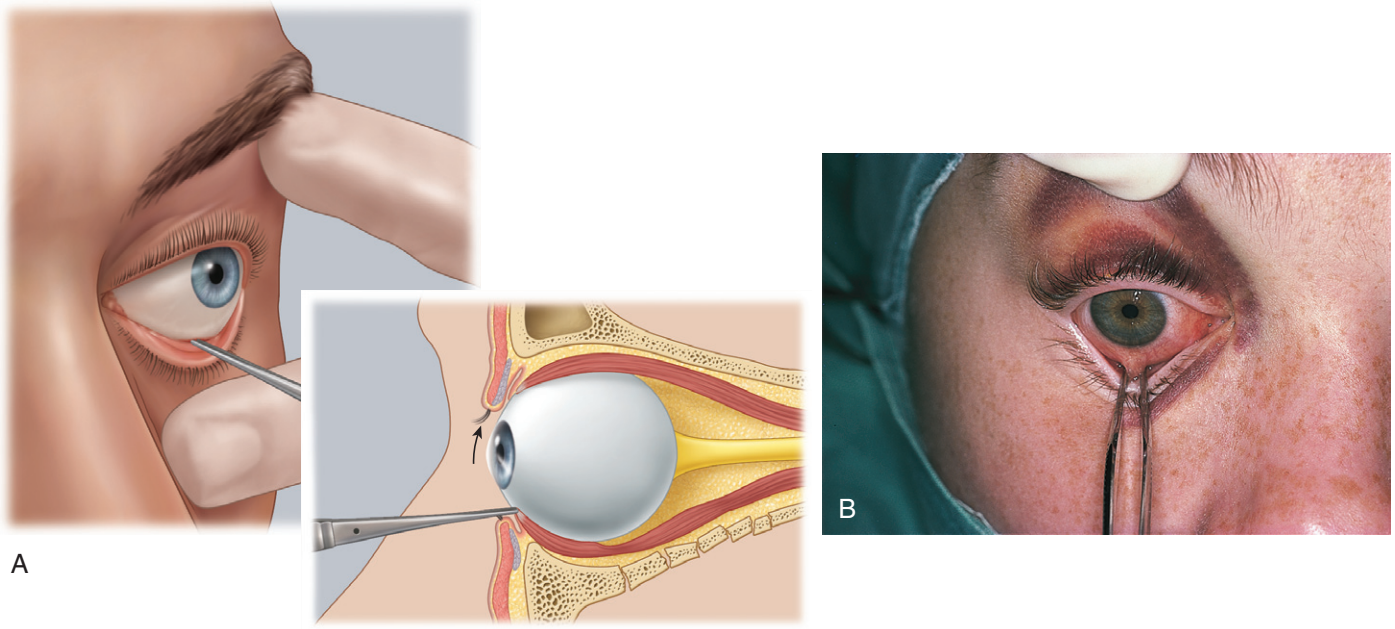


FIGURE 16-8 The forced duction test determines whether there is a physical impediment to ocular motility. **A,** Grasping of the inferior rectus muscle. **B,** Clinical photograph. (From Ward Booth P, Eppley BL, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, WB Saunders, St. Louis, 2012.)

between entrapment of orbital contents and paralysis as a result of neuromuscular injury or edema. The test should be performed routinely in those who cannot rotate the globe into an upward gaze.

Enophthalmos. If the zygomatic injury has produced an increase in orbital volume, usually by lateral and inferior displacement of the zygoma and/or disruption of the inferior, medial, and/or lateral orbital walls, or has resulted in a decrease in orbital soft tissue volume by herniation of orbital soft tissue, enophthalmos can result. This diagnosis is difficult to make acutely unless the enophthalmos is severe because adjacent soft tissue edema always produces a relative enophthalmos. After the swelling has dissipated, enophthalmos becomes more obvious and is frequently associated with ptosis of the globe. The clinical manifestations of enophthalmos are accentuation of the sulcus of the upper lid and narrowing of the palpebral fissure, causing pseudoptosis of the upper lid. The anterior projection of the globe as viewed from above is reduced on the side of injury. Zygomatic fractures are associated with enophthalmos in approximately 5% of cases before treatment.[22,26,46] If enophthalmos is present during the initial examination, it is likely that a great increase in bony orbital volume has occurred.[66,67]

RADIOLOGIC EVALUATION

Nothing is more valuable to the surgeon in determining the extent of injury and the position of the fragments—both before and after operation—than a good skiagram.

H.D. Gillies, T.P. Kilner, and D. Stone, 1927[1]

By 1992, CT essentially supplanted other radiologic methods in the assessment of patients with midfacial injuries.[68] Two-dimensional CT is now considered the best and most useful means of radiologic assessment of the injured facial skeleton.[69] The amount of information that can be obtained with CT is much greater than that which can be obtained from a series of plain films. CT accurately identifies lines of fracture, position and displacement of the ZMC, and status of the zygomatic arch (Fig. 16-9). CT scans are especially helpful in that they allow a complete assessment of the status of the orbital floor and walls and the depth to which one must dissect to reach stable bone. CT has eliminated the question about whether the orbit should be explored. With the accurate image of the internal orbit provided by CT, one can make a decision regarding the necessity for internal orbital reconstruction before surgery.

The status of the orbital soft tissue can also be assessed because of the great contrast provided by CT. Comparison of globe projection from one side with the other helps identify enophthalmos in unilateral injuries.[66,67,70,71] Also, CT scans allow identification of associated craniofacial injuries.[72] For ZMC injuries, it is optimal to obtain axial and coronal high-resolution scans. The axial scan is extremely helpful in evaluating the medial and lateral orbital walls, and the coronal scan defines the extent of injury to the orbital floor (see Fig. 16-9). Reformatted coronal views (from axial scans) are not as helpful but may be necessary if the patient cannot be properly positioned because of injury. Three-dimensional CT scans offer no additional information beyond what is already present in two-dimensional scans but are useful to understand the displacement and fracture patterns.[73,74]

TREATMENT OF ZYGOMATICOMAXILLARY COMPLEX FRACTURES

The methods of treating a fractured malar bone recommended by the various writers who have reported

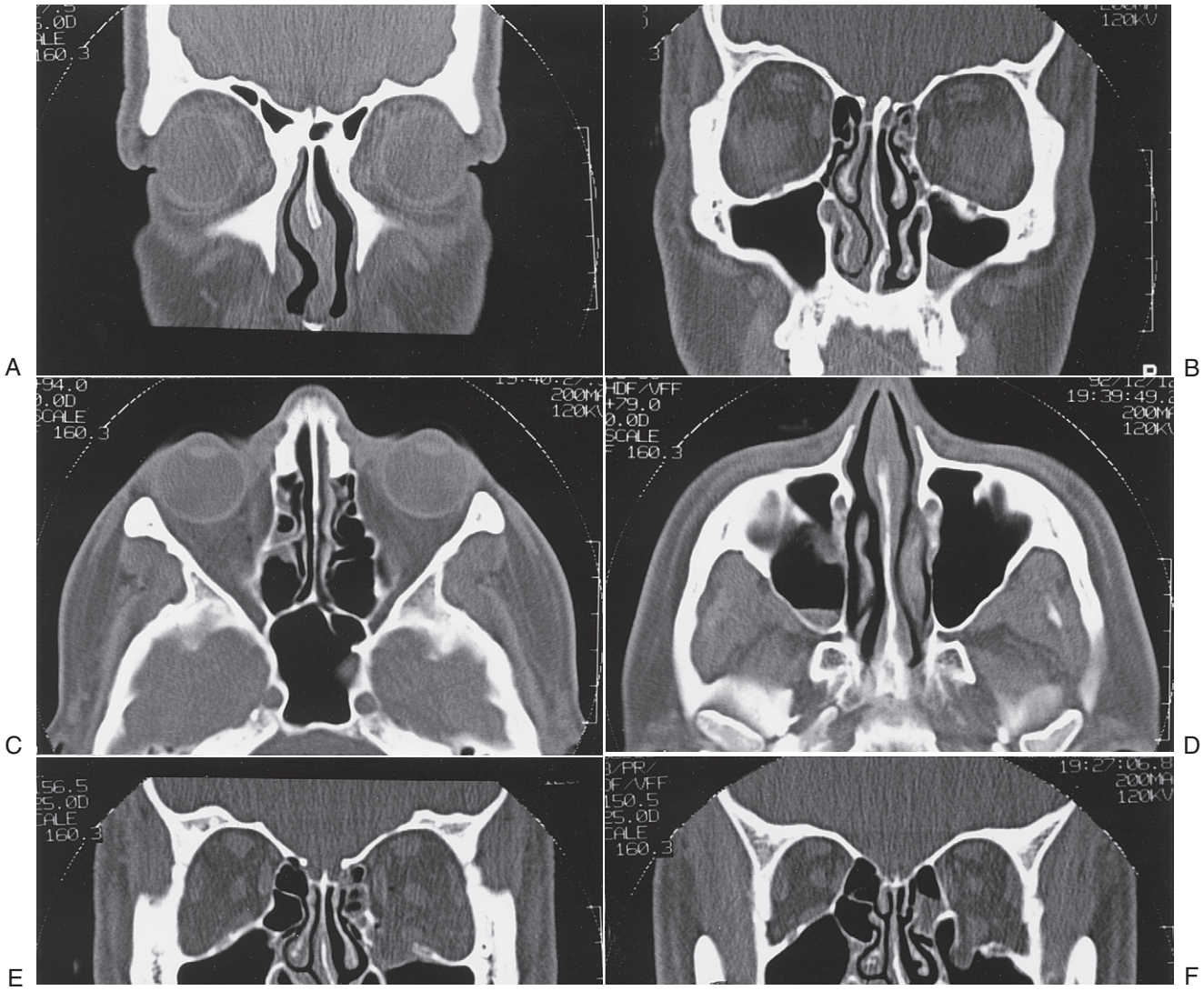


FIGURE 16-9 **A**-**D,** CT scans showing exquisite detail of a patient without a ZMC fracture demonstrating normal anatomy. **A,** Coronal CT scan through the medial orbital rim. One should be alert to the possibility of fractures in this area. If lateral displacement of the medial orbital rim goes unnoticed, proper alignment of the infraorbital rim will cause the ZMC to be laterally displaced. **B,** Coronal CT scan just posterior to the globe. The coronal scan is particularly useful for assessing the status of the medial wall and floor of the orbit. One should carefully compare the size of the orbits and the contour of the floor and walls. The coronal scan is also extremely useful for assessing the position of the malar eminence. One can follow the contour of the malar eminence inferiorly along the zygomaticomaxillary buttress. ZMC fractures have disruption of this area and the malar eminence often rotates inferomedially into the maxillary sinus. **C,** Axial CT scan at the level of the midglobe. This allows assessment of the medial wall, lateral wall, and lateral orbital rim, and the position of the globes in relation to the bony orbit and one another. Note that the lateral orbital wall is fairly straight in its course. ZMC fractures usually show displacement of the zygoma in relation to the greater wing of the sphenoid within the lateral portion of the orbit. Many ZMC fractures associated with significant orbital floor and medial wall fractures show fractures of the medial wall in this view, with the orbital contents herniating into the top of the maxillary sinus and ethmoids. **D,** Axial CT scan just below the infraorbital rim, at the level of the zygomatic arch. This view is useful for showing the status of the zygomatic arch, projection of the malar eminences, and fractures along the infratemporal surface of the ZMC. If scans are taken with the head properly positioned so that similar cuts are made bilaterally, one should compare the right and left sides for symmetry. **E** and **F,** Coronal CT scans of the orbit showing a common location of orbital floor and medial wall fractures. Note the increase in orbital volume that accompanies these injuries. Fracture defects may be small and have little orbital tissue herniating into the sinuses. Many defects associated with ZMC fractures are larger than the one shown. Examination of several cuts identifies the posterior extent of the fracture, allowing the surgeon to determine preoperatively how far posteriorly to dissect and the size of material necessary for reconstructing the defect.

TABLE 16-1 Reported Incidence of Zygomatic Fractures Not Requiring Surgical Treatment

Study	Sample Size	Percentage Not Requiring Surgery
Carlson and Märtensson, 1969[45]	144	16
Wiesenbaugh, 1970[46]	71	21
Lund, 1971[28]	62	42
Melmed, 1972[19]	270	43
Pozatek et al, 1973[76]	85	9
Haidar, 1977[18]	108	43
Larsen and Thomsen, 1978[22]	137	16
Afzelius and Rosén, 1979[13]	214	16
Adekeye, 1980[23]	337	47
Balle et al, 1982[29]	105	25
Pospisil and Fernando, 1984[77]	117	37
Fischer-Brandies and Dielert, 1984[25]	97	12
Foo, 1984[24]	77	30
Ellis et al, 1985[26]	2067	23
Kristensen and Tveterås, 1986[27]	74	49
Kaastad and Freng, 1989[78]	251	9
Covington et al, 1994[79]	259	29

cases include simple digital manipulation under general anesthesia, external manipulation by means of a cowhorn dental forceps grasping the edges of the bone, traction and elevation by means of wire or heavy bone elevators passed through small local external incisions, and elevation via incision in the mucosa of the gingival sulcus at the canine fossa.

Our technique, which has now been used successfully in a number of cases, differs from those mentioned.

H.D. Gillies, T.P. Kilner, and D. Stone, 1927[1]

Since Duverney[75] first described the fractured zygoma, numerous methods have been suggested for treating it. These range from nonintervention and observation to open reduction and internal fixation (ORIF). Because many fractures are nondisplaced or minimally displaced, intervention is not always necessary. Studies have shown that between 9% and 50% of ZMC fractures do not require operative treatment (Table 16-1).

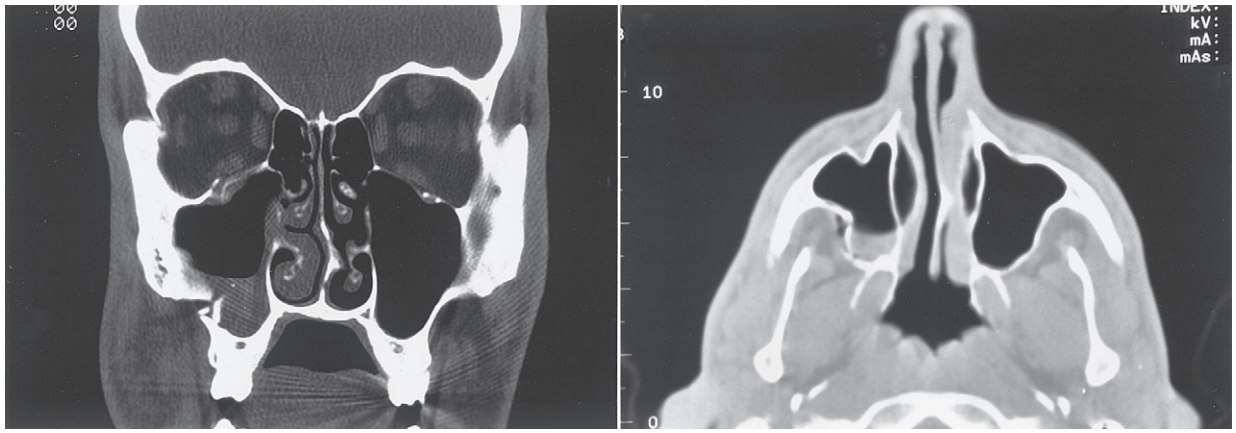
The decision to intervene should be based on signs, symptoms, and functional impairment. The decision need not be made hastily because ZMC fractures are not emergencies and treatment can be delayed, if necessary. However, during the first week following trauma, the soft tissue undergoes changes consistent with the usual sequence of wound healing. The form that they will ultimately take depends on the underlying bony architecture. If a comminuted ZMC is not treated for several days following injury, an excellent reduction may be compromised by the soft tissue scarring and change in morphology that occur between the time of injury and fracture repair. Optimally, fractures are treated before the onset of edema from the traumatic incident. In practice, however, such timing of treatment is rarely possible. When edema is moderate to severe, postponement of surgery for several days makes thorough examination and surgical treatment reliable and much easier tasks. Therefore, postponement of the decision to operate until facial edema resolves is recommended when the necessity for intervention is questionable. This approach may be used in fractures that are minimally displaced, when radiographic examination of the internal orbit shows no major defects. However, if the radiographic findings are so dramatic that intervention is definitely necessary, it may be advantageous to perform the surgery regardless of the facial edema present, because the final soft tissue contour may be superior to that which may occur when surgery is postponed. If the surgeon decides not to intervene, the patient should be observed for 2 to 3 weeks and a soft diet should be prescribed.

One should always remember that if a force is sufficient to produce a fracture of the zygoma, it is also sufficient to produce intracranial injuries. ZMC fractures are not life-threatening injuries and should not be given priority over more acute problems. Treatment need not be hastened if the neurologic state of the patient is in question because zygomatic fractures can be satisfactorily treated in several days, after the facial edema has resolved.

Another important consideration in deciding whether to intervene is the status of the opposite eye. If the patient has diminished vision in the eye on the side opposite the fracture for any reason, one may decide not to treat the displaced ZMC fracture associated with the only normally functioning eye. Although the risk to vision is minimal when ZMC fractures are treated, loss of sight in the only functioning eye would be a catastrophe. Therefore, the patient must be educated so that an informed decision can be made.

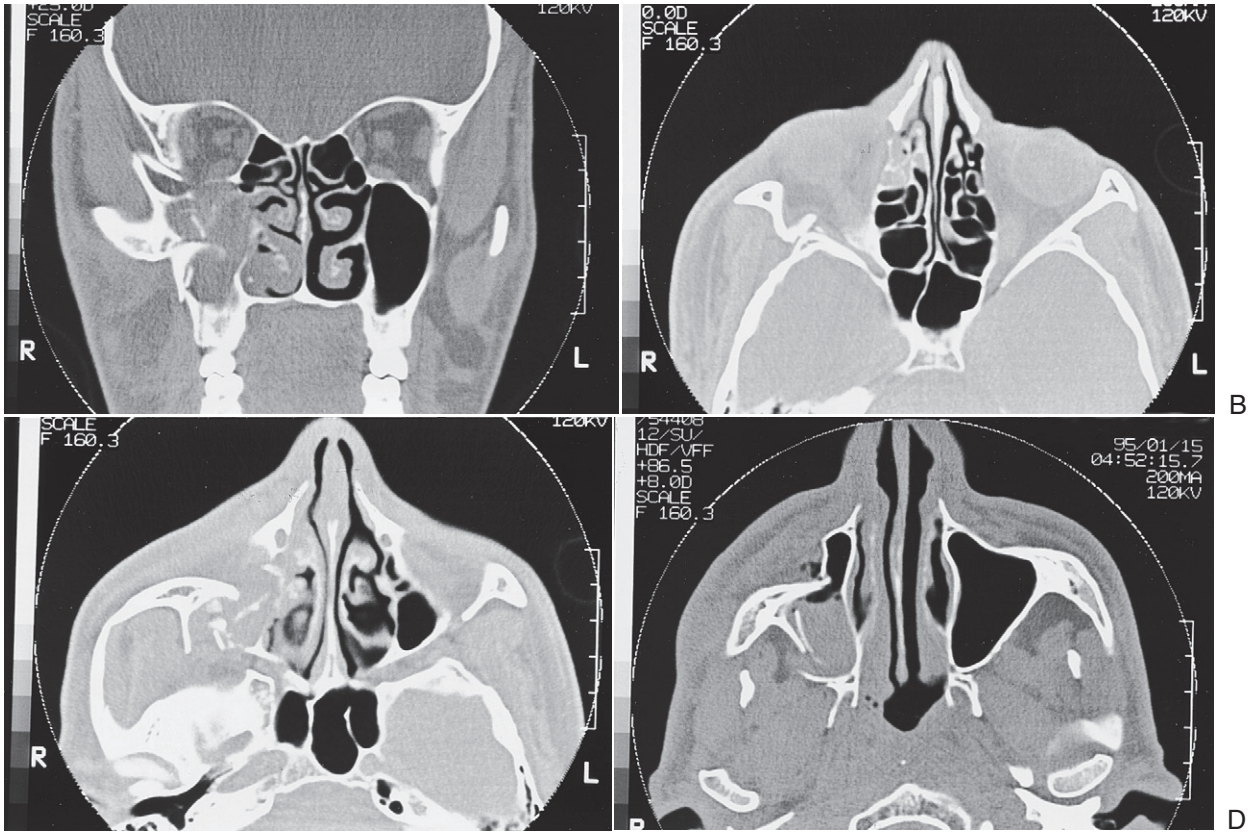
If intervention is deemed necessary, proper treatment, as for any displaced fracture, requires reduction and, if necessary, fixation. Because closed reduction using external manipulation is impossible, all reduction techniques are operative procedures (i.e., open) in the sense that the skin or mucosal surfaces are violated.

One must be aware that ZMC fractures can result from high- and low-energy injuries.[40] Those resulting from altercations seem to be more linear in character and displaced en bloc (Fig. 16-10). These fractures can frequently be treated with limited exposure, simple reduction, and simple methods of fixation, if necessary. Conversely, high-energy injuries, such as those sustained in MVAs, produce more comminution, especially of the adjacent bones, where the ZMC abuts, and are much less amenable to simple methods of treatment (Fig. 16-11). These fractures usually require extended open reduction and rigid fixation techniques. The surgeon must therefore be aware of the nature and extent of the injury as treatment is planned.



A B

FIGURE 16-10 CT scans of a low-energy ZMC fracture. **A,** Coronal scan shows rotation of the ZMC around the zygomaticofrontal suture downward and medially into the maxillary sinus. Note the difference in the position of the malar eminence from one side to the other and the disruption and displacement of the zygomaticomaxillary buttress. However, note that the fracture through the orbital floor is noncomminuted. **B,** Axial scan of the same patient shows posterior displacement of the malar eminence and rotation of the posterior (infratemporal) surface of the ZMC medially into the maxillary sinus. This fracture was treated by open reduction via an intraoral approach and fixation with a single bone plate applied along the zygomaticomaxillary buttress. The orbit was not entered.



A B C D

FIGURE 16-11 CT scans of a high-energy ZMC fracture. **A,** Coronal scan of the posterior orbit showing disruption of the orbital floor and lateral wall. Note also the comminution of the malar eminence and zygomaticomaxillary buttress. **B,** Axial scan at the level of the midglobe showing comminution of the lateral orbital wall (sphenozygomatic suture) and notable posterior displacement of the lateral orbital rim. **C,** Axial scan at the level of the zygomatic arch showing severe displacement of the malar eminence posteriorly into maxillary sinus. Note also the degree of comminution and bowing of the zygomatic arch. **D,** Axial scan at the level of the malar eminence showing severe posterior and medial displacement of the ZMC. This fracture was treated by open reduction using intraoral, coronal, and lower eyelid approaches. Several bone plates were used for fixation and the orbital floor and walls were reconstructed with bone grafts.

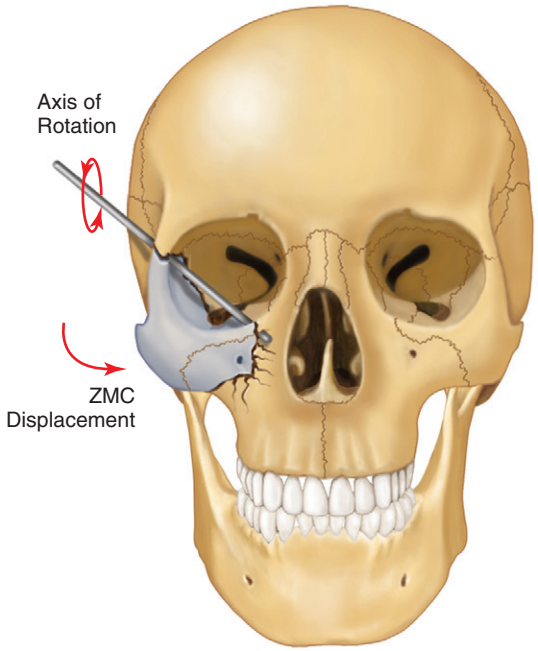


FIGURE 16-12 The ZMC can rotate inferiorly and medially, even when reduced at the frontozygomatic and infraorbital areas.

Many methods are available for the reduction and fixation of zygomatic fractures, which indicates that no one technique is always superior to the others. Few if any procedures are always satisfactory for every type of zygomatic fracture, so the surgeon's judgment and ability to apply a satisfactory modality to a given fracture are the deciding factors in whether the patient receives appropriate treatment. It should be stressed that satisfactory results can be achieved using a number of techniques. It is not so much the actual technique, but the proper application of principles, that produces satisfactory results.

The fractured zygoma is perhaps the least understood and most frequently mistreated facial fracture. Much of the difficulty in treating these fractures stems from the complex and multiple anatomic relationships that the zygoma maintains within the facial skeleton. The most common mistake made in clinical practice is to assume that the ZMC will be in its proper position if the infraorbital and lateral orbital rims have been reduced. One must remember that the fractured ZMC has four major processes that articulate with adjacent bones. Only when three are properly positioned can one be sure of an accurate reduction. It may be more helpful to think of the zygoma as a four-legged chair. If three of the four legs are on the floor, the other must also be on the floor. On the other hand, if two legs are on the floor, two may also be off the floor. Therefore, reducing orbital rim fractures (two legs of the chair) does not guarantee that the entire complex has been properly reduced, because the zygoma can rotate inferiorly and medially (Fig. 16-12). In this case, fractures through the zygomatic buttress of the maxilla and zygomatic arch are left improperly aligned,[80] producing a flattened appearance to the face in the area in which the body of the zygoma normally gives soft tissue support.

Because of the ZMC's difficult anatomic details, and because there are no sensitive clues to accurate and stable reduction, some surgeons have suggested that each ZMC fracture be treated aggressively, with ORIF of at least two of its four major processes.[17,81-85] Dingman and Natvig,[81] for example, have stated that "While closed reduction techniques are popular and attractive in the management of fractures in this region, the experienced surgeon will be quick to see, in many cases, the limitations that closed methods impose." The main controversies in the treatment of ZMC fractures are the following:

1. Should surgical exposure of the zygoma in two or three locations routinely be performed to determine whether the reduction has been adequate?
2. Should fixation devices be routinely applied?
3. Does the internal orbit require reconstruction?

It is noteworthy that the most common treatment errors that lead to poor results also center on these same topics.

DETERMINING WHETHER THE ZYGOMA HAS BEEN PROPERLY REDUCED

There should be no doubt that observation of the fracture in three of its four processes will allow the surgeon to determine the postreduction position accurately. Karlan and Cassisi[80] have shown this to be true in a clinical review of their patients. The question therefore becomes whether this is always necessary. Recommendations in the literature for reduction of ZMC fractures range from closed reduction techniques[78-79] to three- or four-point surgical exposure.[89-92] Incisions used to expose the lateral orbital, infraorbital, and zygomaticomaxillary buttress (intraorally) areas not only take time but also have the potential to produce complications of their own, regardless of the zygomatic fracture for which they are being used (see later, "Complications").

In several cases, however, surgical exposure becomes helpful. First, when preoperative signs and symptoms and/or radiographs indicate the need for internal orbital reconstruction, it is prudent to gain access to the infraorbital rim and orbital floor before elevation of the zygoma. Second, if surgery must be performed while excessive facial edema is present, surgical exposure to determine the position of the ZMC is helpful. Third, if one cannot determine whether the reduction has been adequate during the surgery, exposure will provide the necessary verification. Fourth, surgical exposure is helpful if fixation devices are deemed necessary from the preoperative assessment of the fracture. Thus, the use of surgical exposure depends on the circumstances and experience of the surgeon; however, given these examples, it will be frequently performed. If there is any doubt about the postreduction ZMC position, one should verify it with exposure, remembering that even though the orbital rims are reduced, the body of the zygoma can be rotated medially. Exposure and exploration of other areas help determine when the zygoma has been properly reduced. Fractures at the zygomatic arch and internal orbit along the greater wing of the sphenoid (Fig. 16-13*A*) are sensitive indicators of ZMC position.

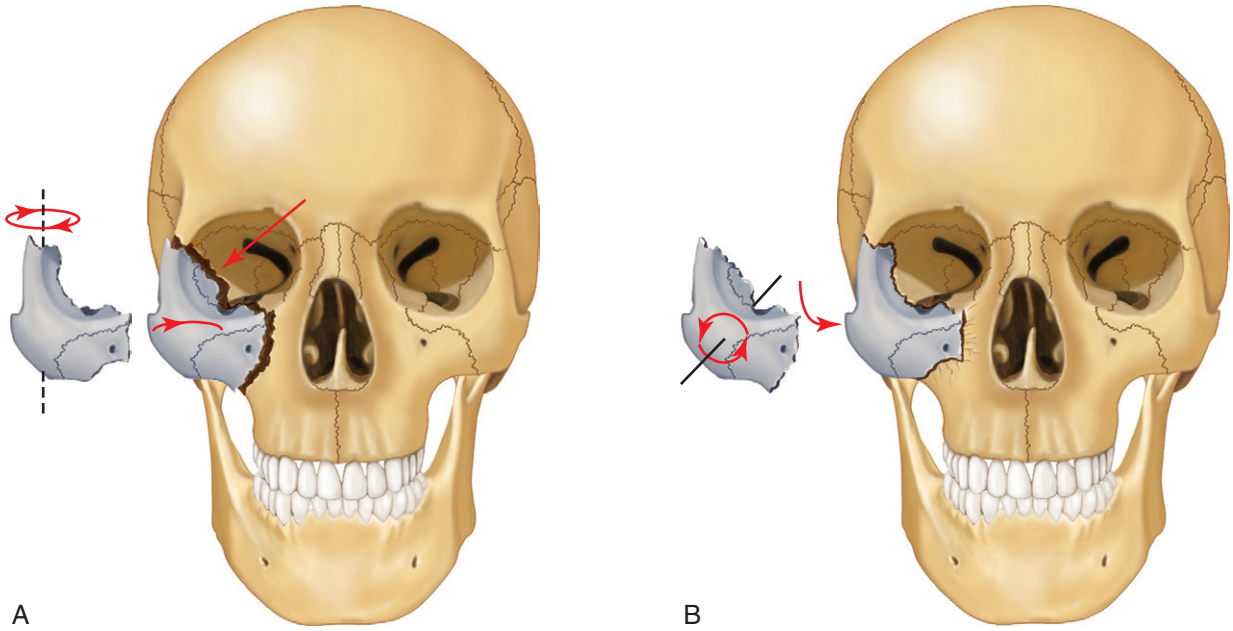


FIGURE 16-13 Anatomic areas for determining the proper reduction of ZMC fracture. **A,** Rotation of the ZMC in the vertical axis is most easily determined by its alignment with the greater wing of the sphenoid along the internal orbit *(straight arrow).* **B,** The zygomaticomaxillary buttress provides a sensitive indicator of malar projection.

However, exposure of the zygomaticomaxillary buttress (intraorally; see Fig. 16-13*B*) provides one of the most valuable clues about the adequacy of ZMC position, if it is not severely comminuted. With this exposure, one will also have excellent exposure of the infraorbital rim. It should be realized that a reciprocal relationship exists between malar projection and facial width. If the zygomatic arch is bowed laterally, the malar eminence is posteriorly displaced (Fig. 16-14). Reduction of ZMC fractures should ensure that the malar eminence is properly projected anteriorly and, if the zygomatic arch is reconstructed, the procedure is done by keeping it flat.

If the surgeon has navigation and/or intraoperative CT scanning available, the amount of surgical intervention can often be minimized. Either of these tools can assess the position of the ZMC and internal orbital floor reconstructions in the operating room. Thus, one can often use a more limited exposure, reduce the fracture, and determine whether the ZMC is in proper position.

Another important point that should be stressed in the treatment of ZMC fractures is the status of the medial orbital rim. Occasionally, a unilateral nasal-orbital-ethmoid fracture occurs on the side of the ZMC fracture, displacing the medial portion of the infraorbital rim laterally. This portion of bone may seem to be very stable and the fact that it is malpositioned may go unnoticed. If the ZMC is reduced into apposition with this laterally displaced fragment of infraorbital rim, the ZMC will be laterally positioned, increasing orbital volume and widening the face (Fig. 16-15). Because a unilateral nasal-orbital-ethmoid fracture may be difficult to diagnose clinically, preoperative CT scans are the best diagnostic tool (see Fig. 16-9*A*). Therefore, one should always search for fractures of the medial orbital rims when assessing preoperative scans.

NEED FOR FIXATION

One of the most controversial topics in maxillofacial surgery is the amount of fixation necessary to prevent postreduction displacement of the fractured ZMC.[93-95] Some have noted that reduction, by itself, does not produce adequate stability of the fractured zygoma, claiming that the downward pull of the masseter muscle will cause a medial rotation of the zygomatic body before healing.* Albright and McFarland[96] went so far as to recommend intermaxillary immobilization following fracture reduction to help reduce the pull of the masseter muscle on the repositioned ZMC. The masseter muscle has often been implicated as a primary cause of postreduction displacement of the fractured ZMC. It was assumed to be capable of exerting sufficient inferiorly directed force on the fractured ZMC to cause movement, even after surgical insertion of fixation devices. However, this contention has never been proven. There is no evidence in the literature that postreduction displacement of a ZMC fracture has occurred in patients. Previous clinical studies simply evaluated patients clinically and radiographically months after surgery and noted an occasional patient with poor ZMC position. It was assumed that because the fractures were simply elevated, or perhaps stabilized with wire fixation, postsurgical displacement had occurred. Therefore, recommendations for fixation have varied from none to the placement of three or four bone plates at different locations around the fractured ZMC.

Ellis and Kittidumkerng[99] have reviewed a series of isolated ZMC fractures treated by different approaches and fixation schemes, both immediately and several weeks after repair, and found no evidence of postreduction

*References 9, 80, 81, 84, and 96-98.

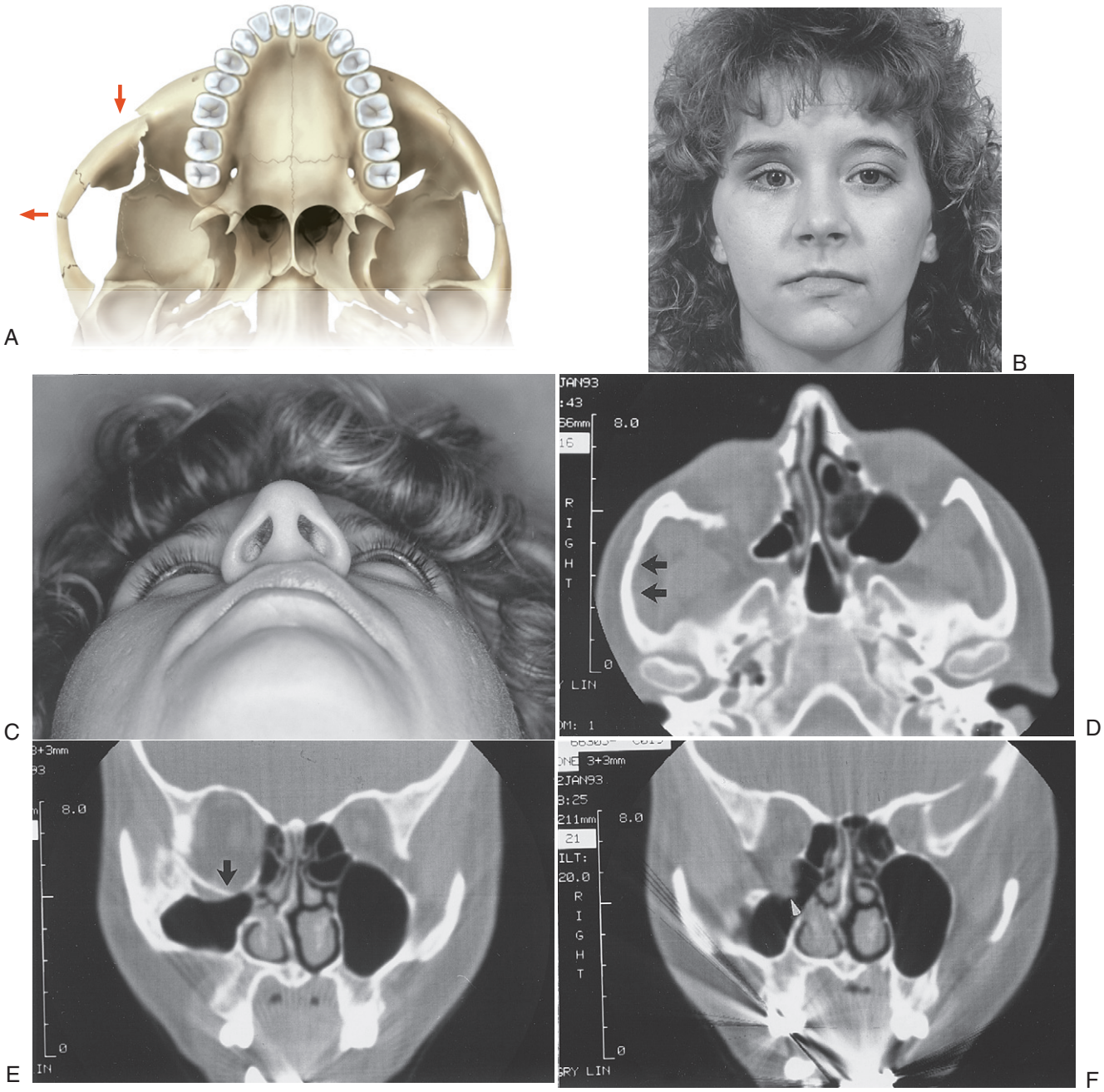


FIGURE 16-14 **A,** The reciprocal relationship between facial width and malar projection is demonstrated. Note that the normal contour of the zygomatic arch is straight, not curved. If the zygomatic arch is reconstructed with a bow, the malar eminence will lack anterior projection. Frontal **(B)** and submental **(C)** views of a patient who had open reduction and internal fixation of a right ZMC fracture 2 years previously. Note the increase in facial width and the decrease in malar projection. Also note the enophthalmos of the right globe. **D,** Axial CT scan showing lateral bowing of the zygomatic arch and the posterior position of the frontal process of the zygoma. One can see that the medial orbital wall was also inadequately reconstructed when the contour is compared with the opposite orbit. Coronal CT scan of the anterior **(E)** and posterior **(F)** orbit showing inadequate orbital reconstruction *(arrow).* The bone grafts in the anterior orbit do not maintain normal orbital shape. The grafts also did not extend far enough posteriorly. Note the great increase in orbital volume.

instability in any patient. Based on their experience and the data generated from their study, various methods can be used successfully to stabilize ZMC fractures. These range from reduction without fixation to reduction with three- or four-point fixation using bone plates.

Such a diversity of treatment options should not be surprising, given the results of a study by Dal Santo et al.[95] Thier study compared masseter muscle force in 10 male control subjects with that in 10 male patients who had sustained unilateral ZMC fractures. The calculation of muscle force was based on measured bite force, electromyograms, and radiographic determination of muscle vectors. It was found that the masseter muscle developed notably less force in patients with ZMC fractures than in



FIGURE 16-15 This patient was treated for a left ZMC fracture. Note lateral displacement of the ZMC and slight telecanthus on the left. This patient had an undiagnosed left nasal-orbital-ethmoid fracture, with lateral displacement of the frontal process of the maxilla. The left ZMC fracture was reduced to this bone, resulting in lateral displacement of the ZMC.

control subjects. Following fracture, masseter muscle force slowly increased, but at 4 weeks after surgery, most patients were still well below control levels. The results of that study cast doubt on the role of the masseter muscle in postreduction displacement of the fractured ZMC and indicate that potentially minimum amounts of fixation are required for such injuries.

Most have disagreed with the concept that fixation should be routinely applied following reduction of zygomatic fractures.* These surgeons applied fixation to zygomatic fractures only where indicated. The indications for the application of fixation seem to vary with the surgeon and type of fracture, so the incidence of fixation application varies widely in the literature (8% to 100%) (Table 16-2).

The efficacy of using simple elevation (without fixation) when indicated has been demonstrated by Larsen and Thomsen.[22] They reexamined 87 patients several months to years after elevation of their fractures by the Gillies temporal approach and found only 2 patients with residual deformity. Similarly, Fischer-Brandies and Dielert[25] reexamined 41 cases of zygomatic fracture treated with elevation using a hook and found no postsurgical displacement. Several other studies in the literature have used ZMC repositioning without fixation, with good results,[29,78-79] verifying that fixation requirements are less than advocated by some. Fixation with one bone plate has been advocated by several surgeons in a certain percentage of ZMC fractures, either at the zygomaticomaxillary buttress* or, more commonly, at the frontozygomatic area.† Champy et al[111] used a single bone plate at the frontozygomatic area in 342 isolated ZMC fractures and found that only 6 (1.8%) had an unsatisfactory result. Tarabichi[100] treated 17 consecutive low-velocity ZMC fractures by a transoral open reduction and internal bone-plate fixation of the zygomaticomaxillary buttress, with excellent results in all but 2 patients, who had comminution of the orbital rim. Covington et al[79] were able to stabilize 30% to 40% of ZMC fractures by one-point fixation. Ellis and Kittidumkerng[99] were able to use one-point fixation in 31% of ZMC fractures reported in their study. Similar results were shown by Shumrick et al.[114]

An important point regarding the stability of ZMC fracture reduction is the state of the fracture ends. Where the osseous processes are not comminuted, the fracture is more likely to remain stable without fixation devices.

TABLE 16-2 Reported Incidence of the Need for Fixation in Addition to Reduction

Study	Sample Size*	Percentage Requiring Fixation
Mansfield, 1948[57]	149	38
Nysingh, 1960[15]	200	60
Knight and North, 1961[35]	120	40
Fryer et al, 1969[104]	196	40
Wiesenbaugh, 1970[46]	75	13
Lund, 1971[28]	26	17
Melmed, 1972[19]	131	21
Pozatek et al, 1973[76]	77	58
Matsunaga et al, 1977[9]	147	100
Haidar, 1977[18]	84	77
Larsen and Thomsen, 1978[22]	137	24
Hoyt, 1979[107]	11	27
Laufer et al, 1976[112]	70	18
Adekeye, 1980[23]	179	23
Balle et al, 1982[29]	79	29
Foo, 1984[24]	43	25
Pospisil and Fernando, 1984[77]	74	39
Fischer-Brandies and Dielert, 1984[25]	81	51
Ellis et al, 1985[26]	1521	30
Champy et al, 1985[108]	1030	79
Champy et al, 1986[111]	695	77
Kristensen and Tveterås, 1986[27]	37	30
Kaastad and Freng, 1989[78]	251	55
Ogden, 1991[86]	105	8
Zingg et al, 1991[87]	813	82
Zingg et al, 1992[88]	1025	78
Covington et al, 1994[79]	259	88

*Only those cases that were surgically treated in these studies are included in this table.

*References 8, 13, 15, 22-29, 33, 61, 78, 88, 79, 95, and 99-111.

*References 95, 99, 100, 112 and 113.
†References 52, 88, 79, 89, 108, 109.

However, when comminution of the fragments has occurred, instability usually results and fixation devices become necessary. Thus, comminuted fractures behave differently from linear fractures. If there is any question about the stability of a reduced zygomatic fracture, it is prudent to apply fixation.

NEED FOR INTERNAL ORBITAL RECONSTRUCTION

By definition, the orbital floor is fractured in ZMC fractures. However, the magnitude and extent of orbital floor disruption vary from a linear crack to fragmentation of the entire floor and medial and lateral walls. Many, perhaps most, low-energy ZMC fractures do not have herniation of periorbital contents into the sinus with entrapment of ocular muscles or enophthalmos. However, these problems do occur in a certain percentage of cases. Davies[115] noted significant orbital floor disruption in 47% of patients with zygomatic fractures. Sacks and Friedland[116] noted this complication in two thirds of ZMC fractures. Crewe[117] noted notable disruption in most zygomatic fractures. Crumley and Leibsohn[64] noted that 39% of zygomatic fractures had comminuted fractures of the orbital floor. The need for orbital floor reconstruction to support the periorbital tissue was necessary in two of three cases of orbital floor exploration performed by Pozatek et al[76] and Wiesenbaugh.[46] Ellis et al[26] found it necessary to place implants in one of three cases on exploration of the orbital floor. The orbital floor and walls were reconstructed in 44% of isolated ZMC fractures in a study by Ellis and Kittidumkerng.[99] A similar study by Shumrick et al found the necessity to reconstruct the internal orbit in only 30% of ZMC fractures.[114]

Although some surgeons believe that so-called exploration of the internal orbit should be performed routinely when operating on ZMC fractures,* most do not. These surgeons would argue that exploration of the orbit should depend on preoperative and intraoperative findings. Fortunately, CT scans have eliminated the debate about when an orbit should be explored. It is now possible to obtain an accurate assessment of the status of the internal orbit before surgery so that adequate treatment can be proscribed and planned.[114,118] If comminution of the orbital floor and walls and/or prolapse of orbital soft tissue into the maxillary and ethmoid sinuses is noted, or if orbital volume has increased from blowout of the floor and walls, reconstruction should be performed.[119,120] Using similar criteria in the preoperative CT scans, Reddy and Ellis were able to classify patients successfully into those who required and those who did not require internal orbital reconstruction.[118] They showed that in those who were determined not to need internal orbital reconstruction, good radiographic and clinical results were obtained.

With the availability of intraoperative CT scanning in some operating rooms, the question about whether to reconstruct the internal orbit in those patients in whom the preoperative CT scan does not show gross disruption can be answered in the operating room. After the ZMC has been reduced, a scan is taken and an assessment of the internal orbit is made. The surgeon can then decide whether it is necessary to reconstruct the orbital walls (Fig. 16-16).

*References 17, 85, 92, 102-111, 113, 113a, 112.

PRINCIPLES IN THE TREATMENT OF ZYGOMATICOMAXILLARY COMPLEX FRACTURES

In the treatment of any ZMC fracture that requires surgical intervention, consideration should be given to each of several steps in a sequential and orderly manner (Box 16-1).

Prophylactic Antibiotics. The incidence of infection following ZMC fracture or fracture reduction is extremely low; however, such an infection is difficult to discern because many surgeons routinely use prophylactic antibiotics. This practice also makes it difficult to determine the effectiveness of antibiotics in preventing infection of these fractures. Because the maxillary sinus is involved, ZMC fractures can be considered compound, and prophylactic antibiotics are probably appropriate, especially given the fact that the orbital contents are also frequently violated. The choice of antibiotics should cover routine sinus bacteria (e.g., ampicillin, amoxicillin, clindamycin, cephalosporin).

Anesthesia. For isolated ZMC fractures, general anesthesia with oral intubation is helpful. The anesthesiologist or anesthetist should be positioned so that the surgeon has access to the side of the fracture and head of the table. It is very important to have complete access to the top of the patient's head for visual comparison of one side with the other. (Reduction of isolated zygomatic arch fractures can be performed with the patient under local anesthetic, with or without sedation when the patient is cooperative, and an intraoral or a percutaneous approach is used.)

Clinical Examination and Forced Duction Test. Following induction of general anesthesia, the surgeon should take the opportunity to examine the patient more carefully. With the patient under anesthesia, the surgeon has more freedom in the examination and can use more digital force than is possible with the patient awake. This examination can help confirm previous diagnoses and may reveal new information. It is very important to look at the patient from the superior view and to visualize both zygomas simultaneously. Unless the swelling is marked, one should be able to determine an asymmetry. Laying the index finger across the infraorbital area or on the malar prominence should help discern the asymmetry (see Fig. 16-4). A forced duction test should also be performed at this time (see Fig. 16-8).

Protection of the Globe. The cornea must be protected from inadvertent trauma. Of the several ways of providing this protection, perhaps the simplest is placement of a scleral shell (corneal shield) after application of an ophthalmic ointment (Fig. 16-17*A*). Temporary tarsorrhaphy can also be used by suturing the dermal surfaces of the upper and lower eyelids together with 5-0 nylon sutures (see Fig. 16-17*B* and *C*).

Antiseptic Preparation. The type of preparation necessary depends largely on the type of approach(es) that are anticipated. It is good practice, however, to prepare the forehead, both periorbital areas and cheeks to the level

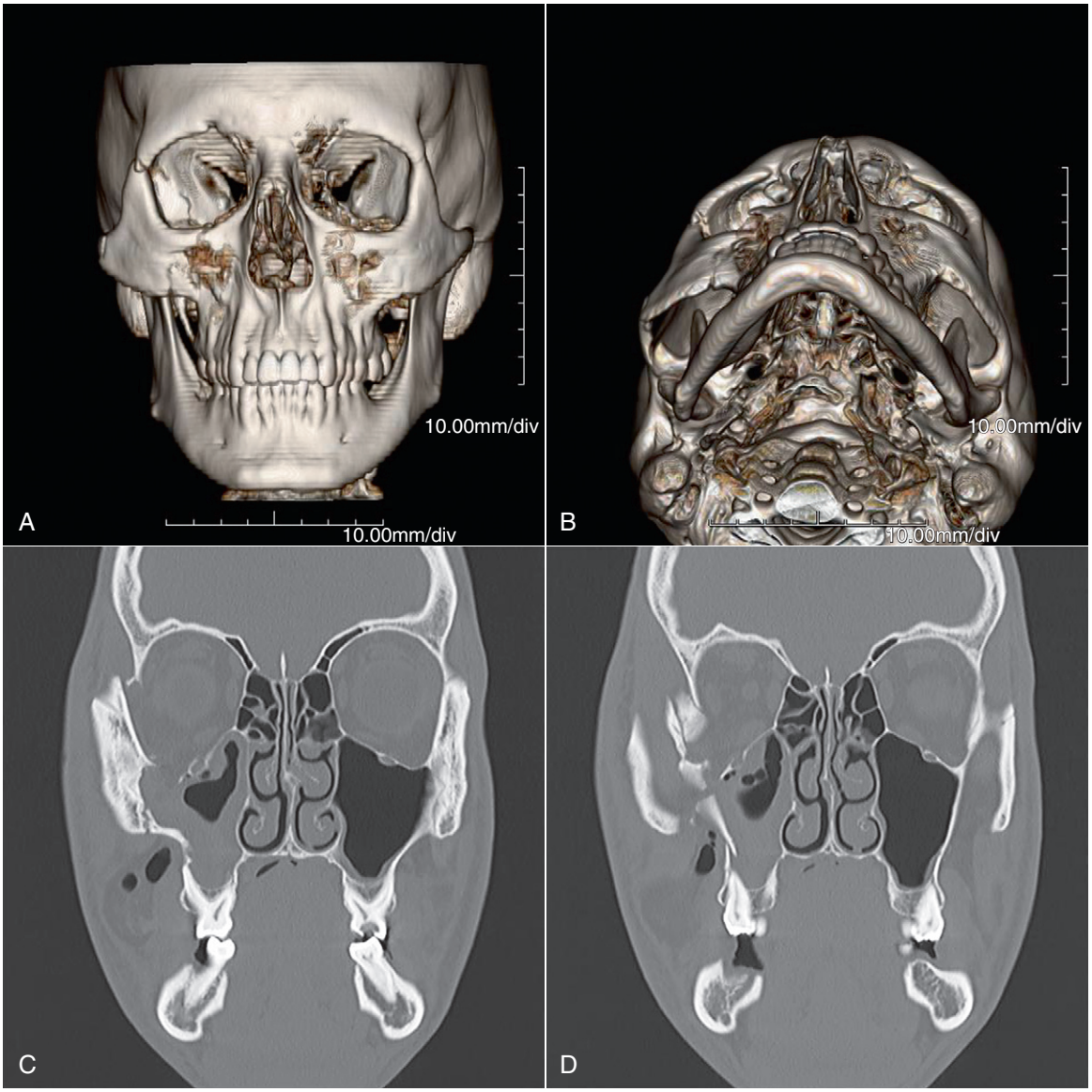


FIGURE 16-16 Use of intraoperative CT scanning. **A, B,** Preoperative three-dimensional CT scans show a moderately displaced right ZMC fracture. Note the lateral displacement of zygomatic arch and posterior displacement of malar eminence. **C, D,** Preoperative coronal CT scans show fractures of the orbital floor.

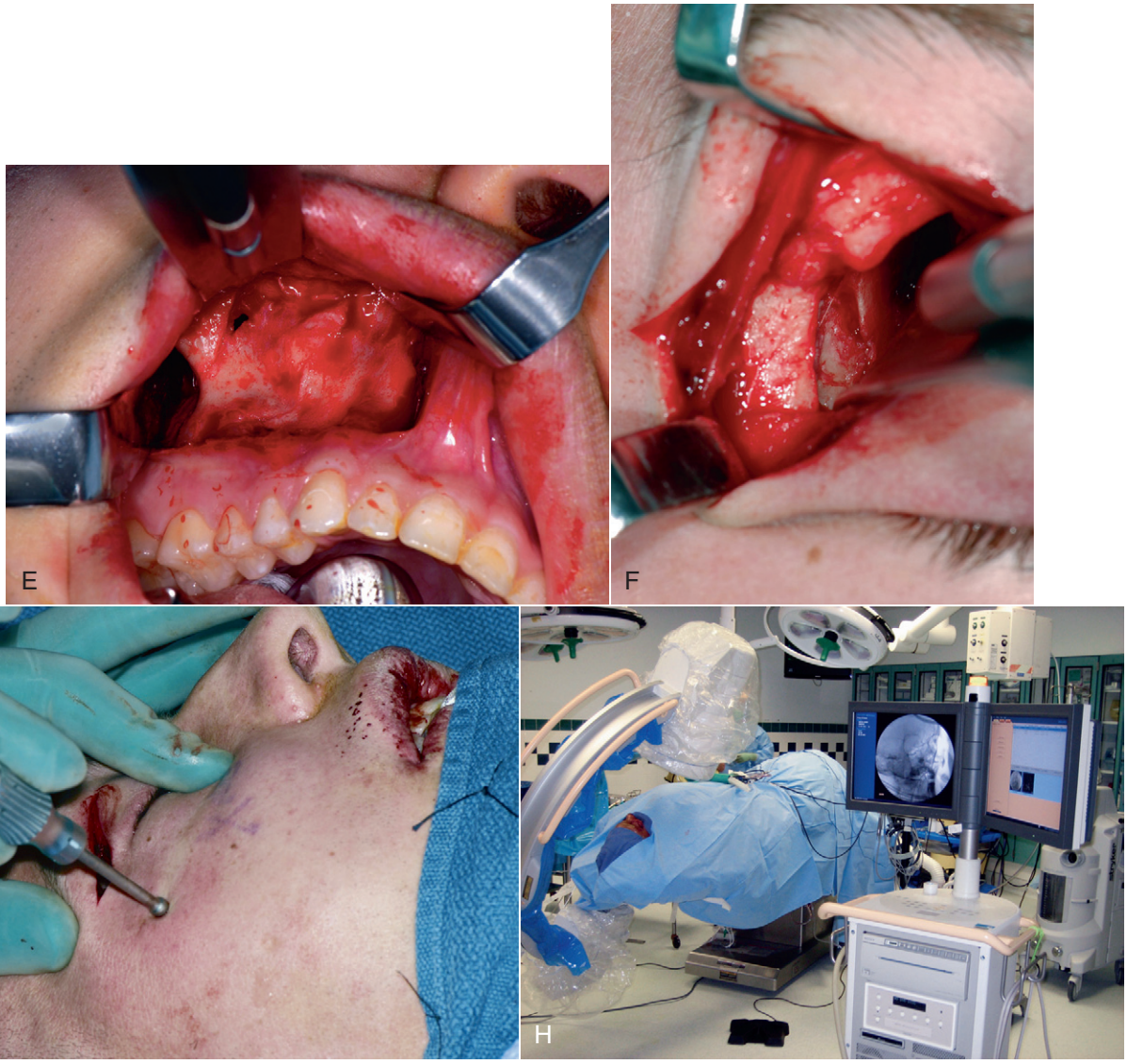


FIGURE 16-16, cont'd The ZMC fracture was exposed using maxillary vestibular **(E)** and upper eyelid approaches **(F)**. **G,** The ZMC was reduced using a Carroll-Girard screw. **H,** An intraoperative CT scan was then obtained to determine whether the reduction of the ZMC was adequate and to determine whether internal orbital reconstruction was necessary.

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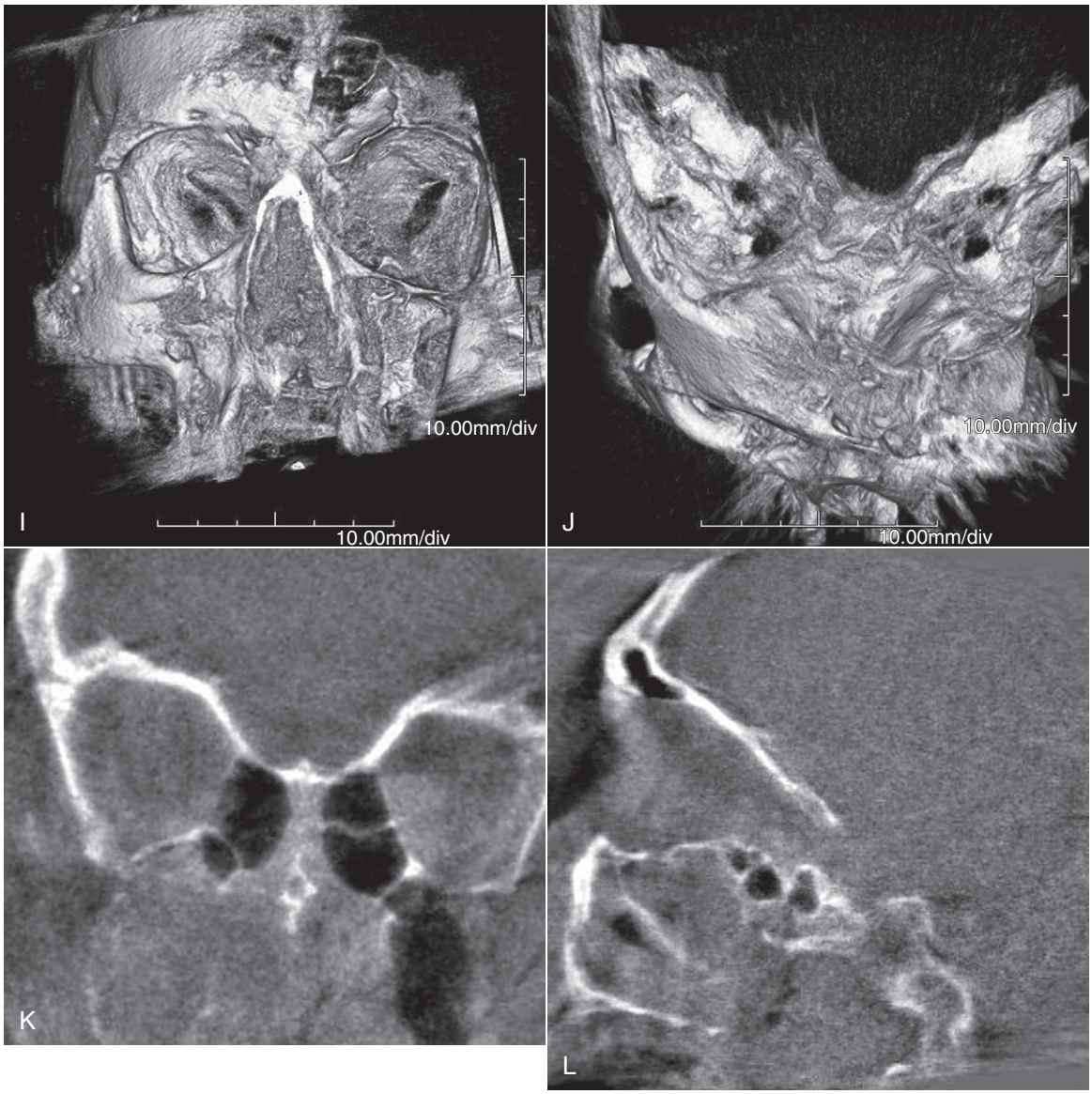


FIGURE 16-16, cont'd **I, J,** Intraoperative three-dimensional reconstructions demonstrated good reduction of the ZMC. Coronal **(K)** and sagittal **(L)** images of the orbit indicated that the orbital floor was in good position, so no internal orbital reconstruction was deemed necessary.

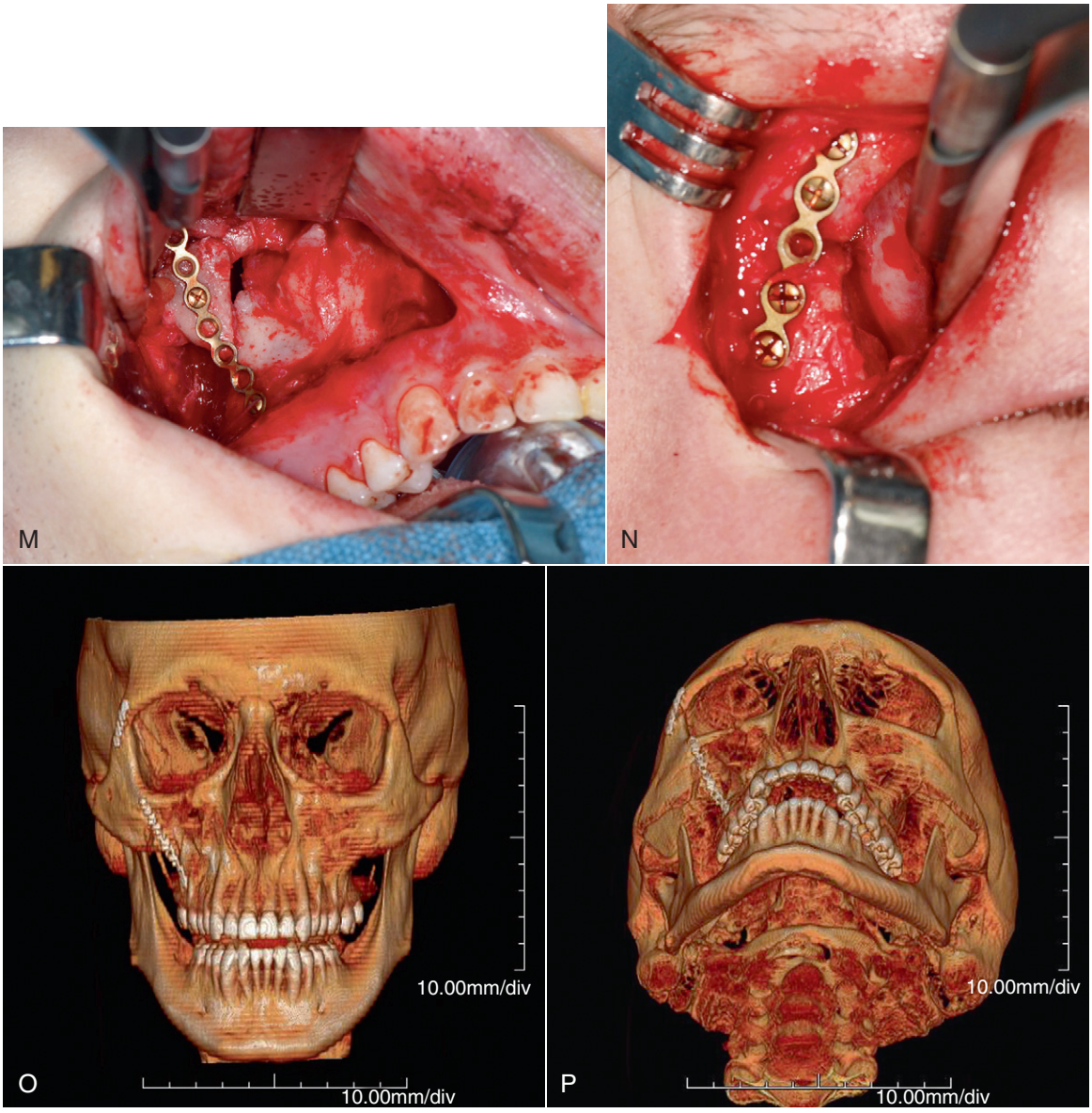


FIGURE 16-16, cont'd The ZMC was stabilized with bone plates across the zygomaticomaxillary buttress (**M**) and frontozyomatic suture areas (**N**) and the incisions closed. **O, P, Q,** Postoperative CT scans demonstrate good position of the ZMC and orbital floor (**R,S,T**).

continued

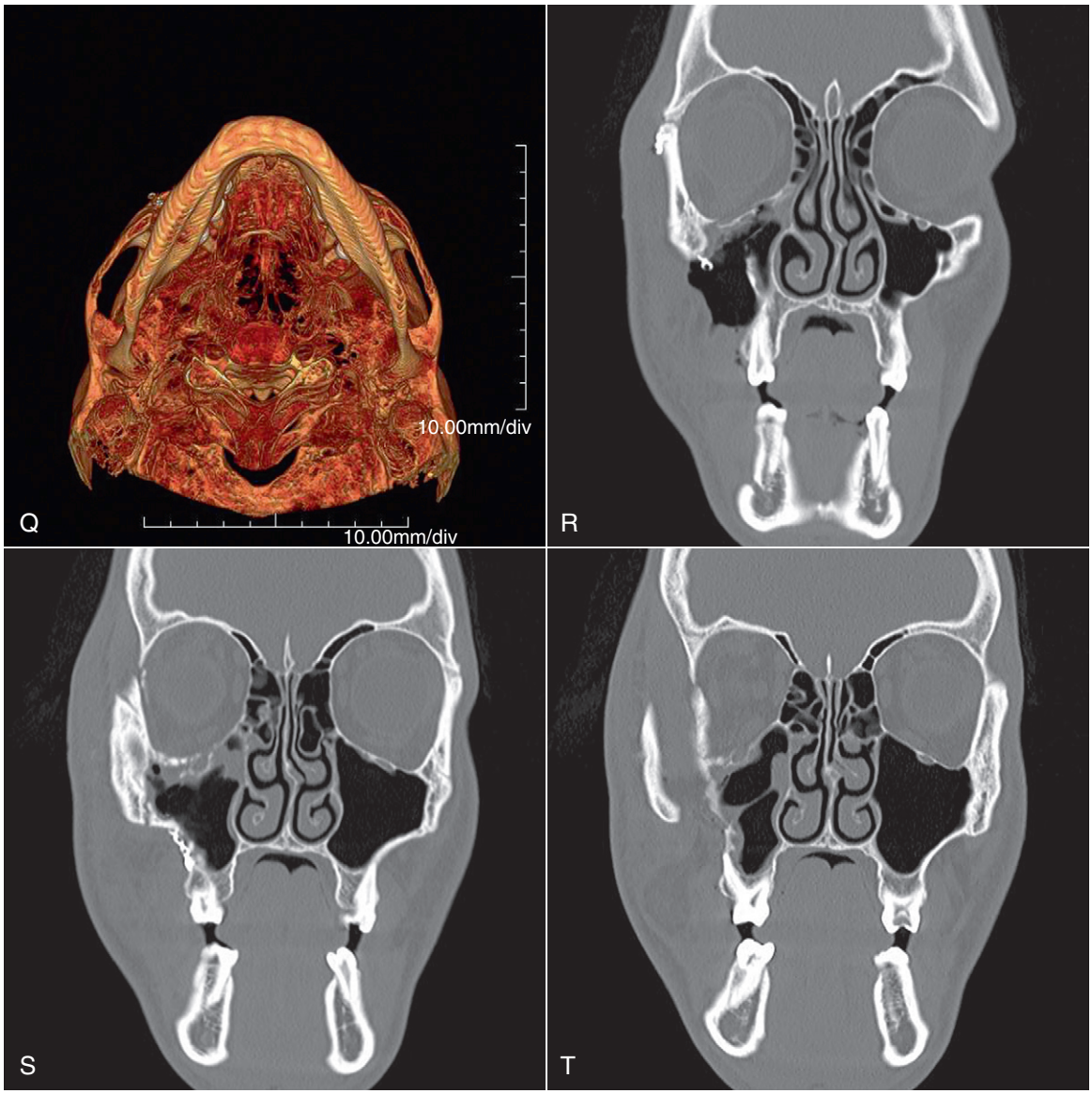


FIGURE 16-16, cont'd

of the mouth, and both sides of the preauricular area. Such preparation allows comparison of the affected side with the opposite side during surgery. Another useful suggestion is always to prepare the mouth with throat pack and antiseptic rinse, because an oral approach to the sinus and/or zygoma is frequently useful. If the preoperative clinical and radiographic examinations suggest that a coronal approach may be necessary, the hair and ears are prepared and draped.

Reduction of the Fracture. The fracture should be reduced by whatever means the surgeon deems appropriate (techniques described later).

Assessment of Reduction. The most important step in the management of ZMC fractures is to determine at the table whether the fracture has been properly reduced. The success or failure of reduction will be obvious for those who have opened the fracture at three sites. If exposure at three sites has not been performed, the orbital margins are the areas that should be palpated first

BOX 16-1 Steps in Surgically Treating a Zygomaticomaxillary Complex Fracture

1. Prophylactic antibiotics
2. Anesthesia
3. Clinical examination and forced duction test
4. Protection of the globe
5. Antiseptic preparation
6. Reduction of the fracture
7. Assessment of reduction
8. Determination of necessity for fixation
9. Application of fixation device
10. Internal orbital reconstruction
11. Assessment of ocular motility
12. Bone graft extraorbital osseous defects
13. Soft tissue resuspension
14. Postsurgical ocular examinations
15. Postsurgical images

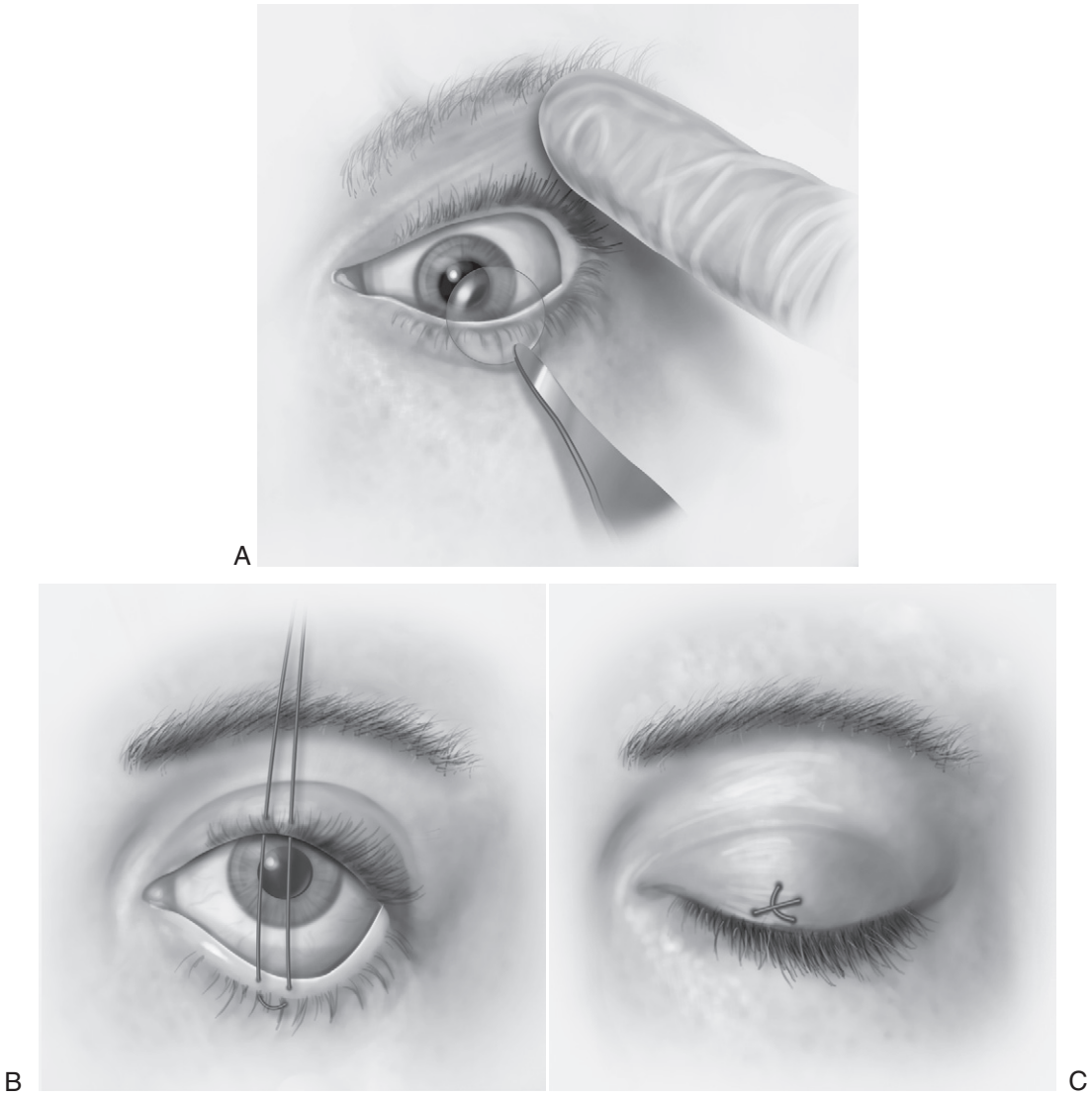


FIGURE 16-17 **A,** Scleral shell used for ocular protection. **B, C,** Technique of temporary tarsorrhaphy.

to determine reduction. If reduction has been satisfactory, these margins will be smooth and continuous. This finding by itself, however, is inadequate verification that the zygoma is properly positioned. Although the zygomaticofrontal suture area provides the strongest pillar of the zygoma, it is one of the worst indicators of proper reduction of the entire complex, even when surgically exposed and evaluated directly. One should also palpate in the maxillary vestibule. If there is any flatness still visible, the zygoma has not been properly elevated. If there is any doubt about proper reduction, exposure is mandatory. In this case, an incision in the maxillary vestibule offers excellent exposure of the zygomaticomaxillary buttress and the infraorbital rim.

For surgeons who have navigation or intraoperative CT scanning available, assessment of the reduction is relatively easy.**Determination of the Necessity for Fixation.** The second most important step in surgically treating zygomatic fractures (following determination of whether the reduction has been satisfactory) is determining whether the reduction will be stable by itself or needs some form of fixation. If constant reduction force is necessary for maintaining ZMC position, the ZMC should be stabilized with some form of fixation device(s). If the zygomatic position is deemed appropriate and does not require constant application of reduction force, one should press with moderate pressure on the malar eminence with the fingers and see whether displacement results. If it does not, fixation devices may be unnecessary. Many minimally displaced cases are stable after they have been reduced. However, if there is any doubt about postreduction stability, the application of fixation devices is prudent.

Application of a Fixation Device. The methods of stabilizing the fractured ZMC vary with the imagination and experience of the surgeon. General principles are involved, however (see later).

Internal Orbital Reconstruction. When indicated, reconstruction should be carried out after repositioning and stabilizing the ZMC fracture. In such cases, the orbital floor and walls should be exposed before elevation of the

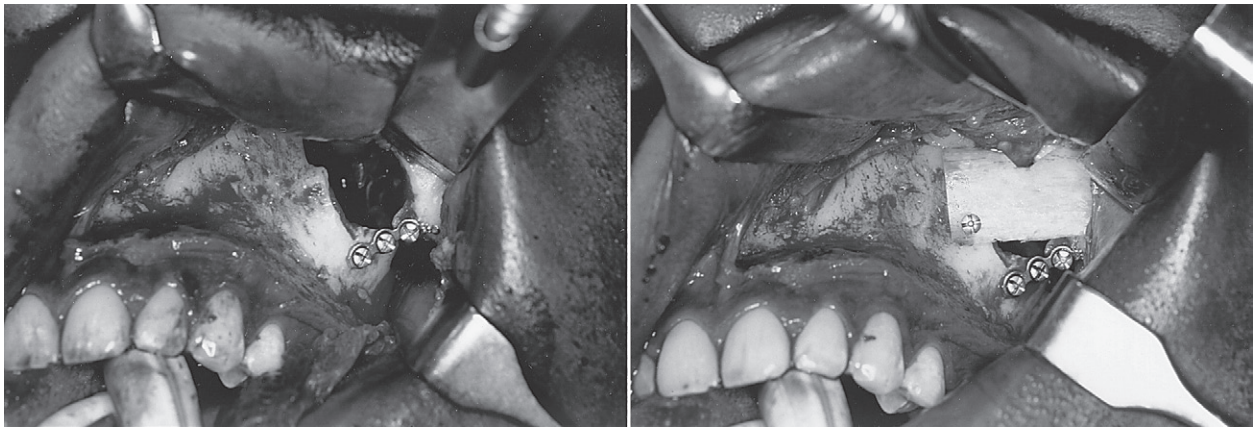


FIGURE 16-18 **A,** Anterior maxillary wall defect after reduction and fixation of a ZMC fracture. **B,** Bone graft placed over the area of defect.

ZMC so that the open orbital rim can also serve as a guide to reduction. However, it is unwise at this point to try to free any trapped tissue, because elevation of the zygoma may separate bone fragments and make this maneuver much easier following reduction. Assessment of the magnitude of the defect to be reconstructed is made following reduction, because the actual defect will then be revealed (techniques described later).

In minimally displaced cases in which no ocular signs of entrapment or enophthalmos are noted preoperatively, and in which the fracture is treated by simple reduction, internal orbital exploration and/or reconstruction is unnecessary unless a postreduction forced duction test produces positive findings (rare). In most of these cases, reduction of the zygoma results in adequate alignment of the orbital floor.[33,114,118] However, one should never avoid reconstructing the internal orbit for fear of causing harm to orbital tissue. This occurrence is extremely rare. For those surgeons who have intraoperative CT scanning capability, the status of the internal orbit after reduction of the ZMC is known and the decision about the need for internal orbital reconstruction can be made during the surgery.

Assessment of Ocular Motility. Another forced duction test should be performed at the end of all active treatment, with the possible exception of suturing, to verify that the treatment did not create entrapment of orbital contents (see Fig. 16-8).

Bone Graft for Extraorbital Osseous Defects. Consideration should be given to grafting areas of missing bone along the anterior maxilla and zygomaticomaxillary buttress. Even though bone plate fixation may provide stabilization of the ZMC by spanning such defects, it is unclear how long bone plates will provide such stability. Reconstruction of the skeleton with bone grafts prevents soft tissue prolapse from the cheek into the maxillary sinus and promotes osseous union across the defect, providing long-term stability (Fig. 16-18).

Soft Tissue Resuspension. In 1991, Phillips et al[121] described a method of soft tissue suspension of infraorbital and malar soft tissues before closing incisions after treating midfacial fractures. They hypothesized that these soft tissues droop if not resuspended; the drooping results in facial asymmetry and provides traction on the lower eyelid, causing ectropion. Yaremchuk and Kim[122] have confirmed this hypothesis and found a 20% incidence of scleral show when the facial soft tissue was not resuspended but no scleral show when the tissue was resuspended. Thus, for fractures in which the soft tissue was completely stripped from the bone, sutures should be passed through the deep surface of the soft tissue of the cheek and secured to structures such as the orbital rim and temporal fascia to raise them into their proper location on the underlying bone (Fig. 16-19; see also Fig. 16-25*F*).

Postsurgical Ocular Examination. The pupillary reflexes should be monitored postoperatively and the fundus examined periodically. Visual acuity must also be checked. Because of surgical edema, binocular diplopia will probably be present, depending on the surgical procedure.

Postsurgical Images. Postoperative images should be obtained whenever the patient is stable. Axial and coronal CT scans are recommended to assess adequacy of reduction and internal orbital reconstruction, if performed.

SURGICAL APPROACHES TO ZYGOMATICOMAXILLARY COMPLEX FRACTURES

Many techniques have been advocated for reducing and stabilizing ZMC fractures. These approaches will be described after a discussion of the surgical approaches used to gain access to the ZMC. Techniques of orbital exploration and reconstruction will then be presented.

A standard series of approaches has been used extensively for approaching the fractured ZMC and orbit. Existing lacerations are often used for this purpose. In the absence of lacerations, properly placed incisions offer excellent access, with minimal morbidity and scarring.

Protection of the cornea during operative procedures is mandatory in all operations in the vicinity of the orbit. If one is operating on the dermal side of the eyelids to approach the orbital rim and/or orbital floor, a temporary tarsorrhaphy (see Fig. 16-17*B* and *C*) or scleral shell (see Fig. 16-17*A*) may be used after application of a bland



FIGURE 16-19 **A,** Appearance of malar soft tissue before resuspension. Note the 3-0 polyglycolic acid suture, which enters through the subciliary incision and passes through the periosteum and malar soft tissue. It is shown without being tied. **B,** Appearance of the malar soft tissue after the suture has been pulled superiorly. Note the elevation of the malar soft tissue mass and the support provided to the lower eyelid. This suture can be tied to one of the screws in a bone plate on the lateral orbital rim, through a hole through the orbital rim, or through the temporal fascia.

eye ointment.[123] These are simply removed at the completion of the operation.

Diluted epinephrine solutions are used before incision for two reasons. The first is the hemostasis that they provide. The second is to separate the tissue before incision intentionally. This latter use becomes important when one operates on the thin eyelids. The solutions can be used to cause the tissue to balloon out, facilitating incision. One must remember, however, to mark the line of incision before injecting the solution into the eyelids, because the tissue will be distorted and a perceptible crease may disappear following injection.

Maxillary Vestibular Approach

The maxillary vestibular is one of the most useful approaches for open treatment of ZMC fractures. Access to the entire facial surface of the midfacial skeleton—from the zygomatic arch to the infraorbital rim to the frontal process of the maxilla—can be achieved in a relatively safe manner through this approach. Its greatest advantage is the hidden intraoral scar that results. This approach is also relatively rapid and simple, and complications are few.

Technique. The length of the incision and amount of subperiosteal dissection depend on the area of interest and extent of surgery. If one is interested in only half of the midface—for example, with a unilateral ZMC fracture—the incision can be made on one side only, leaving the other side intact. Submucosal injection of a vasoconstrictor can reduce the amount of hemorrhage during incision and dissection. The incision is usually placed approximately 3 to 5 mm superior to the mucogingival junction. The incision extends as far posteriorly as necessary to provide exposure, usually to the first molar tooth, and traverses mucosa, submucosa, facial muscles, and periosteum. Periosteal elevators are used to elevate the tissue in the subperiosteal plane. Almost no anatomic hazards exist except the infraorbital neurovascular bundle above and the posterosuperior alveolar vessels along the posterior maxilla, which infrequently cause bleeding. The entire anterior face of the zygoma can be easily exposed. Fractures through the infraorbital rim, anterior maxilla, and zygomaticomaxillary buttress can easily be identified and treated (see Fig. 16-18).

Restitution of the nasolabial muscles should be performed as three uniform steps during closure of the maxillary vestibular incision. The first step involves identification and resetting of the alar bases, the second involves eversion of the tubercle and vermilion, and the third involves closure of the mucosa. To help control the width of the alar base, an alar cinch suture is placed before suturing the lip. A V-Y advancement closure of the

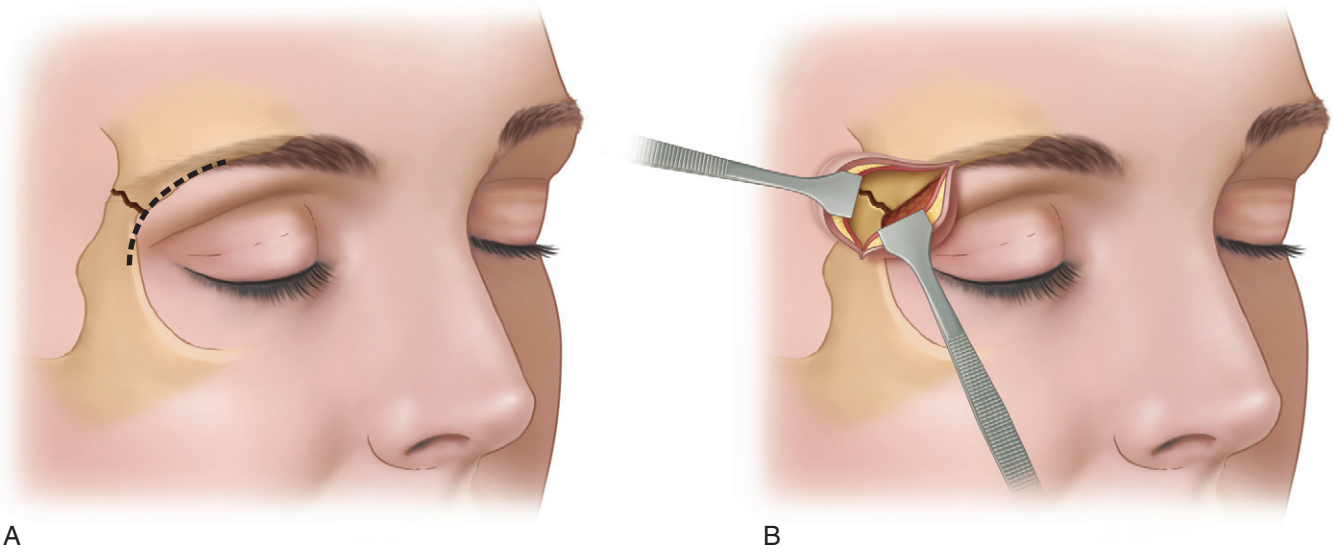


FIGURE 16-20 Supraorbital eyebrow approach to frontozygomatic suture. **A,** Most of incision is within the confines of the eyebrow. **B,** Exposure of the fracture.

maxillary vestibular incision is recommended where the incision has been placed across the base of the nose and subperiosteal dissection of the tissue along the piriform aperture has occurred. When closing the horizontal incision, one should begin in the posterior and work anteriorly with running resorbable sutures (3-0 chromic catgut) through the mucosa, submucosa, musculature, and periosteum. The superior aspect of the incision is gradually advanced toward the midline by passing the needle anteriorly in the lower margin of the incision as compared with the upper margin. This maneuver, in addition to the V-Y closure, helps lengthen the relaxed musculature so that it reattaches in its proper position.

Supraorbital Eyebrow Approach

A popular approach used to gain access to the lateral orbital rim is the eyebrow incision (Fig. 16-20). No important neurovascular structures of any significance are at risk when this approach is used, and it provides simple and rapid access to the frontozygomatic area. Because the incision is made almost entirely within the confines of the eyebrow, the scar is usually imperceptible. However, the scar will not be hidden in those who have no eyebrows extending laterally along the orbital margin. In this case, another incision is indicated. An additional disadvantage of this approach is that it does not afford a great amount of surgical access.

Technique. Before incising the skin, the surgeon should palpate the lateral orbital rim to reveal the location of the fracture site. It is usually in the frontozygomatic suture area, which is at the interolateral aspect of the eyebrow. However, the fracture may be more inferiorly positioned, and in that case the incision may need to be placed a given amount below the eyebrow.

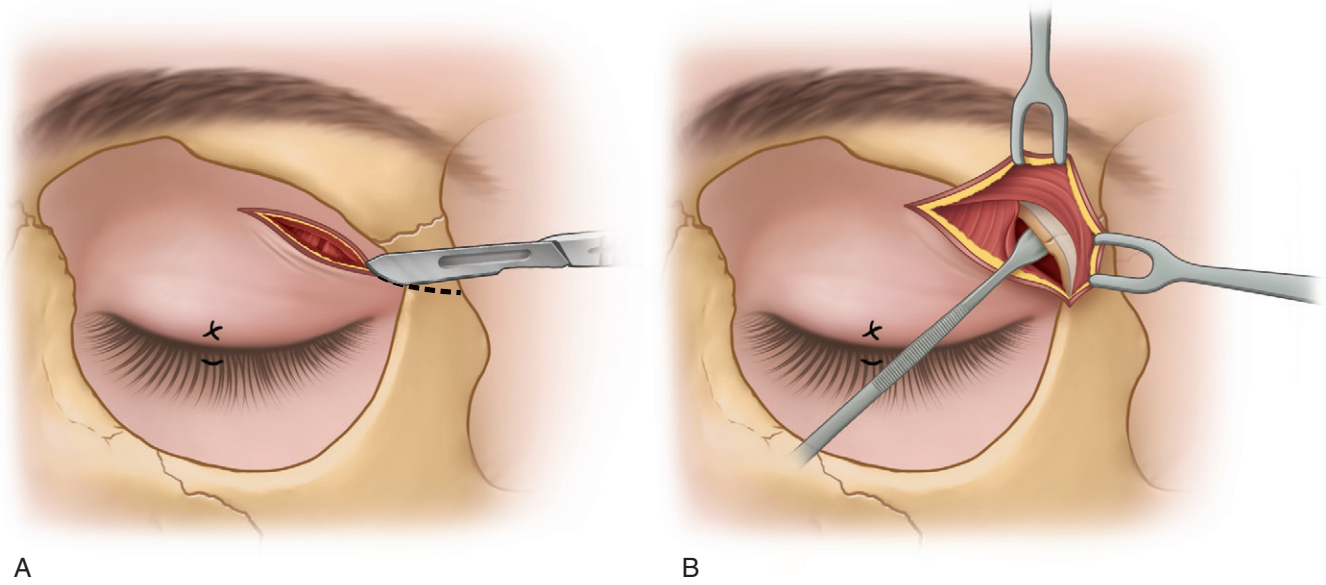
The surgeon supports the skin over the orbital rim using two fingers and a 2-cm incision is made. It should be stressed that there is no reason to shave the eyebrow before incision because the hair may not grow back. The incision should be parallel to the hair of the eyebrow to prevent cutting hair shafts, which also may retard the growth of eyebrow hair. The incision is made to the depth of the periosteum in one stroke and, after minimal undermining, another incision through the periosteum completes the sharp dissection.

Two sharp periosteal elevators are used to expose the lateral orbital rim on the lateral, medial (intraorbital), and posterior (temporal) surfaces. The fracture is usually located at the inferior extent of the wound; this location necessitates wide undermining of the periosteum to allow the tissue to be retracted inferiorly to provide better access to the fracture (see Fig. 16-20*B*). It should be noted that if one stays in the subperiosteal space, there is almost no chance of damaging vital structures. The incision is closed in two layers, the periosteum and skin.

Upper Eyelid Approach

The upper eyelid approach to the superolateral orbital rim is also called the upper blepharoplasty, upper eyelid crease, and supratarsal fold approach. In this approach, a natural skin crease in the upper eyelid is used to make the incision (Fig. 16-21*A*). The advantage to this approach is the inconspicuous scar it creates, rendering it one of the best approaches to the region of the superolateral orbital complex.

Technique. If the tissue is edematous, the skin surrounding the opposite orbit can be used to obtain an appreciation of the direction of the creases. If a lid crease is not readily detectable, a curvilinear incision along the area of the supratarsal fold that trails off laterally over the lateral orbital rim works well. The incision should be similar in location and shape to the superior incision in a blepharoplasty. However, the incision may be extended farther laterally as necessary for surgical access. The incision should begin at least 10 mm superior to the upper lid margin and be 6 mm above the lateral canthus as it extends laterally. The incision is through both the skin and orbicularis oculi muscle. The surgeon develops a



A B

FIGURE 16-21 Supratarsal fold approach to the lateral orbital rim. **A,** Location of incision. **B,** Dissection into the medial orbit.

skin-muscle flap superiorly, laterally and, if necessary, medially, using scissor dissection in a plane deep to the orbicularis oculi muscle. The dissection is carried over the orbital rim, exposing periosteum. The skin-muscle flap is retracted until the area of interest is exposed. The periosteum is divided 2 to 3 mm posterior to the orbital rim with a scalpel. Periosteal elevators are used to perform subperiosteal dissection of the orbit and orbital rims (see Fig. 16-21*B*). The wound is closed in two layers, periosteum and then skin and muscle.

Lower Eyelid Approaches

Several approaches to the orbit through the skin surface of the lower eyelid have been described. They differ in the level at which the skin incision is made and level of dissection to the infraorbital rim. The subtarsal approach is one of the more frequently used approaches for access to the infraorbital rim and orbital floor. The subtarsal incision is made in a natural skin crease at or below the level of the tarsus, approximately half the distance between the lash margin and orbital rim (Fig. 16-22). It extends laterally and inferiorly, similar to the skin creases. The main advantages of the subtarsal approach are the following: (1) it is relatively easy; (2) the incision is placed in a natural skin crease so that the scar is imperceptible; and (3) it is associated with minimal complications. It has few disadvantages.

The subciliary approach, also called the infraciliary approach, or blepharoplasty, has been favored by a number of U.S. surgeons over the past 20 years. The skin incision is made approximately 2 mm inferior to the gray line of the lower eyelid, along the entire length of the lid (see Fig. 16-22). The incision may be extended laterally approximately 1 to 1.5 cm in a natural crease inferior to the lateral canthal ligament. The main advantage to this incision is the imperceptible scar that it creates. The disadvantages are the following: (1) the procedure is technically difficult for the novice; and (2) a higher risk of postoperative ectropion exists.[109,124-128]

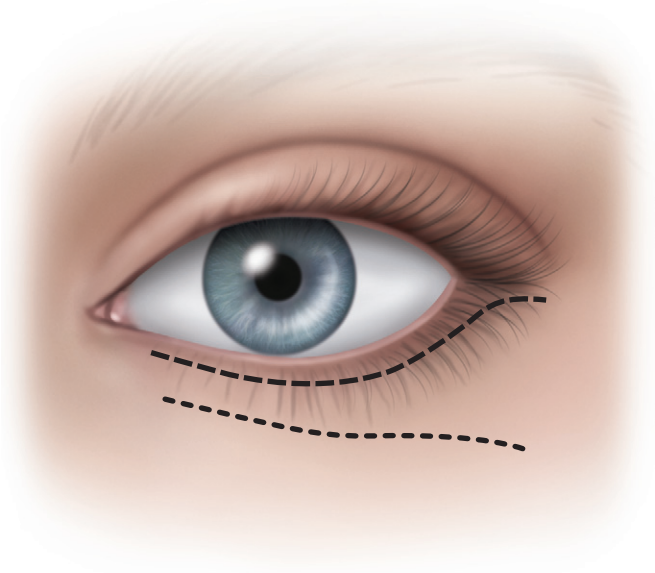


FIGURE 16-22 Incisions used to expose the infraorbital rim and orbital floor—subciliary (or blepharoplasty) incision *(dashed line)* and subtarsal incision *(dotted line)*.

Dissection Technique for the Subciliary or Subtarsal Approach. After the skin has been incised, the surgeon has three options. The first is to dissect between the skin and muscle until the orbital rim is reached, at which point another incision through muscle and periosteum to bone is made (Fig. 16-23*A*). The second option is to incise through muscle at the same level as the skin incision and dissect down just anterior to the orbital septum to the orbital rim (see Fig. 16-23*B*). The third option is a combination of these, in which subcutaneous dissection toward the rim proceeds for a few millimeters and is followed by incision through the muscle at a lower level, producing a stepped incision, with dissection then

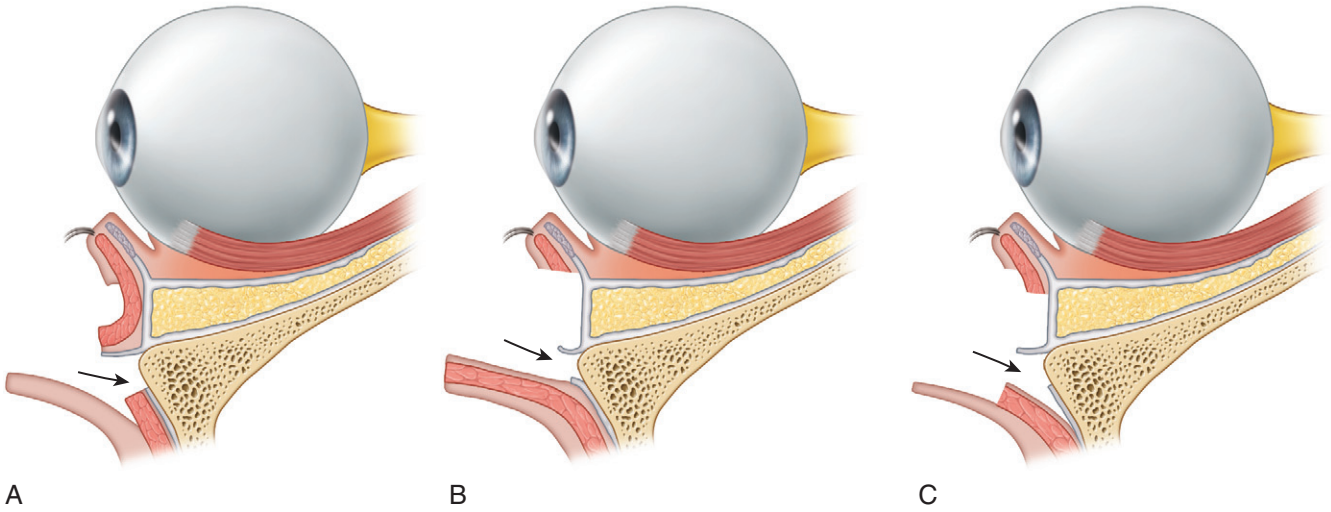


FIGURE 16-23 Cross-sectional anatomy of the dissection through the eyelid for the subtarsal (or subciliary) incision. **A,** Skin flap elevated from the orbicularis oculi muscle to just below the level of the infraorbital rim. An incision through the orbicularis oculi muscle and periosteum is then made. **B,** Skin-muscle flap dissected from the orbital septum to just below the level of the infraorbital rim, where an incision is then made through the periosteum. **C,** Three to 4 mm of skin are undermined before dissection through the orbicularis oculi muscle to the orbital septum, which is then followed inferiorly. The incision is then made through the periosteum.

following the orbital septum to the rim (see Fig. 16-23*C*).[129] Fine scissors are useful during the dissection to the infraorbital rim no matter which option is chosen, with the surgeon using a spreading motion.

Although advocated by a number of surgeons, each option has advantages and disadvantages. The first option, in which the surgeon makes a subcutaneous dissection producing a skin flap to the level of the rim, leaves an extremely thin skin flap. It is a technically difficult flap to elevate, and accidental buttonhole dehiscence can occur. A further problem that may occasionally be seen is a slight darkening of the skin in this area following healing. Presumably, the thin skin flap becomes avascular and acts essentially as a skin graft. An increase in the incidence of ectropion has also been noted, as opposed to when the dissection is made deep to the orbicularis oculi.[126] Entropion, lash problems, and skin necrosis have occasionally been experienced after the skin-only flap.[77] The second option, in which the dissection is made between muscle and orbital septum, is technically less difficult. Care must be taken, however, because the thin orbital septum can be easily violated, resulting in periorbital fat herniating into the wound. The skin and muscle flap, however, presumably maintains a better blood supply and pigmentation of the lower lid has not been a finding.[130] The third technique, in which a layered dissection is used, is probably the simplest of the three and prevents the disadvantages of the others. An added advantage of leaving a 4- to 5-mm strip of muscle attached to the lower tarsus is that if it remains functional, it may help maintain the position of the lower eyelid on the globe.

With any of these techniques, the incision through the periosteum should be placed 3 to 4 mm below the orbital rim to prevent insertion of the orbital septum along the orbital margin. Subperiosteal dissection exposes the floor and medial and lateral walls of the orbit. The subciliary incision can also be used to expose the lateral orbital rim. When the incision is used for this purpose, lateral extension of the skin incision for 0.5 to 1 cm and wide subperiosteal dissection permit the necessary access to the lateral aspect of the orbit, up to and including the frontozygomatic suture.[131] In the process of subperiosteal dissection, the lateral palpebral ligament and suspensory ligaments are stripped from the orbital tubercle of the zygoma. This stripping presents no apparent problem if the injury is acute and the periosteal tissue is securely sutured at the completion of the operative procedure. This technique is not recommended for the inexperienced surgeon because it can be fraught with difficulties in access and postoperative swelling. Properly performed, however, it is an excellent method for simultaneously exposing the infraorbital and frontozygomatic areas, and healing produces an imperceptible scar. Exposure of the lateral orbital rim via a subtarsal incision is not recommended because the lateral portion of the incision is usually some distance inferior to the orbital rim.

Closure should be in at least two layers, the periosteum and skin. Attempting to suture the orbicularis oculi is difficult and of little value. The running subcuticular suture is an excellent suture for the thin skin of the eyelid.

Transconjunctival Approach

The transconjunctival approach, also called the inferior fornix approach, was originally described by Bourguet in 1928.[132] Two basic transconjunctival incisions have since been described, the preseptal and retroseptal approaches, which vary in the relationship of the orbital septum to the path of dissection (Fig. 16-24). Tenzel and Miller[133] have developed the transconjunctival retroseptal incision and Tessier[134] elaborated on the transconjunctival preseptal incision (see Fig. 16-24*B*). The retroseptal approach is more direct than the preseptal approach and is easier

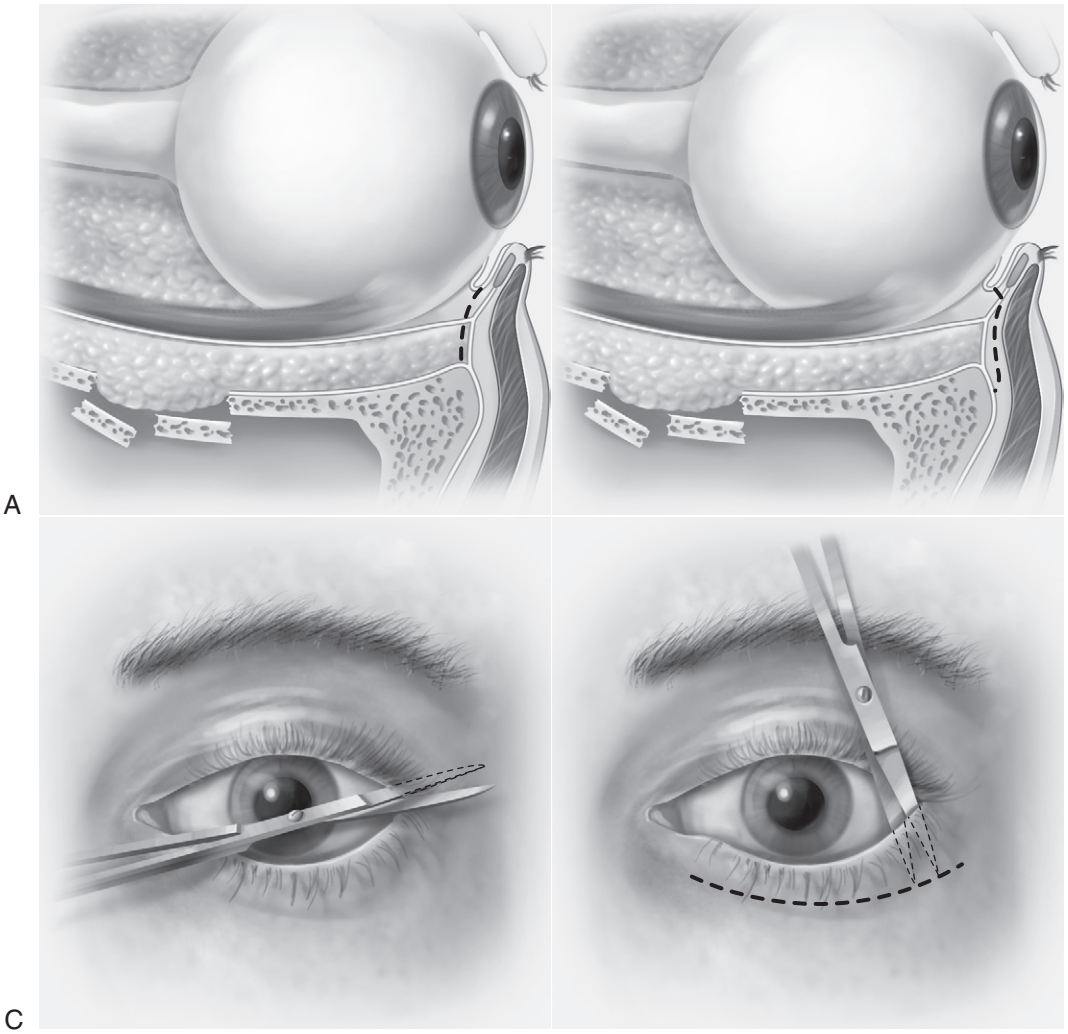


FIGURE 16-24 Transconjunctival incisions. **A,** Retroseptal approach. **B,** Preseptal approach. **C,** Initial full-thickness incision through the lateral canthus. **D,** Inferior cantholysis performed to release the lower eyelid.

continued

to perform (see Fig. 16-24*A*). Converse et al[135] have added a lateral canthotomy to the transconjunctival retroseptal incision for improved lateral exposure. The advantage of the transconjunctival approaches is that they produce superior cosmetic results when compared with any other commonly used incision because the scar is hidden behind the lower lid. Other advantages are the following: (1) these techniques are rapid; and (2) no skin or muscle dissection is necessary. In a study by Wray et al,[126] in which the transconjunctival approach was used for orbital floor and rim fractures, lateral canthotomy was necessary for improving access in 56% of approaches.

Technique for Retroseptal Transconjunctival Approach Combined With Lateral Canthotomy. In any transconjunctival approach, the cornea must be protected. Because a tarsorrhaphy is precluded, a corneal shell should be placed to protect the globe (see Fig. 16-17*A*). The lower eyelid is everted by two traction sutures placed through the tarsal plate. The surgeon performs a lateral canthotomy as the initial step by inserting one end of sharp iris scissors into the lateral palpebral fissure and cutting through in a horizontal (lateral) direction (see Fig. 16-24*C*). With eversion of the lid margin by the traction suture, the scissors are directed inferiorly to transect the inferior portion of the lateral canthal tendon (inferior cantholysis; see Fig. 16-24*D*). When transection is completed, the eyelid will fall away freely. The scissors are then used to undermine the palpebral conjunctiva just below the tarsus. The conjunctiva can be undermined medially to the lacrimal punctum. One beak of the scissors is brought out of the pocket and the conjunctiva and lower lid retractors are incised (see Fig. 16-24*E*). A suture can be passed through the incised conjunctiva in the fornix and used to retract the tissue superiorly. Blunt dissection toward the orbital rim is performed with scissors while the lower eyelid is being retracted anteriorly. With retraction of the globe and retraction of the lower eyelid, an incision is made with a scalpel through the periorbita, just posterior to the orbital rim (see Fig. 16-24*F*). A broad malleable retractor should be placed as soon as feasible to protect the globe and confine the periorbital fat. Subperiosteal dissection of the orbital

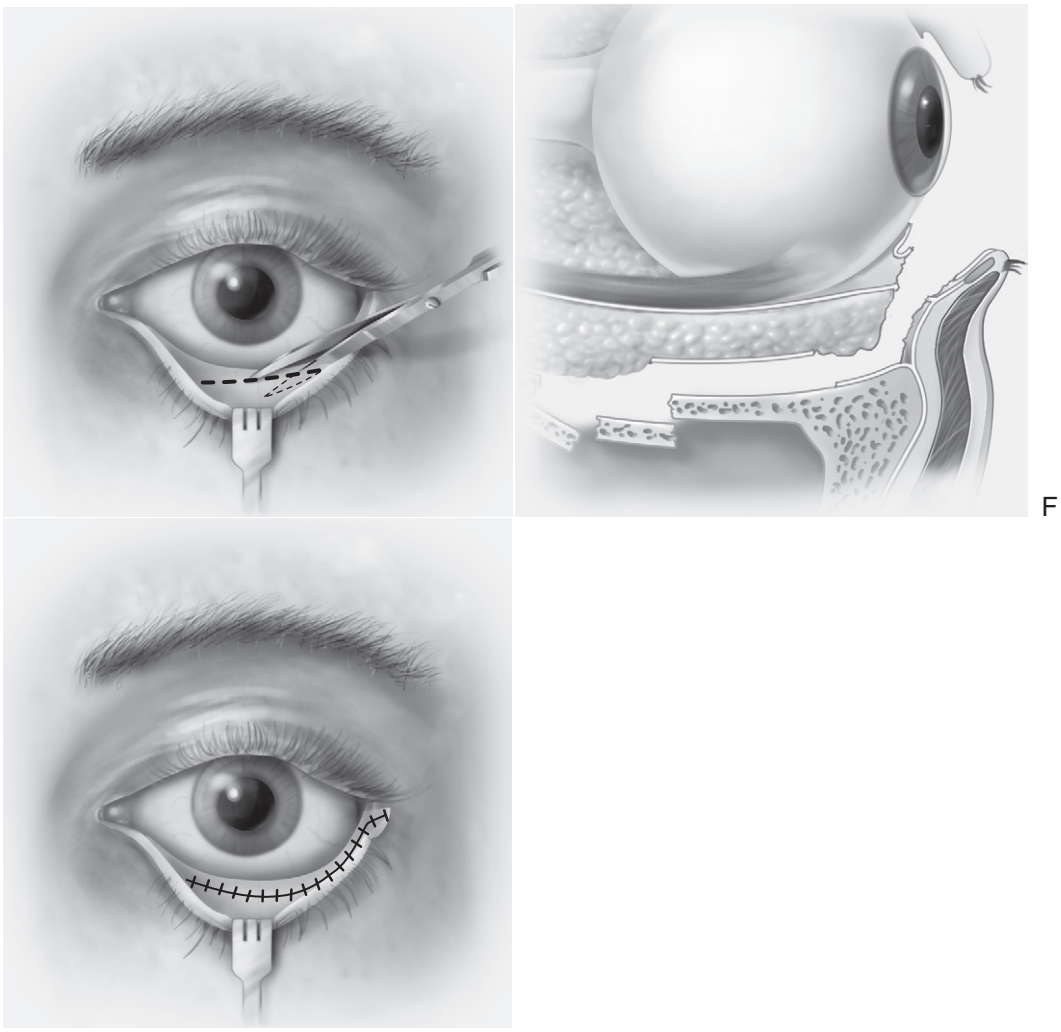


FIGURE 16-24, cont'd **E,** Conjunctival incision. **F,** Cross-sectional anatomy of the dissection. **G,** Canthotomy closure in two layers.

contents can then proceed. To facilitate retraction of the lower eyelid, the periosteum can be dissected anteriorly over the orbital rim and a few millimeters onto the face of the maxilla.

The periosteum may be difficult to close; some surgeons do not attempt closure of this layer.[136] The transconjunctival incision is closed with the use of running 6-0 gut sutures; the inferior limb of the lateral canthal tendon and tarsal plate is sutured to the inner aspect of the lateral orbital rim using 4-0 slowly resorbing or nonresorbing sutures (see Fig. 16-24*G*). Placement of these sutures is critical to adapting the lower eyelid to the globe properly. The surgeon should pass the suture along the medial side of the lateral rim of the orbit, attempting to pass it through the superior portion of the lateral canthal tendon, which is still attached to the lateral orbital tubercle. The small skin incision at the lateral canthus is closed with 6-0 sutures.

Coronal Approach

The coronal, or bifrontal, flap, modified to include some of the advantages of the modified preauricular flap of Al-Kayat and Bramley,[137] is an extremely useful incision for surgery of the zygoma and arch. Although it may initially appear as a radical approach to the management of zygomatic fractures, it provides excellent access to the orbits, zygomatic bodies, and zygomatic arches, with almost no complications.[138] It is an extremely useful incision if there is comminution of the supraorbital and lateral orbital rims, and zygomatic body and arch. The scar produced is hidden within the hairline and is therefore invisible.

Technique. In contrast with the earlier practice of extensive shaving of the head before incision, shaving the hair from the operative field is unnecessary, other than for surgical convenience. A 2-cm strip of hair can be removed in the immediate area of the incision and the adjacent hair prepped. If the hair is long, it can be tied off in clumps with sterile elastics (once prepped) to minimize the annoyance of loose hair in the operative field during the procedure. The drapes can be sutured or stapled to the scalp, covering the posterior scalp and confining this hair. For bilateral procedures, a strip across the superior aspect of the head is shaved.

In placing the incision, two factors should be borne in mind. The first is the hairline of the patient, not just the present but the anticipated future hairline. In males, minor recession of the hairline with age may make the scar visible if it is placed just behind the hairline. Therefore, the incision should be placed along a line extending from one preauricular area to the other, several centimeters behind the hairline (Fig. 16-25*A*). The incision can even be made farther posteriorly if necessary, without a significant reduction in access to the operative field. The second factor that should be considered is the amount of inferior access required for the procedure. Usually, the coronal incision may extend inferiorly to the level of the anterior border of the helix. If necessary, the coronal incision can be extended inferiorly to the level of the lobe of the ear, providing improved access at the inferior portion of the wound when necessary for zygomatic arch and infraorbital exposure.

The incision is made with a no. 10 blade through skin, subcutaneous tissue, and galea. At this point, the surgeon encounters a plane of loose areolar connective tissue overlying the pericranium (see Fig. 16-25*B*). The flap margin can be rapidly and easily lifted and dissected from the pericranium within this plane. On incision, the anterior and posterior wound margins are elevated for 1 to 2 cm to allow for the application of hemostatic clips (Raney clips), which prevent continuous bleeding from the vascular scalp throughout the procedure. Little hemorrhage should be encountered throughout the remainder of the procedure, although small vessels running through the pericranium from the skull may require cauterization. The anterior flap is elevated from the pericranium with finger dissection or the use of a blunt periosteal elevator. Along the lateral aspect of the skull, the temporal fascia becomes visible where it inserts into the pericranium, with the plane of dissection superficial to it. Once the flap has been elevated to within approximately 2 cm of the body of the zygoma and zygomatic arch, these structures can usually be seen through the covering fascia. The superficial layer of temporal fascia is incised approximately 2 cm superior to the zygomatic arch, beginning at the root of the zygomatic arch and continuing anteriorly and superiorly (see Fig. 16-25*C*). On incision of the superficial layer of temporal fascia, a layer of fat and areolar tissue is encountered (see Fig. 16-25*D*). Further dissection inferiorly at this level provides safe access to the zygomatic arch. From the root of the zygomatic arch, a periosteal incision is then made along the superior aspect of the arch and it is exposed subperiosteally (see Fig. 16-25*E*). The pericranium is now incised across the forehead and down along the lateral orbital rim. The periosteal incision at the lateral rim is connected to that over the zygomatic arch. Periosteal elevation then exposes the frontozygomatic fracture line and is continued around the lateral orbital rim into the orbit. The infraorbital rim can also be visualized to some extent with wide undermining. If access to the infraorbital area is necessary, the zygomatic arch and body should be thoroughly dissected before exposing the infraorbital areas to relax the tissue.

After fracture reduction and fixation have been accomplished, the wound is closed in layers. A lateral canthopexy is performed by drilling a hole through the lateral orbital rim just below the frontozygomatic suture for the passing of the suture. The suture can be secured to the temporal fascia or tied to the bone plate or wire in the zygoma at the frontozygomatic suture area. The periosteum over the zygomatic arch is difficult to close, and passing the suture may damage the temporal branch of the facial nerve. Instead, closure of the incised temporal fascia is performed (see Fig. 16-25*F*). The scalp incision is closed in two layers with the use of 2-0 sutures through the galea and sutures or staples on the skin surface. The use of a flat suction drain is optional. The skin sutures or staples are removed in 7 to 10 days.

REDUCTION TECHNIQUES

Temporal Approach

An approach that has been popular through the years for reduction of ZMC and zygomatic arch fractures is the temporal approach. First described by Gillies et al in 1927[1] for use in zygomatic arch fractures, this approach has proven versatility for zygomatic arch and ZMC fractures. One of its greatest advantages is that it allows the application of great amounts of controlled force to disimpact even the most difficult zygomatic fractures. It is, therefore, especially useful in late treatment of a fracture, when partial consolidation has already occurred. The Gillies temporal approach is also a quick and simple method, rarely requiring more than 15 to 20 minutes unless fixation techniques are necessary.[81] The temporal approach is associated with few complications. Although the middle temporal veins may be encountered during instrumentation,[139] the hemorrhage encountered is rarely of any consequence.

Some have noted that this technique should be reserved for zygomatic arch fractures only, being ineffective for displaced or rotated zygomatic body fractures.[36,76] An overwhelming majority of surgeons, however, disagree with this presumption and use the Gillies temporal approach as the main method for reducing zygomatic fractures.*

Technique. A 3- × 3-cm area of hair is shaved approximately 2.5 cm above and 2.5 cm anterior to the helix of the ear. It is unnecessary to isolate the area completely from adjacent hair. A cotton pellet is placed within the external auditory canal to prevent blood from entering during surgery. Frequently, the bifurcation of the superficial temporal artery is visible once the area has been shaven and serves as an excellent landmark for incision. A 2.5-cm incision is made through the skin and subcutaneous tissue at an angle running from anterosuperior to posteroinferior in the area previously shaved. This incision can usually be placed superior to the bifurcation of the superficial temporal artery, between—and thereby avoiding—both branches. The incision is carried down through skin and subcutaneous tissue until the white glistening surface of the temporalis fascia is visualized (Fig. 16-26*A*). This incision can usually be performed

*References 13, 15, 18, 19, 22, 24, 26, 28, 29, 33, 35, 81, 101, 102, 106, and 140-142.

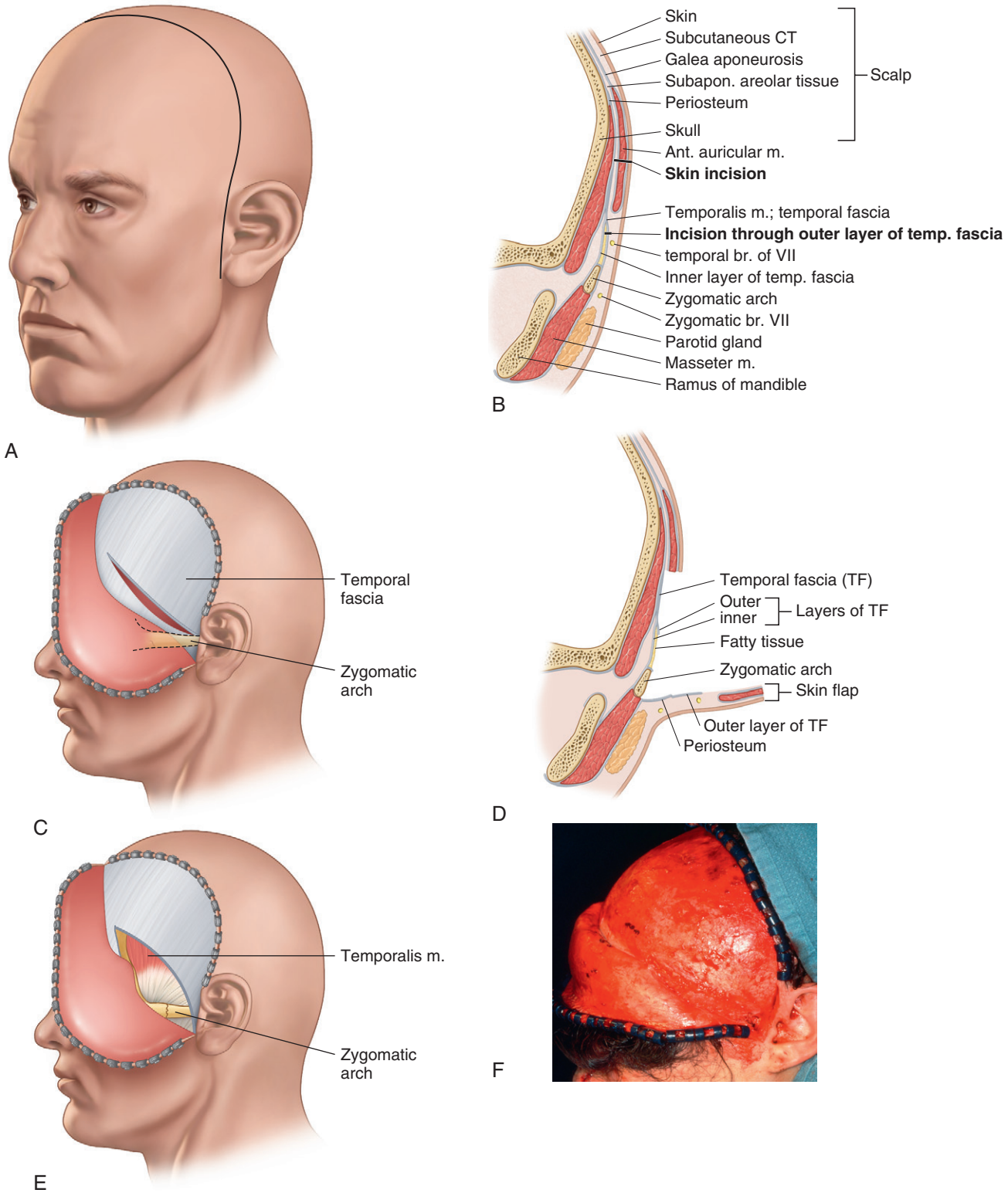


FIGURE 16-25 Coronal incision for exposure of ZMC fracture. **A,** Location of incision. The incision should be placed well behind the hairline. **B,** Anatomic layers of scalp and temporal area. **C,** Dissection of the flap anteriorly above the pericranium and temporal fascia. A second incision is made through the superficial layer of temporalis fascia and pericranium above the supraorbital rim. **D,** Anatomic layer of dissection through the superficial layer of temporalis fascia. Note that the temporal branch of the facial nerve is retracted laterally, protected by dissection in this plane. **E,** Subperiosteal dissection of the lateral orbit and zygomatic arch. **F,** Superficial layer of temporalis fascia is suspended by suturing it in a higher position than originally incised.

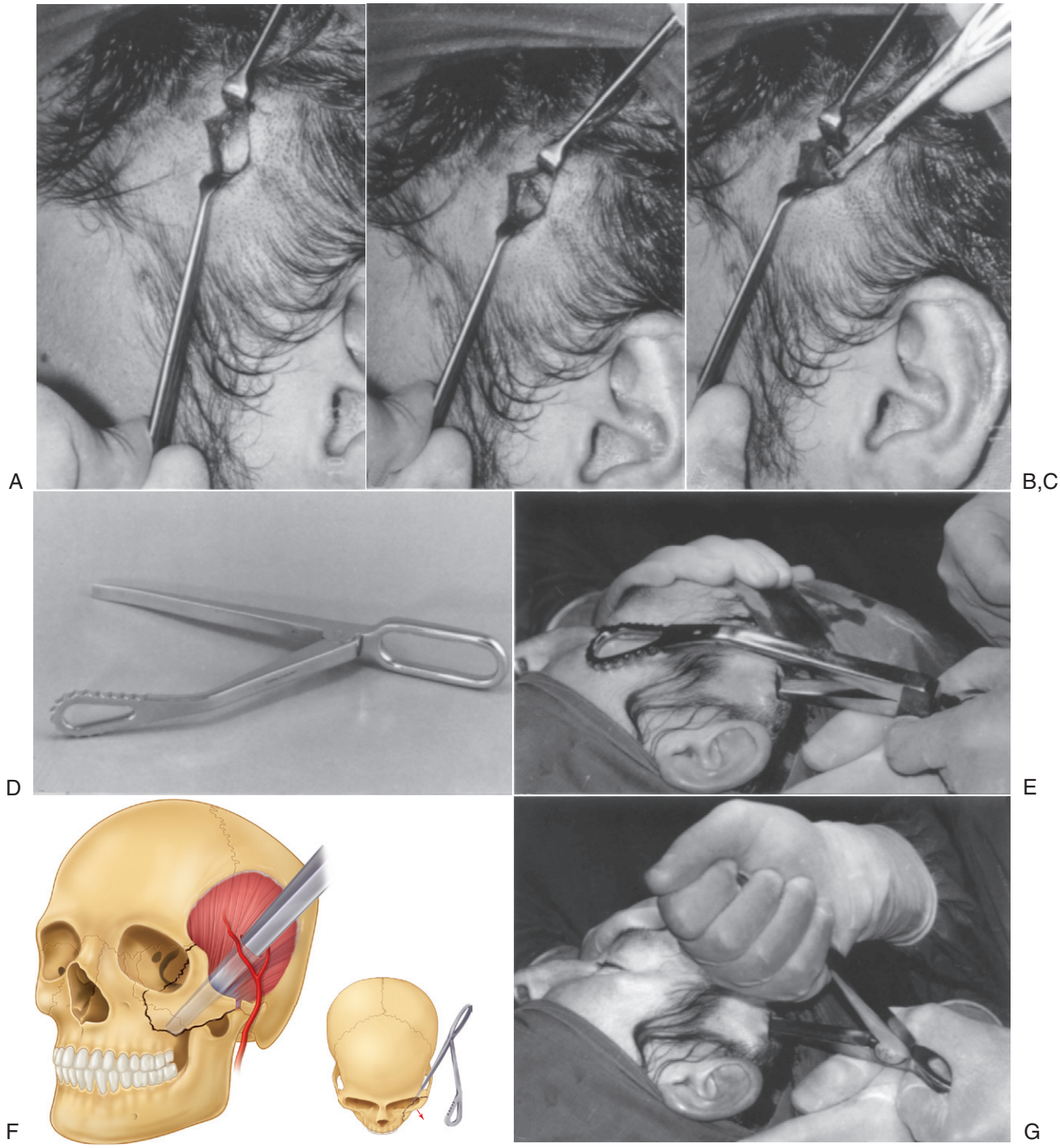


FIGURE 16-26 Gillies temporal approach to elevation of the zygoma. **A,** Initial incision to the level of the temporalis fascia. **B,** Second incision through the temporalis fascia to the underlying muscle. Note the muscle, which usually bulges out slightly through the incision. **C,** A flat periosteal elevator is inserted deep to the temporalis fascia (between the fascia and the muscle) and is swept anteriorly and posteriorly because it is advanced inferiorly. In this manner, the deep surface of the temporalis fascia is freed from the temporalis muscle. The periosteal elevator is advanced inferiorly until the medial surface of the zygomatic arch and temporal surface of the zygomatic body are identified. It is then withdrawn and the Rowe zygomatic elevator **(D)** is inserted in this same plane **(E)**. When the handle of the Rowe elevator is allowed to contact the skin, the depth of the blade beneath the zygoma can be determined. **F,** Two hands are used to elevate the zygoma. The working end of the elevator should be on the temporal surface of the zygomatic body for initial elevation. **G,** Gillies approach.

with one stroke of the scalpel. At this level, one should be above the point where the temporalis fascia splits into two layers, one attaching laterally and one medially to the zygomatic arch. It is important that the incision be above this point of bifurcation so that the elevator can be easily placed medial to the zygomatic arch. If the incision is below the layer of temporal fascia bifurcation, the elevator will be placed within the space above the arch and medial placement will be difficult.

After exposure of the temporal fascia has been completed, a second, deeper incision is carefully made the full length of the skin incision through the fascia (see Fig. 16-26*B*). At this point, one should see the underlying temporalis muscle bulge through the incision. If this is not seen, the possibility of the incision being placed too low and into the space above the arch should be suspected. In this case, the incision should be deepened until the temporal muscle is visible. Remember that the temporalis muscle is the key structure in this dissection. A flat instrument, such as a large Freer elevator or the broad end of a no. 9 periosteal elevator, is then inserted between the temporalis muscle and temporalis fascia (see Fig. 16-26, *C*). The instrument is swept back and forth as the tip is moved inferiorly until the medial aspect of the zygomatic arch and infratemporal surface of the body of the zygoma are felt. The instrument should glide freely in this plane because there is no dense attachment between the temporal muscle and temporal fascia. It may be difficult, however, to pass the instrument medially to the zygomatic arch if medial displacement has occurred, especially in areas of fracture. In this case, the tip of the instrument must be pressed medially until the medial aspect of the zygomatic arch is reached. The entire extent of the arch and zygomatic body should be palpated with the instrument to determine the location and extent of fractures. Bimanual palpation with one hand placed externally over the soft tissue of the side of the face is frequently helpful.

The periosteal elevator is removed and a flat instrument of sufficient rigidity is inserted into this same plane to reduce the fracture. Originally, a Bristow elevator was used and it was necessary to use the superior margin of the wound and adjacent skull as a fulcrum to obtain the leverage necessary for reducing the fracture. It was necessary to place gauze under the instrument at the point of fulcrum to prevent bruising the scalp. Although this instrument can still be used, it should be used with care because damage to the cranium has occurred.[143] An ingenious instrument that has since been designed for zygomatic elevation, and allows one to exert large amounts of controlled force without using the skull as a fulcrum, is the Rowe zygomatic elevator (see Fig. 16-26*D*). It has a flat blade on its working end for insertion medial to the zygomatic arch and body. It has two handles for grasping during use. The first handle is in a direct line with the working end and is used primarily for stabilization. The second handle is on the external lifting lever, which is in turn attached to the area of the stabilizing handle. When the stabilizing handle is kept in one position and the lifting handle is activated, the working blade can generate large amounts of force beneath a zygoma. The instrument was designed so that the two arms are approximately the same length. With this feature, the surgeon can be constantly aware of the depth of insertion of the working blade by collapsing the hinge between the two arms and seeing where the external handle lies in relation to the zygoma (see Fig. 16-26*E*).

Once the Rowe zygomatic elevator is in position at the proper depth, the external handle is elevated as the other handle stabilizes the working blade position. Firm anterior, superior, and lateral elevation is applied to the body of the zygoma in cases of ZMC fractures or to the arch in cases of arch fractures (see Fig. 16-26*F* and *G*). During elevation, an assistant must palpate the frontozygomatic and infraorbital areas while steadying the head against the elevator's pull. An audible crunch or crack usually accompanies the elevation. If strong resistance is felt, one must consider that the zygoma is greatly impacted, in which case more force may be necessary, or that the tip of the elevator may have been placed too far medially through the temporal muscle. In the latter case, one may be applying elevation to the coronoid process or medial aspect of the ramus of the mandible. Once the body of the zygoma has been elevated, the instrument's working blade should be swept posteriorly and laterally, reducing or ironing out any zygomatic arch fractures. The surgeon must then verify that any steps at the osseous zygomatic processes have been eliminated. Once verification of adequate reduction and resistance to displacement has been accomplished, the elevator is withdrawn and the incision is closed in one or two layers.

Buccal Sulcus Approach

Another popular technique for the reduction of zygomatic fractures is the approach through the maxillary buccal sulcus. Keen published an article on this technique in 1909[143] and it is favorably used by many surgeons today.[36,144] The major advantage, as in most intraoral approaches, is the prevention of any external scar. The buccal sulcus approach can be used for both ZMC and zygomatic arch fractures. Although the use of this approach for elevation has several laudable attributes, unstable fractures may require external incisions for the application of stable methods of fixation.

Keen Technique. A small incision (≈1 cm) is made in the mucobuccal fold, just beneath the zygomatic buttress of the maxilla. The incision can be made from anterior to posterior or from medial to lateral and should extend through mucosa, submucosa, and any buccinator muscle fibers. The sharp end of a no. 9 periosteal elevator or curved Freer elevator is inserted into the incision. Using a side to side sweeping motion, the surgeon makes contact with the infratemporal surface of the maxilla, zygoma, and zygomatic arch and dissects the soft tissue in a supraperiosteal manner. A heavier instrument can then be inserted behind the infratemporal surface of the zygoma and, using superior, lateral, and anterior force, the surgeon reduces the bone (Fig. 16-27*A*). The use of one hand over the side of the face to assist in the reduction procedure is extremely helpful. One should take care to avoid using the anterior maxilla as a point of fulcrum.

Several different instruments can be used to accomplish this maneuver, including those designed specifically

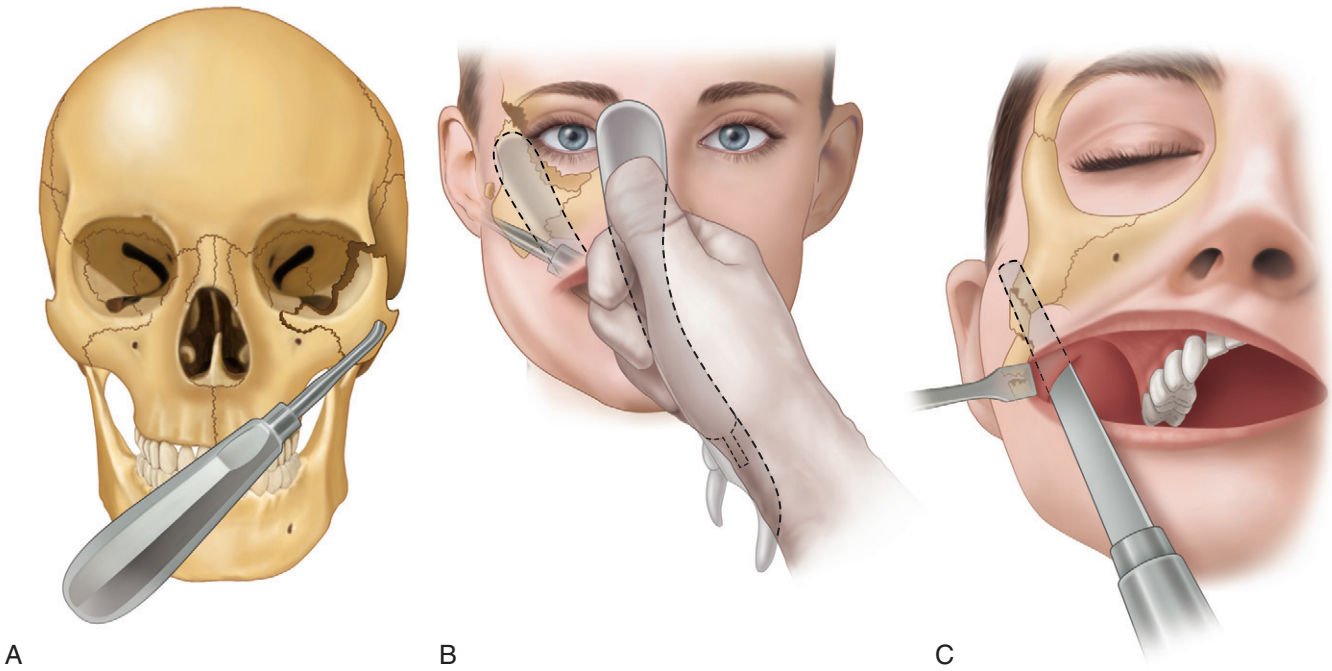


FIGURE 16-27 Intraoral approach to reduction of the ZMC and arch. **A,** Suitable elevator inserted on the temporal surface of the zygomatic body for elevation. **B,** Dental extraction forceps used in a manner similar to that for the Rowe zygomatic elevator. **C,** Flat instrument used to reduce a depressed zygomatic arch.

for this purpose, such as the Monks or Cushing (joker) elevator. However, any suitable instrument of sufficient rigidity with a bend on the end to engage the infratemporal surface of the zygoma can be used. A right angle retractor, bone hook, large Kelly hemostat, or urethral sound are satisfactory instruments for this purpose. Another instrument that can be used successfully through the buccal sulcus approach is a simple dental extraction forceps (see Fig. 16-27*B*). It is used in a manner similar to a Rowe zygomatic elevator in that the hinge portion of the forceps is the stabilizing handle and one of the forceps handles is the elevating handle. The other forceps handle becomes the working end and engages the posterior aspect of the zygoma. Controlled force can be easily applied in this manner.

A flat instrument, such as a Seldin retractor, can then be used to follow the medial surface of the zygomatic arch and elevate it laterally, if necessary. This same approach is used on isolated zygomatic arch fractures. It must be stressed that when the temporal surface of the zygomatic body is followed laterally, one must stay close to bone or the instrument may become placed on the medial side of the coronoid process. Although some clinicians think that the intraoral approach cannot be used effectively for zygomatic arch fractures,[81,145] this has not been the experience of all. The incision in the mucobuccal fold does not have to be sutured.

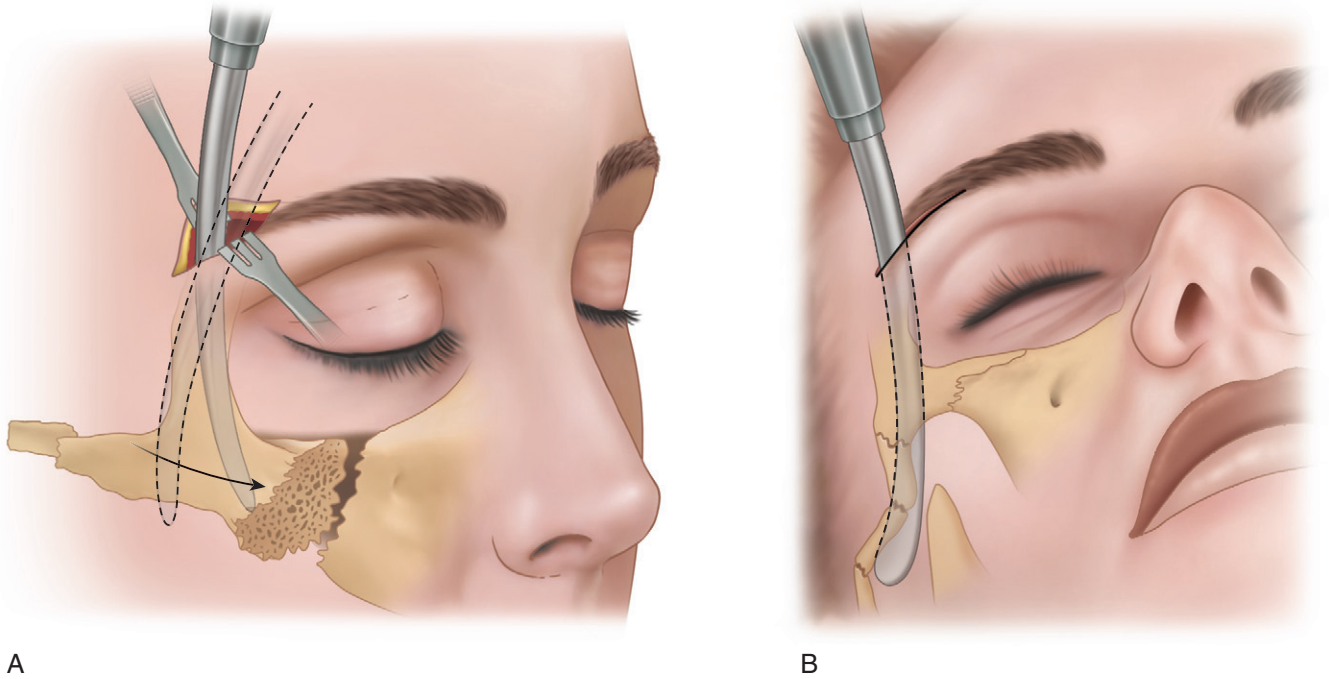
Lateral Coronoid Approach

In 1977, Quinn[146] described a lateral coronoid approach for the reduction of zygomatic arch fractures. This approach is not useful for fractures of the ZMC but is a simple method for isolated fractures of the arch. A 3- to 4-cm intraoral incision is made along the anterior border of the ramus through the mucosa and submucosa. The incision is not made down to the bone but to the depth at which the temporal muscle inserts on the ramus. The wound is deepened superiorly, following the lateral aspect of the temporal muscle with blunt dissection. This route of dissection will bring the instrument (or finger) between the temporal muscle and zygomatic arch, which should be readily palpable. The buccal fat pad will probably be encountered but is of no concern. A flat-bladed heavy elevator is inserted into this pocket, with the surgeon taking care to ensure its proper placement lateral to the coronoid process, and the arch is elevated while the surgeon palpates extraorally along the arch (see Fig. 16-27*C*). The wound is closed in one layer.

Elevation from Eyebrow Approach

In the United States, a popular technique for the elevation of zygomatic fractures is the eyebrow incision (described earlier).[17,81,76,147] The advantage to this technique is that the fracture at the orbital rim is visualized directly and fixation of the fracture at this point can be undertaken through the same incision, when necessary. The disadvantage is that it is difficult to generate a large amount of force, especially in the superior direction.[9,36,102]

Technique. Once exposure of the fracture at the frontozygomatic area of the lateral orbital rim has been accomplished, a heavy instrument is inserted posteriorly to the zygoma along its temporal surface. The instrument is then used to lift the zygoma anteriorly, laterally, and superiorly while one hand palpates along the infraorbital rim and body of the zygoma (Fig. 16-28*A*). Useful instruments for this purpose are the Dingman zygomatic elevator, urethral sound, or even large Kelly hemostat. The



A B

FIGURE 16-28 Elevation of ZMC from the eyebrow approach. **A,** Dingman zygomatic elevator is placed along the temporal surface of the zygoma for anterior, lateral, and superior elevation. **B,** Elevator is used to reduce the zygomatic arch fracture.

arch can also be approached from this exposure and reduced (see Fig. 16-28*B*).

Percutaneous Approach

A direct route to elevation of the depressed zygoma is through the skin surface of the face overlying the zygoma. This approach has been used extensively worldwide. The advantage to the technique is that one can produce forces anteriorly, laterally, and superiorly in a direct manner, without having to negotiate adjacent structures with the instruments. The major disadvantage is a scar on the face in a very noticeable location. However, in practice, scarring is more a theoretical than real disadvantage because the incision sites are rarely visible 2 to 3 weeks after surgery.

Technique. The percutaneous approach is probably the simplest of all techniques because no soft tissue dissection is necessary. Several instruments can be used to elevate the zygoma. The bone hook, introduced by Strohmeyer in 1844,[148] has probably been the most widely used instrument and is advocated by many (Fig. 16-29*A*).* The point of the hook is simply inserted through the soft tissue of the malar area at a point just inferior and posterior to the prominence of the zygoma so that it engages the infratemporal aspect (see Fig. 16-29*B*). Poswillo[152] draws two intersecting lines on the face to determine the proper location for application of the bone hook. The first is a vertical line dropped from the lateral canthus of the eye. The second is a horizontal line drawn laterally from the ala of the nose. A small stab incision is made at the point of intersection of these lines and the hook is inserted. The hook is then rotated to engage the temporal surface of the zygoma. One must be cognizant of the area of application of the point of the hook on the back of the zygoma, ensuring that the hook has not slipped into the inferior orbital fissure, which can cause venous hemorrhage that might result in ocular injury. Strong traction in any direction can then be applied to reduce a displaced zygoma (see Fig. 16-29*C*).

A large bone screw, such as the Carroll-Girard screw, is another instrument that has been used with some frequency for elevating zygomas (Fig. 16-30). It resembles an elongated corkscrew with a T bar handle and contains threads on its working end. This screw can be threaded into the body of the zygoma following placement of a hole and can then be used as a handle to reduce the displaced zygoma (see Fig. 16-30*B*). An advantage to its use is that one can control the ZMC position in all three planes of space.

Any of these instruments (and probably others) are helpful when the clinician uses the transcutaneous approach to the zygoma. The possible application of one or two monofilament sutures is all that is necessary to care for the wounds created by their use.

FIXATION TECHNIQUES

The application of plate and screw fixation techniques to ZMC fractures has replaced all the older techniques of fixation. There is no better method of providing stable fixation to an unstable ZMC fracture than to secure it rigidly internally with bone plates and screws. The obvious advantage to bone plates is that stabilization in three planes of space can be provided, even across areas of comminution or bone loss.

Each case must be individualized, because the fixation requirements differ greatly from one fracture to the next. Some fractures may require no fixation; others may require three or four bone plates. When plate and screw

*References 25, 103, 104, 109, 110, 149-153.

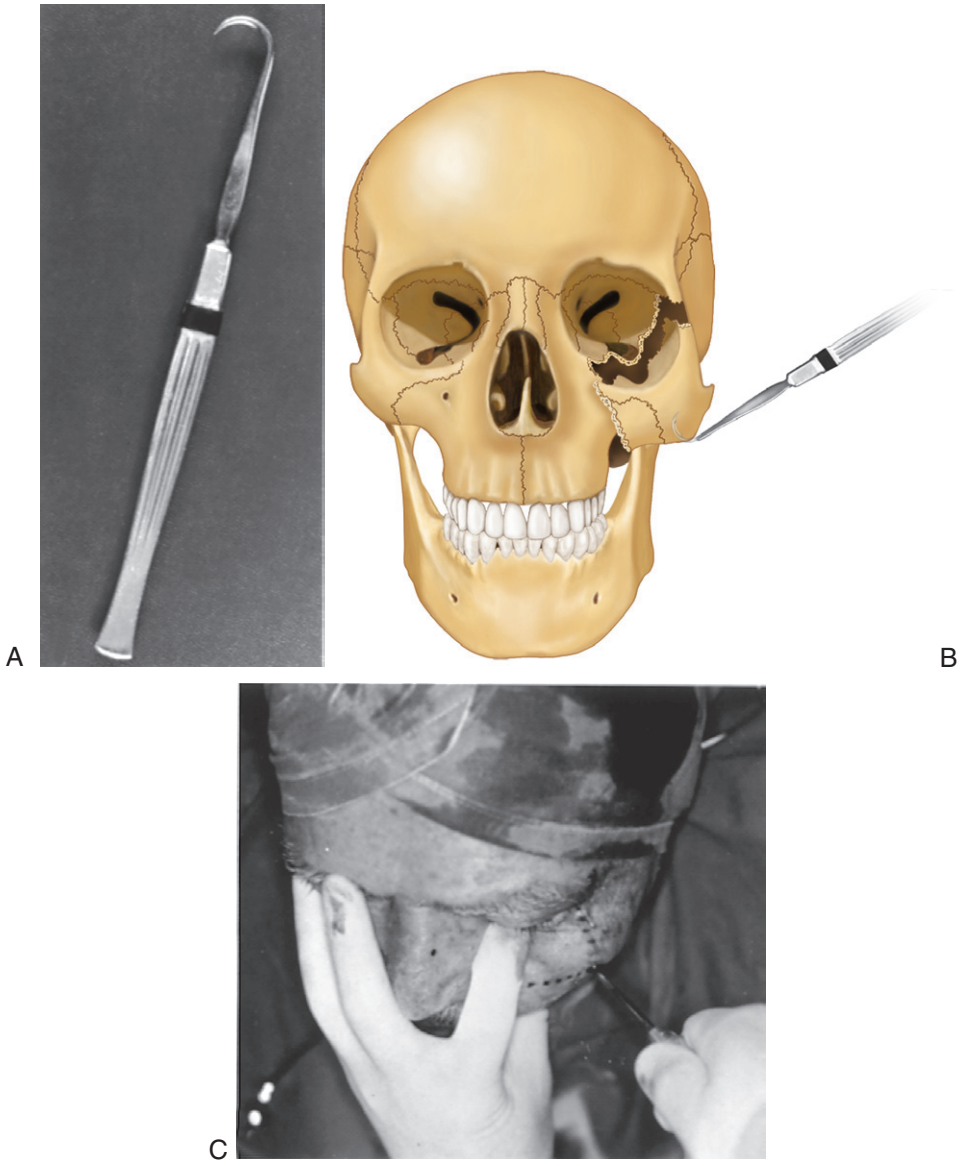


FIGURE 16-29 Elevation of the ZMC with a bone hook. **A,** Bone hook. **B,** Anterior and lateral traction with the use of a percutaneous bone hook. **C,** Clinical photograph of the use of a bone hook. The *dotted lines* on the face represent those discussed in the text. Note finger placement at the infraorbital rim during elevation to detect reduction.

fixation is used, there are general principles of its application for ZMC fractures.

1. Use self-threading bone screws. The thin bones of the midface lend themselves to the application of self-threading screws. It has been shown that self-threading screws have more holding power in thin bones than when the holes are tapped.[154-157]
2. Use hardware that will not scatter postoperative CT scans. Titanium plates and screws have the advantage of not causing scatter in CT scans. Vitallium causes more scatter, so if it is selected, smaller plates and screws should be used to minimize CT artifacts.[158-161]
3. Place at least two screws through the plate on each side of the fracture. The three-dimensional stability provided by plate and screw fixation demands that the bone plate be adequately secured to each fragment. At least two screws are necessary for stabilizing a bone plate to a bone fragment.
4. Avoid important anatomic structures. One should position bone plates so that the screws do not impale structures, such as the tooth roots and infraorbital nerve. If the fracture through the zygomaticomaxillary buttress is low, one should select an L-, a T-, or Y-shaped bone plate so that both of the lower screws are positioned horizontally in the alveolar process. The use of straight plate in this case might cause the lower screw to impale a tooth root (Fig. 16-31).
5. Use as thin a plate as possible in the periorbital areas. The skin overlying the orbital rims is very thin and becomes more so over time. If a bone

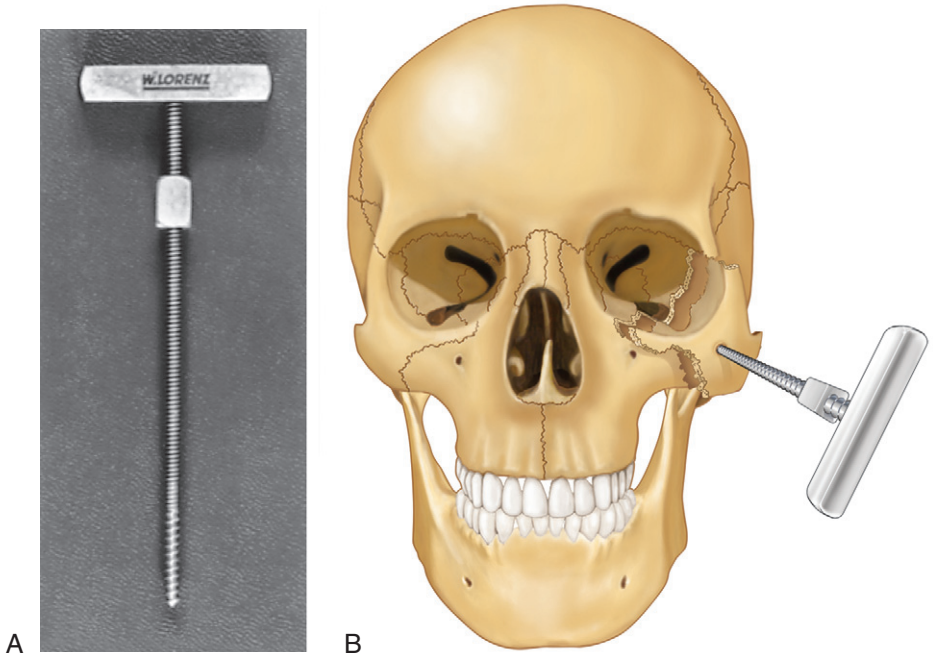


FIGURE 16-30 Elevation of the ZMC with a bone screw inserted percutaneously. **A,** Carroll-Girard bone screw. **B,** Use of a bone screw.



FIGURE 16-31 Vertical placement of two holes below the fracture at the zygomaticomaxillary buttress can jeopardize the tooth roots *(right).* In such cases, an L, T, or Y plate may avoid the tooth roots.

plate must be placed along the orbital rim, it should be extremely thin to prevent visibility and reduce palpability. This is especially true of the infraorbital rim. If possible, one may wish to avoid placing bone plates in this location unless absolutely necessary.

6. Place as many bone plates in as many locations as necessary to ensure stability. Many fractures can be adequately stabilized with a single bone plate applied at the frontozygomatic area* or at the zygomaticomaxillary buttress[95,99,100,113] (see earlier). However, when the articulations between the ZMC and adjacent bones are comminuted, it will be necessary to apply additional bone plates in additional areas. One can determine how much fixation is necessary by forcefully trying to displace the repositioned ZMC during surgery. This maneuver is especially easy if a Carroll-Girard screw has been inserted. If the ZMC is stable against the application of moderate force, it is unlikely that postreduction displacement will occur and no more fixation is necessary. If there is some mobility after the application of a bone plate, another plate may be necessary.
7. If concomitant fractures of other midfacial bones exist, it will be necessary to apply fixation devices more liberally. For example, if the maxillary alveolus, hemimaxilla, or complete maxilla is unstable, a bone plate at the zygomaticomaxillary buttress will no longer be able to provide the primary means of support for the repositioned ZMC. In such cases, primary fixation of the frontozygomatic area will be necessary. It is imperative that the dentition be placed into maxillomandibular fixation before bone plates are applied when concomitant maxillary fractures are present.
8. In areas of comminution or bone loss, span the gap with the bone plate. Comminution of fractures through the zygomaticomaxillary buttress and infraorbital rim is common. If small bone fragments are missing, it is imperative that the gap be maintained by the bone plate; otherwise, the ZMC will be malpositioned. When the gaps are more than a few millimeters, bone grafts can be attached to the bone plate or laid (and stabilized) over the bone plate to promote osseous healing across the defect.

*References 52, 88, 79, 89, 108, 109, 111, and 161.

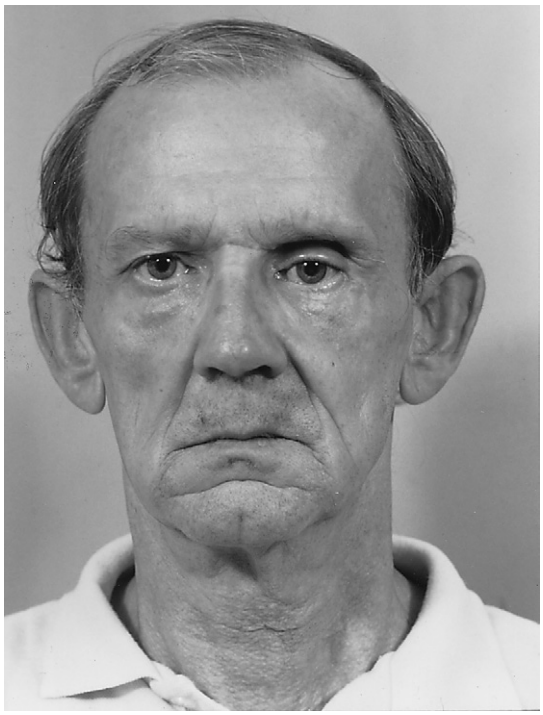


FIGURE 16-32 Patient 8 months after treatment of a left ZMC fracture. Note the deepening of the supratarsal fold, a hallmark of enophthalmos. The slightly inferior position of the globe is also evident.

INTERNAL ORBITAL RECONSTRUCTION

In the treatment of ZMC fractures, internal orbital reconstruction is a supplementary procedure that is frequently but not always indicated. When indicated, internal orbital reconstruction is a vital component of treating ZMC fractures (Fig. 16-32). The complications of ZMC fractures that are most difficult to correct secondarily are those of the orbit. When internal orbital reconstruction is not performed when indicated, or is performed inadequately (see Fig. 16-14), postsurgical enophthalmos can result.

Postsurgical enophthalmos is one of the most distressing complications after treatment of ZMC fractures. It usually results if the orbital floor and walls have not been reconstructed when indicated or have been inadequately reconstructed.[88,91,162-167] Studies have shown that posttraumatic enophthalmos is most commonly caused by an increase in the size of the bony orbit.[66,67] Lateral positioning of the ZMC is one of the most effective methods for increasing orbital volume because of the cross-sectional area of the orbit at the level of the displaced ZMC. However, concomitant fractures of the orbital floor and/or medial wall are common with ZMC fractures and can also increase orbital volume.[147]

Any patient with presurgical enophthalmos should be suspected of having orbital disruption but traumatic edema may mask the problem, making clinical examination difficult. CT has made preoperative assessment of the status of the bony orbit possible, with a great degree of accuracy. In reviewing treatment of ZMC fractures over a 10-year period, Covington et al[79] found a reduction in orbital exploration from 90% in 1985 to 30% in 1989 because of the increasing use of preoperative CT scans. Studies have shown that the CT scan allows predictable determination preoperatively of whether the orbital floor and/or walls require reconstruction.[114,118] This information permits more comprehensive preoperative planning of the surgery and avoids unnecessary orbital exploration. The availability of intraoperative CT scanning in some operating rooms will also affect the decision about when internal orbital reconstruction is necessary.

Intrasinus Approach to the Orbital Floor

The antral approach to the orbital floor historically has been predicated on the ability to realign the orbital floor without making external incisions when the orbital floor has been depressed but there is no herniation of soft tissue through the periorbita. Packing the sinus with gauze or a balloon to provide support to the orbital floor for 2 weeks was thought to allow healing to take place.[45,168-172] In practice, however, this technique is rarely possible and should not be used as the routine approach to the orbital floor.

More recently, surgeons have begun to use an endoscope for evaluating and reconstructing the orbital floor.[173-178] Approaching the orbital floor through a bone window in the lateral maxilla allows the insertion of an endoscope to examine the status of the floor and, if necessary, reconstruct it by insertion of a material, such as bone, metallic mesh, or porous polyethylene into the orbital defect.

External Approach to the Internal Orbit

Reconstruction of the orbital floor is performed through a lower eyelid approach (subciliary, subtarsal, or transconjunctival). Using any of the approaches to the infraorbital rim and internal orbit (see earlier), the surgeon gently elevates the periorbita along the floor of the orbit. It must be remembered that the orbital floor is inferior to the level of the rim, so when the periorbita is elevated, one must be careful to follow the contour of the rim or perforation of the periorbita will occur. The subperiorbital dissection should extend beyond the full length of the access incision in the skin. Comminution of the infraorbital rim is commonly present. It is always easier to dissect the periorbita from sound bone toward the fractured areas. Protection of the periorbita and globe is facilitated after dissection by placement of a malleable retractor. The area of disrupted orbit may be a narrow crack along the floor, usually along the infraorbital groove, or severely comminuted floor and walls. When comminuted, it becomes difficult to dissect the periorbita from the thin bone spicules. One must continue dissection posteriorly along the orbital floor and medial wall until sound bone is found. Thus, dissection far posterior to the globe is usually necessary. Areas of periorbital fat dehiscence through the periorbita into the maxillary or ethmoid sinuses must be gently freed. Small bone spicules can be removed if free from soft tissue attachments.

For fractures in which the defect is a narrow linear groove, no reconstruction is usually necessary. When a larger defect is noted and the periorbita has been disrupted, reconstruction of the internal orbit is necessary

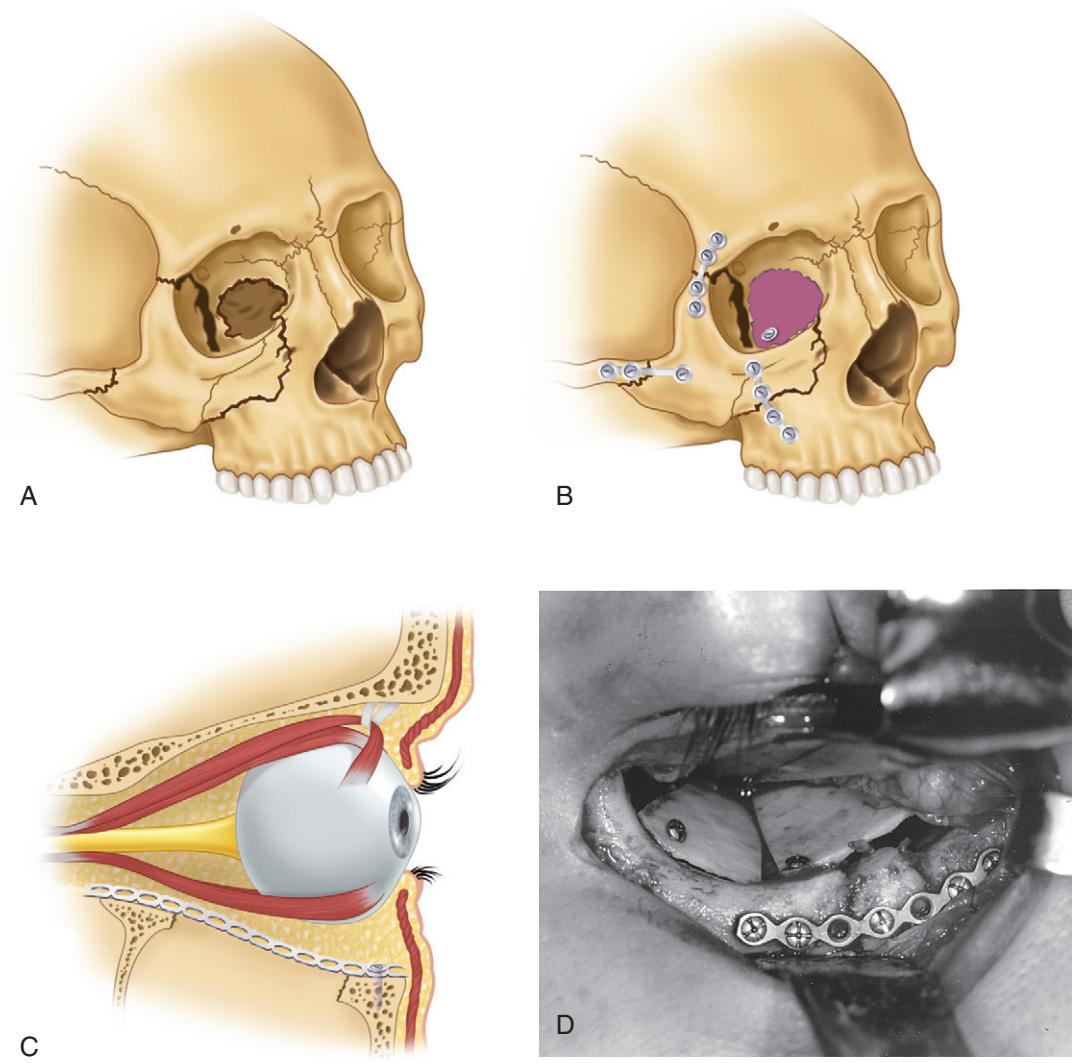


FIGURE 16-33 **A,** Right ZMC fracture with comminution of the orbital floor and medial wall. **B,** After reduction and internal fixation of the ZMC fracture, the internal orbit is reconstructed with a graft or implant that completely bridges the defect or comminuted area. **C,** Stabilization of the graft or implant is performed with a lag screw or by other means. **D,** Reconstruction of the left orbital floor and medial wall with the use of calvarial bone grafts secured with lag screws.

for preventing enophthalmos and ptosis of the globe (Fig. 16-33).

Materials Used for Orbital Reconstruction

A number of materials have been used to reconstruct the internal orbit, including autologous bone, autologous cartilage, allogeneic bone and cartilage, methyl methacrylate, silicone polymer, polyurethane, aluminum oxide ceramic, Teflon (polytetrafluoroethylene polymer), gelatin film (Gelfilm), Supramid, polyethylene, polyvinyl sponge, polydioxanone plates, polyglactin mesh or plates, polylactide plates, porous polyethylene, lyophilized dura, and metal sheets or mesh (Potter and Ellis have written a comprehensive review[179]).

Bone has been used extensively for many years with excellent results and is often chosen when the orbital defect is large. Autologous bone can be obtained from a number of donor sites. Historically, the most common source was the iliac crest. However, split ribs, the anterior surface of the opposite maxilla, and the buccal or lingual cortex of the mandible have also been used with good success. From 1980 to 2000, the calvarium became one of the primary donor sites.[180-182] When bone is used, it should be borne in mind that some resorption will eventually take place, so adequate volume should be transplanted to offset this eventuality. Although the use of allogeneic bone and cartilage is less common, it may have merit.[183] The possibility of infection from the open sinus does not seem to be a problem with bone.

Although autologous grafts may appear to be ideal, there is associated donor site morbidity and increased operative time involved in graft harvesting and carving. Furthermore, calvarial bone grafts are difficult to shape and are brittle. These factors led to the development and acceptance of alloplastic substitutes for use in orbital reconstruction. Criticism directed at the use of alloplastic materials cites the complications of infection, extrusion, and implant displacement. There have been reports of

late complications developing with implanted silicone that have necessitated its removal as long as 18 years postoperatively.[184-194] Morrison et al[195] have reviewed 311 cases of silicone implants placed over a 20-year period and found that at least 13% required removal for one reason or another. However, when used in small defects and properly stabilized, silicone, Teflon, and other alloplasts have proven useful. Porous polyethylene has become popular in recent years for orbital reconstruction. It comes in various thicknesses, is easy to bend, and maintains its shape. Another advantage is that it offers sufficient rigidity to confine the orbital contents and can be stabilized easily with plate and/or screw fixation.[196]

A major advantage to bone, cartilage, and bioresorbable products, such as gelatin film and polydioxanone or polyglactin plates, when compared with alloplastic implants, is that not only are they able to provide the necessary support to orbital tissue, but they are also incorporated or replaced in the body, minimizing the chance of late reactions. Gelfilm has been shown to undergo slow degradation over a 10-week period, with bone bridging occurring simultaneously in orbital floor defects created in adult rhesus monkeys.[197] Unfortunately, many of the biodegradable products are not stiff enough to be useful in large orbital defects.[198-202] In such cases, bone, porous polyethylene, or metallic implants should be considered.

Metallic mesh has become popular for orbital reconstruction in recent years.[203-208] Even though metallic mesh is exposed to open sinuses, it is rare to have to remove any because of infection. Advantages of the use of metallic mesh are that it can be made to conform to the desired contours, it is stiff enough to maintain adequate support of the periorbital tissue, and it is extremely thin. Also, it is readily visible on postoperative CT scans.

Principles of Orbital Implant and Transplant Placement

Because the objective of orbital reconstruction is to support the periorbital soft tissue and partition the maxillary or ethmoid sinuses from the orbit, any of the materials discussed will suffice. The decision is usually based on the availability of the products, preference of the surgeon and, most importantly, size of the defect. When the defect is large, autologous bone, porous polyethylene sheets, and metallic mesh are the materials of choice. The use of other materials, when the defect is large, necessitates a very large or thick implant, which would be difficult to stabilize along the minimal osseous margins. Alloplastic implants, such as silicone and Teflon, should be reserved for smaller defects. No matter which material is used, however, certain principles should be kept in mind.

1. The size of the implant or transplant. As large an implant or transplant as necessary for covering the entire defect should be used. The implant or transplant must be of sufficient size to be supported along most margins by sound bone. Before the placement of any implant or graft, one must be certain that its posterior edge is resting on sound bone. Perhaps the most common error in placement of an implant or transplant is leaving the posterior edge unsupported. To ensure proper placement, dissection back toward the orbital apex is necessary for establishing the posterior extent of the defect. If it is impossible to establish a sound posterior margin, the posterior edge of the material must be well supported laterally and medially. Alternatively the material can be cantilevered to adjacent sound bone with the use of plate and screw fixation.[209]
2. The thickness of the implant or transplant. The thickness of the implant or transplant is usually determined by the flexibility of the material. If flexible, a thicker piece is necessary for reconstructing a large defect without allowing sagging of the periorbital soft tissue into the sinus.
3. The volume of the implant or transplant. More bulk can be implanted if there is notable preoperative enophthalmos present. Most surgeons advocate the placement of more volume of implant or transplant than considered necessary for reestablishing the former position of the floor and walls. The added bulk should be posterior to the axis of the globe to displace it anteriorly. When bone is used, more than necessary is placed because remodeling and resorption will minimize its size (≈20% to 30%).[182] The volume necessary is difficult to predict; however, postsurgical exophthalmos is rare.
4. Tension-free placement of the implant or transplant. The implant or transplant must be passive when inserted into the wound. In other words, there should be no tendency for an implant to buckle or for its edges to curl up or down, or for the implant to migrate when placed. If any of these occurs, the pocket is too small or the implant too large.
5. Stabilization of the implant or transplant. The implant or transplant must be fashioned so that it cannot be displaced or must be secured with sutures, wires, or bone screws (see Fig. 16-33*C*). Usually, orbital implants migrate anteriorly. This tendency is probably because the implant is improperly sized and placed under tension. The implant should not extend over the infraorbital rim. It usually can be placed so that its anterior end is behind the rim, with the rim acting as a physical impediment to anterior migration (see Figure 16-33, *C*). Stabilization with bone screws and/or bone plates will prevent migration.[210]
6. Careful closure of the wound. The periorbita must be carefully closed with resorbable sutures. This closure is extremely important because it ensures the proper positioning of the orbital septum and helps adapt the tissue over the implant or transplant.

PATIENTS TREATED FOR ZYGOMATICOMAXILLARY COMPLEX FRACTURES

It should be obvious from the earlier discussion and review of the literature that all ZMC fractures do not have to be treated in the same manner. Some require less surgical exposure and fixation than others.* The use of

*References 78, 87, 89, 99, 113, 114, and 118.

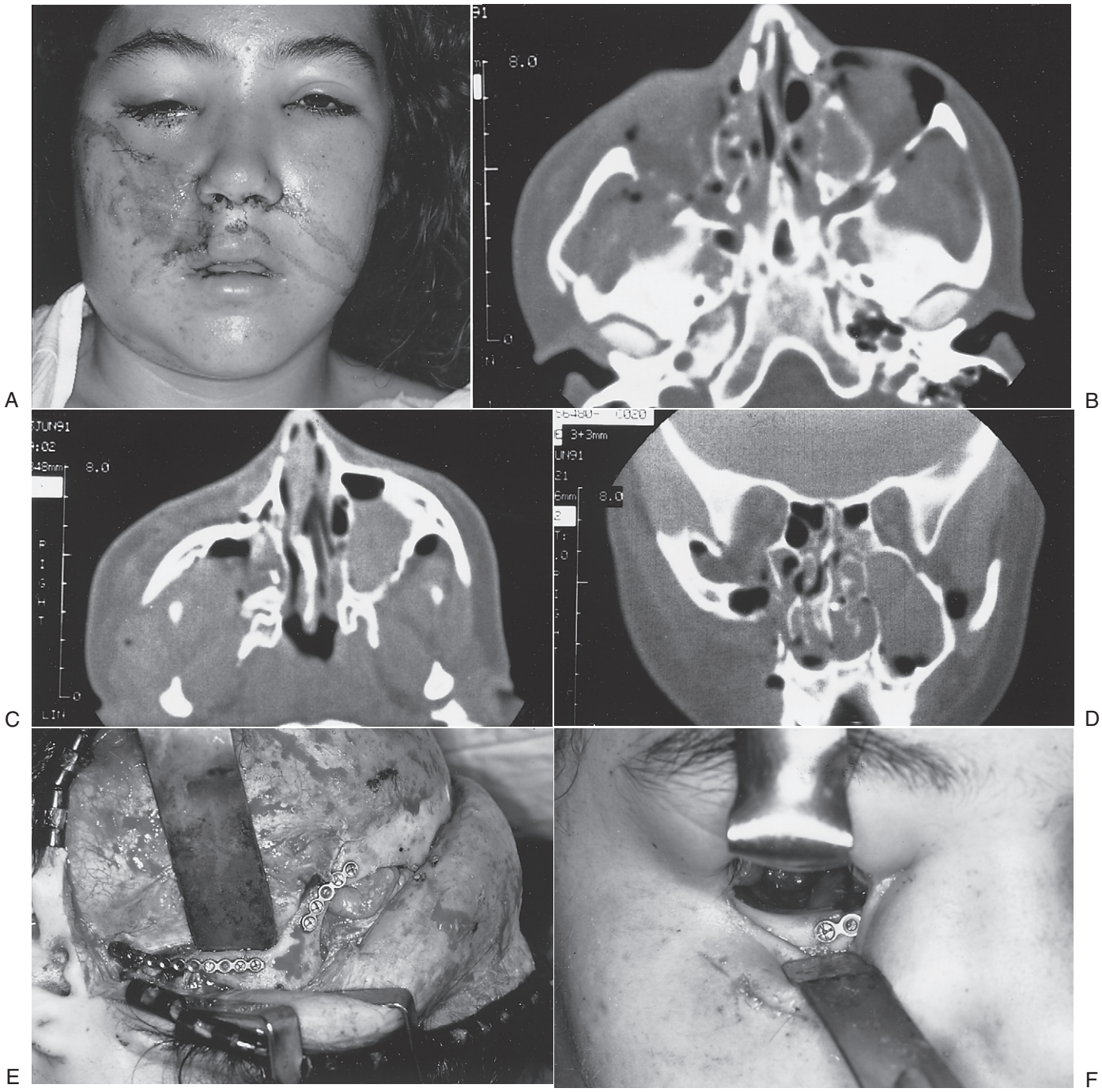


FIGURE 16-34 **A,** Patient sustained severe (high-energy) right ZMC and internal orbital fractures. **B,** Axial CT scan through the zygomatic arch shows retrusion of the entire ZMC and fracture of the arch at the junction with the articular eminence of the temporal bone. **C,** Axial CT scan showing posterior displacement of the ZMC. **D,** Coronal CT scan behind the globe showing disruption of the floor. **E,** Coronal exposure demonstrating reconstruction of the zygomatic arch and bone plate at the frontozygomatic suture area. **F,** After stabilizing the ZMC in position, a large orbital floor defect is present, with only the infraorbital nerve spanning it.

preoperative CT scans has allowed more accurate planning of treatment by identifying the severity of the injuries. Those that are severely displaced or segmented and/or have comminuted articulations usually require extensive internal orbital reconstruction. An aggressive approach to such fractures should be taken and should expose at least the zygomaticomaxillary buttress, infraorbital rim and internal orbit, and lateral orbital rim (Fig. 16-34). In many such cases, the zygomatic arch may also require exposure. The decision to use a coronal approach is based on the amount of displacement of the ZMC posteriorly and laterally and on comminution of the arch.[211,212] If the other articulations located more anteriorly appear to be significantly comminuted,

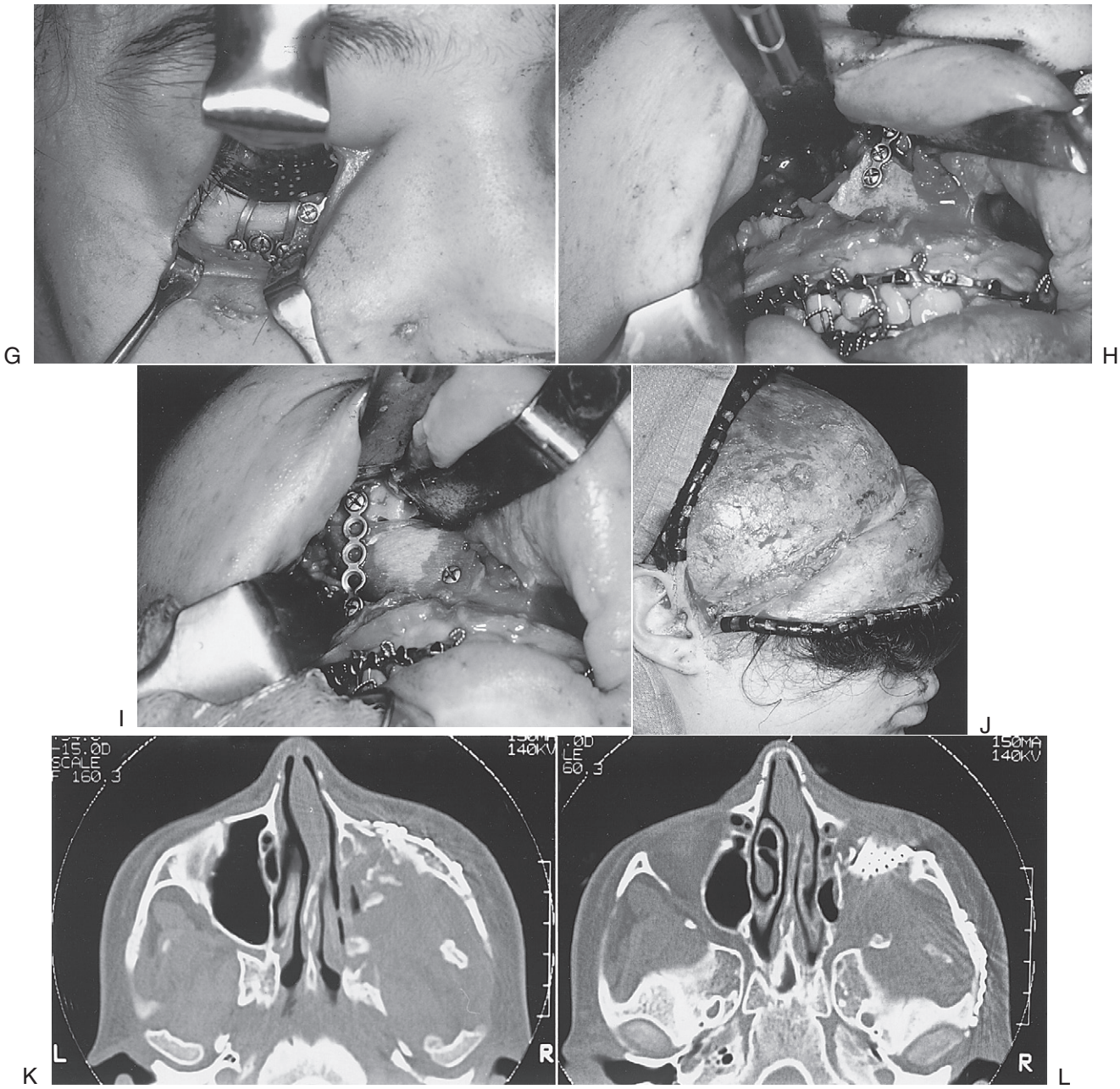


FIGURE 16-34, cont'd **G,** Orbital floor reconstruction bone plate used to span the defect. Autologous bone was placed on top of the plate. **H,** After repositioning the ZMC, a large defect of the lateral maxillary wall is present. Bone plates were used to span the defect. **I,** Bone grafts used to reconstruct the lateral maxillary wall. **J,** Resuspension of facial soft tissue performed before closure. This photograph shows resuspension of the outer layer of the temporalis fascia. **K,** Postoperative axial CT scans showing repositioning of the ZMC and reconstruction of the zygomatic arch **(L).**

exposure and reconstruction of the arch provide another point for reduction and stabilization.

Fractures that do not require internal orbital reconstruction (as determined by the preoperative or intraoperative CT scans) and whose articulations are not comminuted (as determined from the preoperative CT scan) can be treated less aggressively.[99,114,118] In such cases, the treatment algorithm presented in Figure 16-35 can be used. The first step is to elevate the ZMC into what is thought to be the proper position. The surgeon determines this position mainly through palpation of the articulations, visual assessment of malar projection, and/or intraoperative CT scanning and/or navigation. Occasionally, an audible crunch will be heard and palpated when the ZMC is elevated into position. The most important step is to ensure that the ZMC is properly positioned. With minimal edema, the proper position is often easily ascertained by palpating the malar eminences bilaterally.[44] If the surgeon is unsure of the ZMC position, it is mandatory that exposure be performed to examine

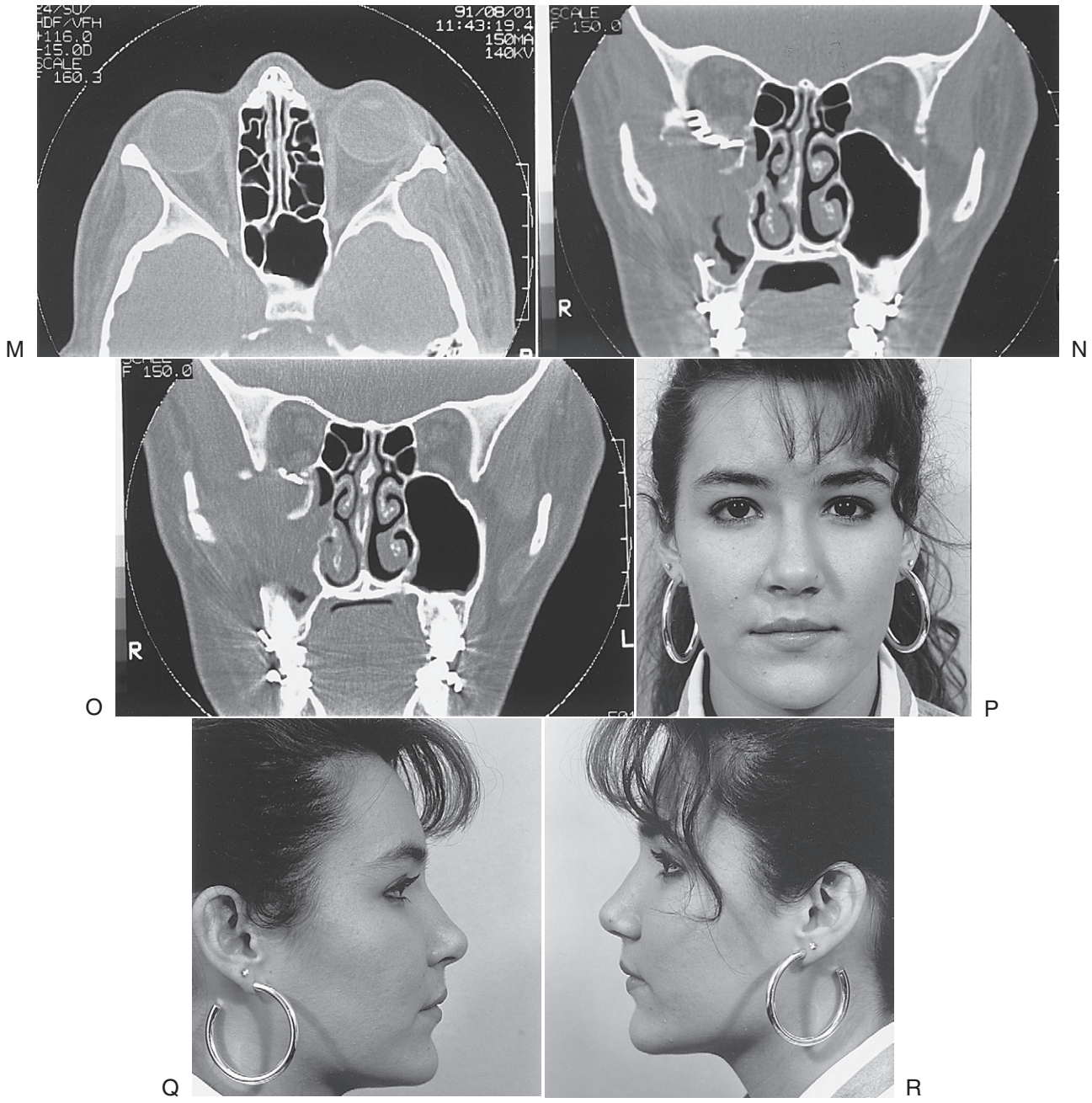


FIGURE 16-34, cont'd **M,** Alignment of the zygomaticosphenoid suture**. N,** Postoperative coronal CT scans show reconstruction of the orbital floor with metal plate and bone graft just posterior to the globe and, more posteriorly, in the orbit **(O).** Frontal **(P),** right **(Q),** and left **(R)** lateral photographs of patient 18 months after surgery.

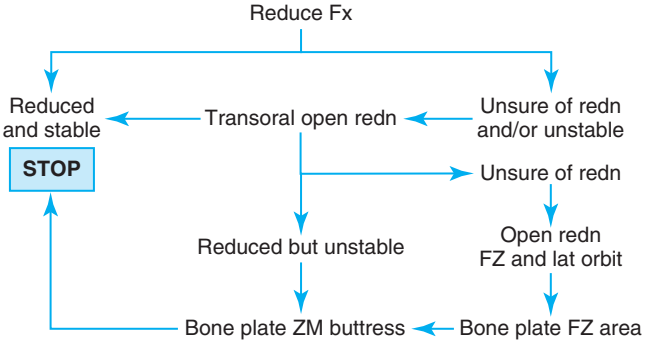


FIGURE 16-35 Treatment algorithm for ZMC fracture without the need for internal orbital reconstruction.



FIGURE 16-36 Patient with low-energy ZMC fracture treated with reduction using a Carroll-Girard screw without fixation. **A,** Frontal and inferior **(B)** views of the patient before surgery. Note the significant contour deficit present. **C,** Axial CT scan at the level of the midglobe shows displacement of the lateral orbital rim and disruption of the lateral orbital wall. **D,** Axial CT scan at the level of the zygomatic arch shows medial rotation of the posterior (infratemporal) surface of the ZMC into the maxillary sinus. **E,** Coronal scan posterior to the globe shows notable displacement of the ZMC, but minimal comminution of the orbital floor. After reduction, the position of the ZMC was thought to be satisfactory, and the ZMC did not displace to digital pressure. No exposure or fixation was therefore applied. CT scans taken after surgery showed satisfactory reduction of the fracture. **F,** Postoperative axial CT scan at the level of the midglobe shows good alignment of the lateral orbital wall.

continued

alignment with adjacent articulations. If one is satisfied with the position of the ZMC and it is firm in its position, as determined by the surgeon attempting to displace it, no fixation is required and no further surgery is necessary (Fig. 16-36). If the ZMC can be positioned properly but must be held in position, it must be stabilized.

In either of these cases, exposure of the zygomaticomaxillary buttress via the maxillary vestibular approach can be performed easily. The use of this approach as an initial point of exposure is predicated on the basis of the following: (1) the scar is hidden; (2) the access for reduction is good; and (3) if fixation is necessary, a bone plate placed on the zygomaticomaxillary buttress provides the best mechanical method of preventing postsurgical displacement in isolated ZMC fractures. After exposure of the entire face of the anterior maxilla and zygoma, a

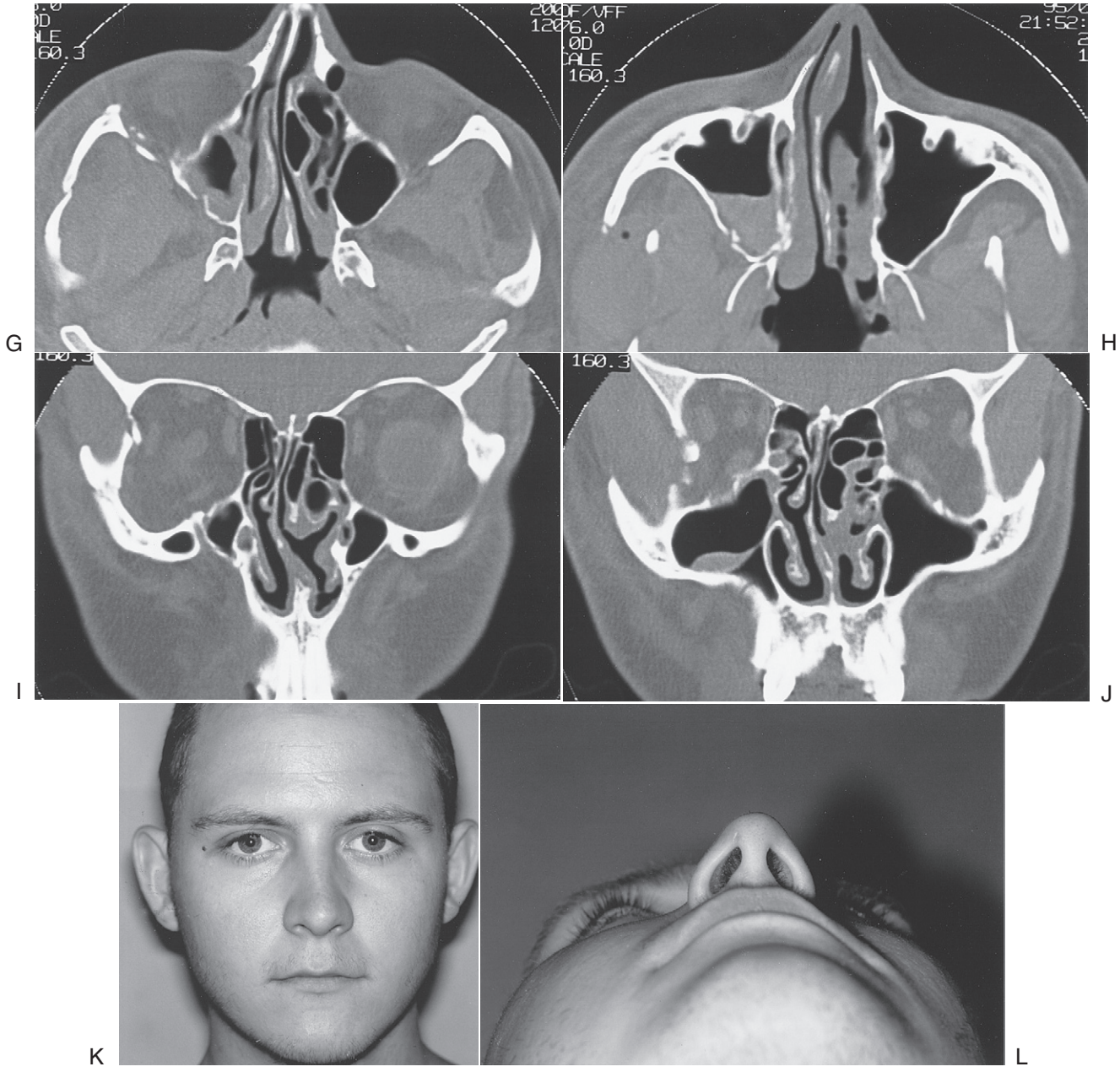


FIGURE 16-36, cont'd **G,** Postoperative axial CT scan at the level of the zygomatic arch shows satisfactory reduction. **H,** Postoperative axial CT scan just below the infraorbital rim shows good position of the malar eminence. **I,** Postoperative coronal CT scan at the posterior surface of the right globe shows satisfactory reduction of the fractures through the lateral orbital wall and floor. **J,** Coronal CT taken behind the right globe shows a satisfactory contour of the right zygomaticomaxillary buttress and maintenance of the contour of the orbital contents. Frontal **(K)** and inferior **(L)** views of patient 5 weeks later showing good symmetry.

broad surface area is visible for assessing alignment of the infraorbital rim and zygomaticomaxillary buttress. Because the ZMC is often comminuted, there will usually be an area in which the fractured zygomaticomaxillary buttress can be aligned with the alveolar process. If the ZMC is found to be reduced and stable, no further surgery is necessary. If the zygomaticomaxillary buttress can be aligned but rotates medially into the maxillary sinus when not supported, a single bone plate is placed at the zygomaticomaxillary buttress. If the ZMC is stable after this one point of fixation, no further surgery is necessary (Fig. 16-37).

If the adequacy of reduction is still uncertain after exposure of the zygomaticomaxillary buttress, or if stability is not adequate even after placement of a bone plate on the zygomaticomaxillary buttress (unlikely), exposure of the frontozygomatic area is performed. The surgeon exposes the frontozygomatic area using an approach through the upper eyelid—an upper blepharoplasty incision. Although a lateral brow approach can also be used,

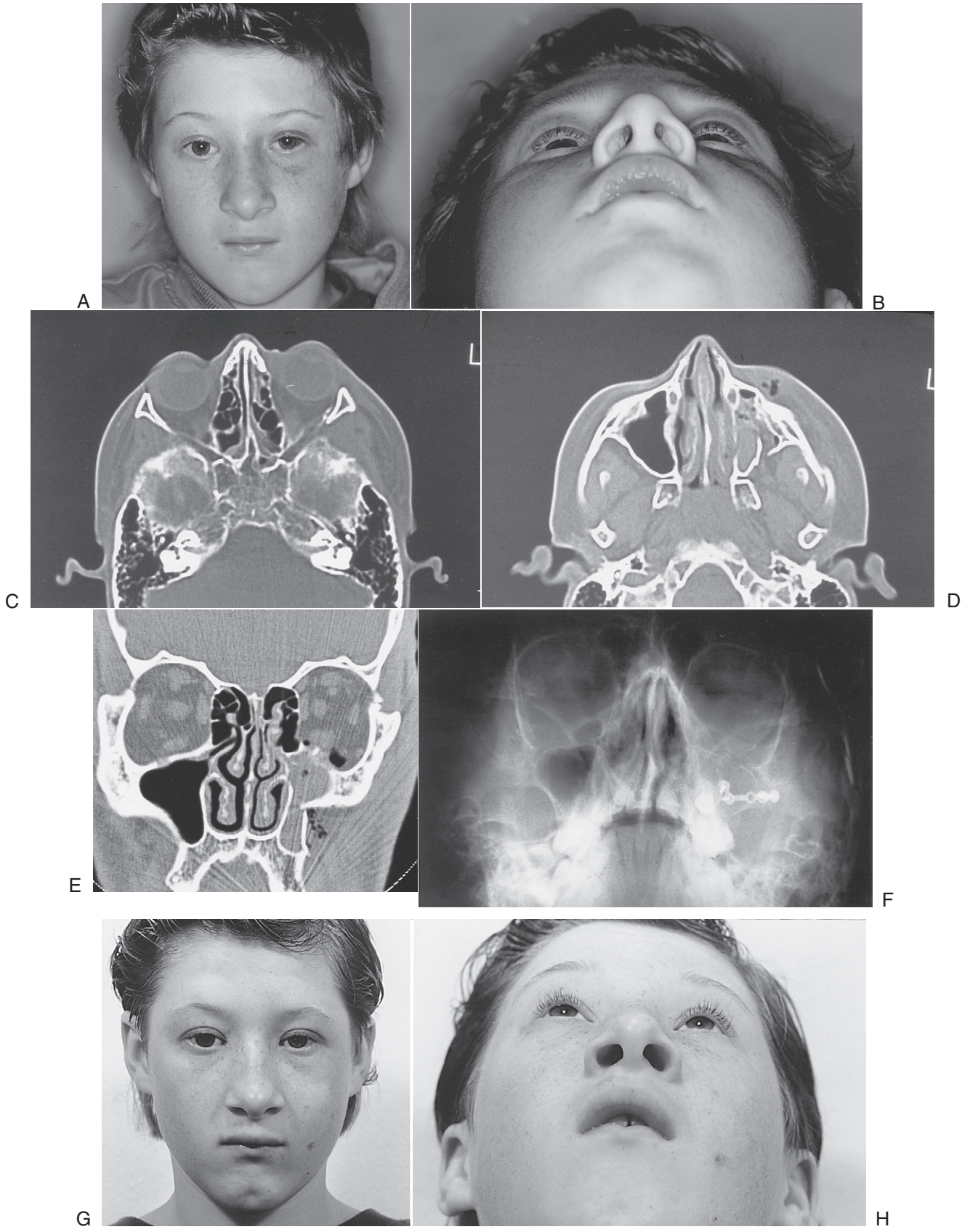


FIGURE 16-37 Patient with low-energy ZMC fracture treated by reduction using Carroll-Girard screw and bone plate fixation along the zygomaticomaxillay buttress. **A,** Frontal and inferior **(B)** views of patient with left ZMC fracture. **C,** Axial CT scan at midglobe level shows minimum displacement of the lateral orbital rim. **D,** Axial CT scan just below the infraorbital rim shows posteromedial displacement of the malar eminence. Note that the fractures are not comminuted. **E,** Coronal CT scan showing noncomminuted orbital floor and walls. The patient underwent reduction with a Carroll-Girard screw, but the malar eminence kept rotating inferomedially into the maxillary sinus. Transoral open reduction and internal fixation with a single bone plate was performed, producing stability of the ZMC. **F,** Postoperative radiography showing symmetry obtained and location of the bone plate. **G,** Frontal and inferior **(H)** views of patient 4 months after surgery.

the exposure provided is considerably less than with an upper eyelid approach unless the incision is extended below the eyebrow. However, this extension often results in a noticeable scar that crosses the resting skin tension lines. Alignment of the fracture through the frontozygomatic area and along the inside of the lateral orbital wall (sphenozygomatic suture area), when combined with alignment of the zygomaticomaxillary buttress, provides excellent assessment of reduction. Whether placement of fixation devices across the frontozygomatic fracture area is necessary is based on the ability to move the ZMC using the Carroll-Girard screw. If necessary, the type and amount of fixation are at the discretion of the surgeon. Because this area is readily palpable, very thin devices should be used. Thin bone plates or a transosseous wire may be all that is necessary when combined with a plate at the zygomaticomaxillary buttress. If the reduction and stability are judged to be adequate, no further surgery is necessary.

If one is still unsure of the reduction (unlikely for a low- or medium-energy injury), the infraorbital rim is exposed via an approach through the lower eyelid. The infraorbital rim can be aligned and, if necessary, stabilized with fixation devices. However, fixation devices in this area should be avoided unless absolutely necessary because of the thinness of the overlying tissue and the tendency for scar formation between the tissue of the lower eyelid and hardware. If fixation hardware is deemed necessary, the thinnest possible device should be used.

ZYGOMATIC ARCH FRACTURES

Fractures of the zygomatic arch are usually the result of fractures of the entire ZMC. However, isolated fractures of the arch without other injuries do occur when a force is applied directly from the lateral aspect of the head. The incidence of these injuries varies, but usually isolated zygomatic arch fractures constitute fewer than 10% of zygomatic injuries.[26] However, others have noted higher incidences, possibly related to the nature of the population.[12,36,76] Conceivably, many isolated zygomatic arch fractures may go unnoticed by the patient or are deemed of insufficient significance to seek treatment.

Isolated zygomatic arch fractures characteristically result in a V-shaped indentation of the lateral aspect of the face, with the apex deep toward the sigmoid notch (Fig. 16-38). There may be only one definite line of fracture, with bending or greenstick fractures in two other areas to produce a W-type configuration of the arch and a V-shaped cosmetic deformity. Occasionally, three definite lines of fracture producing two free segments occur. In this case, the normal convexity of the temporal area is lost. Flattening of the side of the face was noted in 57% of isolated zygomatic arch fractures in a study by Ellis et al.[26]

Accompanying zygomatic arch fractures may be trismus as a result of impingement of the fractured segment on the temporal muscle (see Fig. 16-38*A*). This finding was noted in 45% of 166 isolated zygomatic arch fractures by Ellis et al[26] and in 67% of those in Knight and North's series.[35] The patient may have difficulty in shifting the mandible toward the injured side. An occasional yet interesting finding has also been some visual disturbances, such as diplopia, occurring early after injury and subconjunctival ecchymosis.[8,96]

The necessity for treatment of these injuries is based on clinical detection of cosmetic or functional disturbances. In the study by Ellis et al,[26] 20% of zygomatic arch fractures were not treated. However, other studies have shown a variable ratio of treated versus nontreated zygomatic arch fractures.[20]

Reduction of these fractures can be simply accomplished by any of the techniques already described for ZMC fractures. A percutaneous bone hook, the Gillies temporal approach, and an intraoral approach all are acceptable techniques. The need for stabilizing zygomatic arch fractures varies with the location of the injury, number of fractures, and displacement of the segments. Ellis et al[26] have found that 10 of 126 (7.3%) isolated zygomatic arch fractures treated in their study required fixation. Others have reported that almost every zygomatic arch fracture is stable, once elevated.[35,36,76]

Stabilization of depressed zygomatic arch fractures has been achieved in a number of ingenious ways. Usually, the use of percutaneous circumferential wires or heavy sutures passed around the arch with an aneurysm needle or Mayo trocar and tied to an external object has served this purpose well (Fig. 16-39). Plastic oral airways,[213] metal eye shields,[214] short pieces of endotracheal tubing,[215] and orthopedic finger splints[216,217] all have been used as the external devices. The passing of an awl and tightening of wires in this region of the face may be expected to damage branches of the facial nerve but this complication has not been reported. Some have placed materials such as gauze and balloons between the zygomatic arch and lateral aspect of the mandible through an intraoral approach[81]; however, this approach is usually unnecessary.

Occasionally, the zygomatic arch requires ORIF. Fractures that are in several segments or that are grossly displaced are candidates for this form of treatment. The zygomatic arch can be safely approached from a coronal incision. Once the arch has been identified by subperiosteal dissection, it can be manually repositioned and stabilized. Long, thin bone plates are used to maintain the normal arch morphology. When plates are used, one should be cognizant of the normal flat configuration of the zygomatic arch. Bone plate fixation that provides too much curvature to the arch results in a noticeable cosmetic deformity. Although the zygomatic arch is called an arch, in reality it is not all that curved.

Following reduction of zygomatic arch fractures, one must protect the side of the head from injury. The force of the weight of the head resting on a pillow is sufficient to displace even a properly reduced fracture. Many materials are available that can be taped to the side of the head to protect the zygomatic arch following reduction. Commonly used and readily available materials that can be formed and applied for this purpose are paper cups, metal eye patches, aluminum finger splints bent in a staple configuration,[218] and a number of others (Fig. 16-40). Ideally, they should be left in place for 2 to 3 weeks.

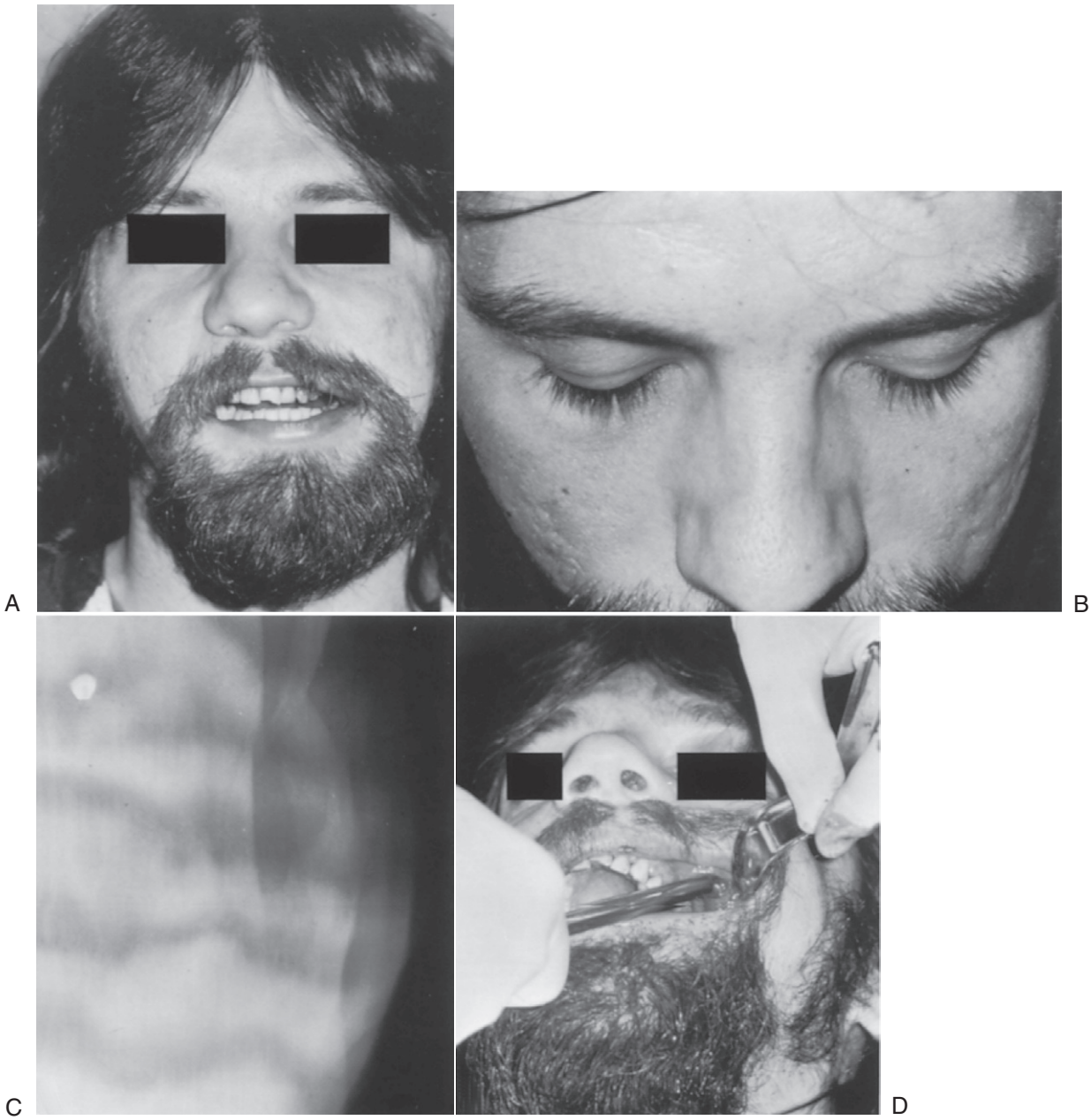


FIGURE 16-38 Reduction of a zygomatic arch fracture via the intraoral route. **A,** Preoperative photographs of the patient showing limited mandibular opening and a flattening over the left zygomatic arch **(B). C,** Preoperative intraoral tangential radiograph showing medial displacement of zygomatic arch fragments. **D,** Urethral sound inserted via a small incision in the maxillary vestibule. The instrument is placed medial to the zygomatic arch and the area of the displaced fracture is elevated while the other hand palpates along the arch **(E).**

continued

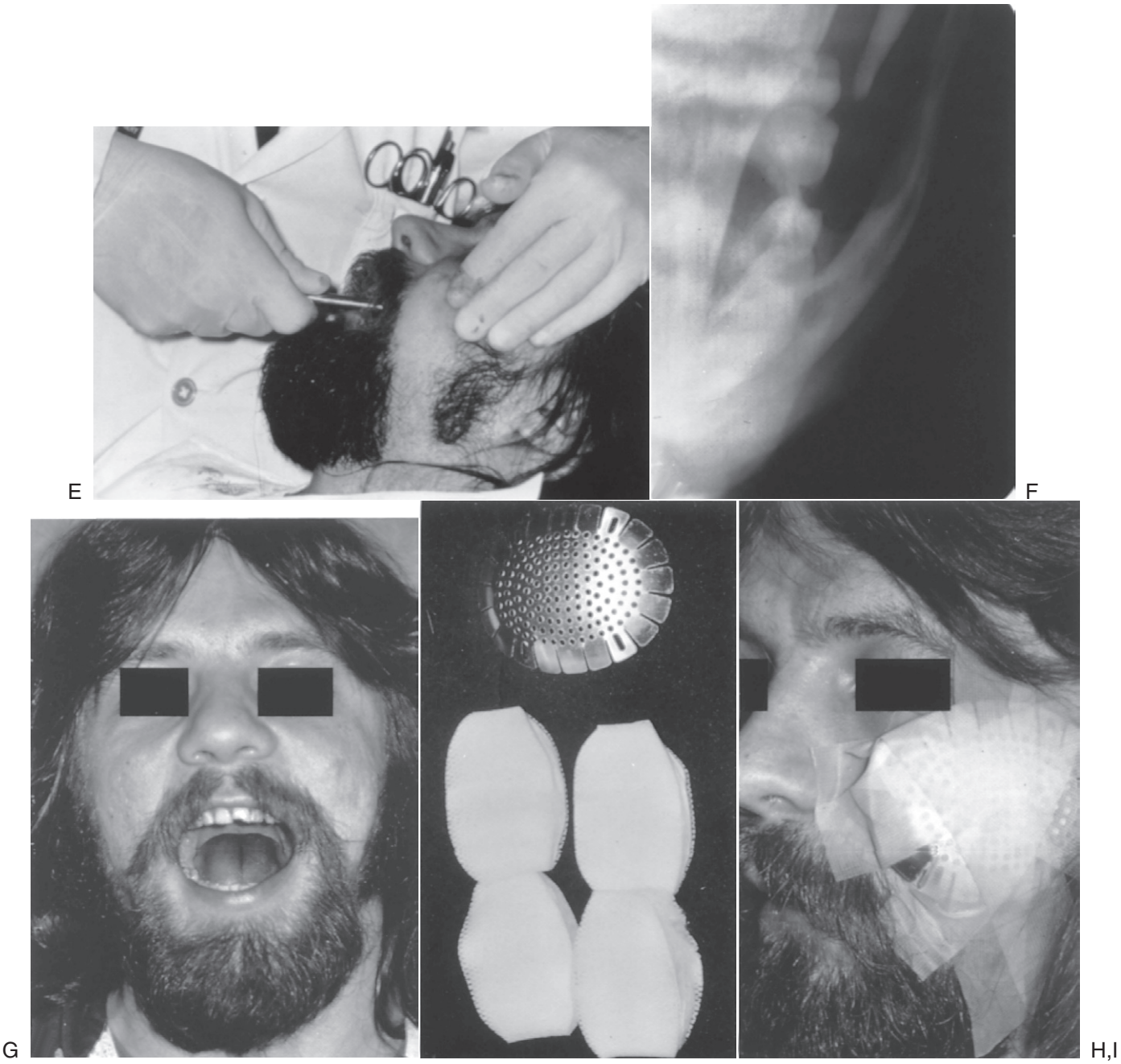


FIGURE 16-38, cont'd **F,** Postoperative radiograph showing reduction. **G,** Immediately postoperatively, patient has regained mobility of the mandible. **H,** Metal and gauze eye patches can be used to protect the reduced arch for several days **(I).**

COMPLICATIONS

PERIORBITAL INCISION PROBLEMS

Several complications can result from the incisions described earlier for approaching the infraorbital rim and orbital floor and walls. Minor complications, such as dehiscence, hematoma or seroma formation, and lymphedema, are more bothersome than difficult to handle. Hematomas and seromas, when large, can be decompressed with a needle and syringe. Lymphedema subsides with time, especially if the lower lid is supported in its proper position. Dehiscence of a wound may require nothing more than observation unless it is large, at which point wound care and resuturing may be necessary.

One problem that may accompany any incision to gain access to the infraorbital rim and internal orbit is a vertical shortening of the lower lid following healing (Fig. 16-41). This shortening probably occurs as a result of scarring between the tarsal plate and periosteum, shortening the orbital septum. To help prevent this, superior support of the lower lid for several days (or until gross edema has resolved) following surgery is beneficial. The most direct method of achieving this support is through the use of a Frost suture, in which a suture is placed through the dermal surface of the lower lid just inferior to the gray line and is taped to the forehead (Fig. 16-42).

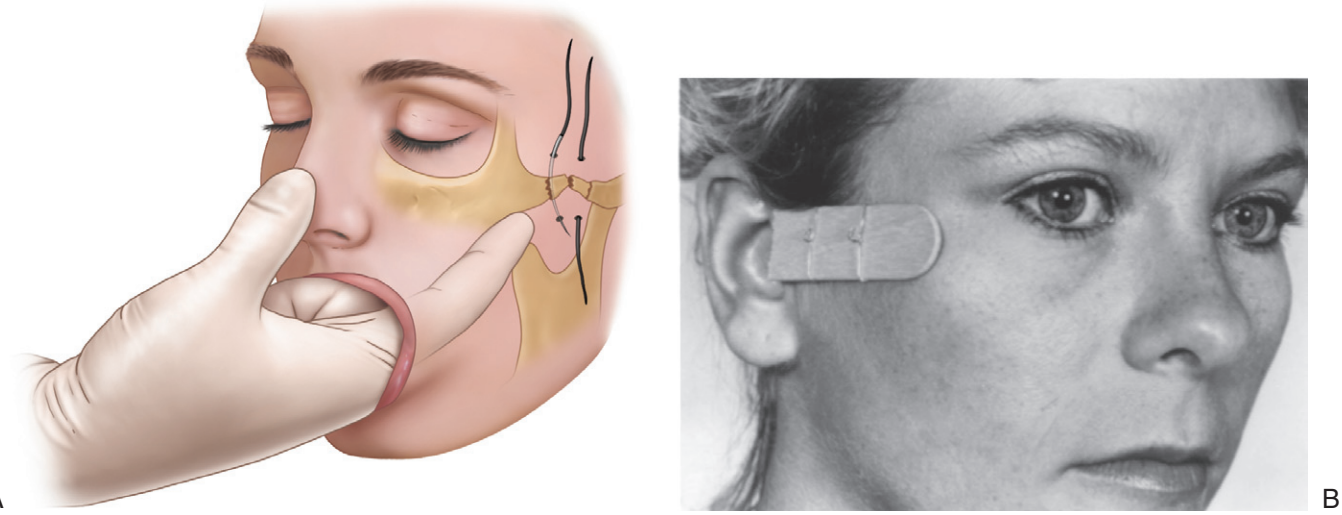


FIGURE 16-39 One method of stabilizing a reduced zygomatic arch fracture. **A,** A large curved needle or an awl is used to pass heavy suture or fine wires around the zygomatic arch. **B,** The wires are then secured to a stable object, such as a tongue blade and aluminum finger splints, until healing has occurred.



FIGURE 16-40 Methods of protecting a reduced zygomatic arch fracture. This can be easily accomplished by using an oral airway **(A),** aluminum finger splint material **(B),** or other suitable object that can be taped over the area for several days.

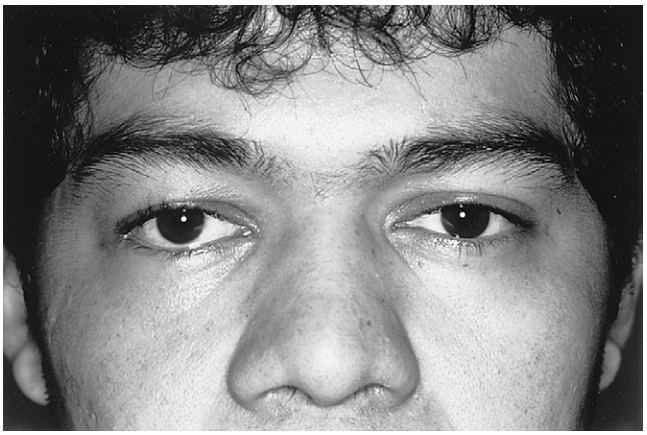


FIGURE 16-41 Patient 7 weeks after treatment of a right ZMC fracture through the transconjunctival approach with lateral canthotomy. Note 1 mm of scleral show.

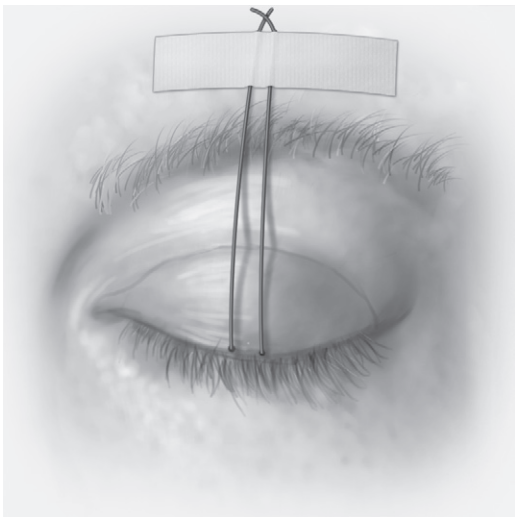


FIGURE 16-42 Frost suture placed through the skin of the lower lid and taped to the forehead to provide support to the lower lid.



FIGURE 16-43 **A,** Patient 8 weeks after treatment of a left ZMC fracture through the transconjunctival approach with lateral canthotomy. Note 3 mm of scleral show, entropion, and the unnatural appearance of the lateral canthus, with the lid not touching the globe. **B,** Six weeks after surgical correction.

This technique closes the eye, supports the lower lid, aids in the dissipation of lid edema, and allows one to examine the globe and vision by simply removing the tape from the forehead and opening the eyelids.

Postsurgical deformities of the lower eyelid are distressing problems, although most cases are self-limited. Ectropion, or an outward curl to the lower eyelid, is classified as mild when there is only slight lifting of the lid from the globe. Moderate ectropion is associated with lifting of the lid from the globe and a shortening of the vertical height of the lower eyelid. Severe ectropion is a combination of shortening of the eyelid and true eversion of the eyelid, not just a lifting away. Mild and moderate ectropion usually resolve with the passage of time and with gentle massage of the lid. Severe ectropion may require surgical correction. Entropion, or an inward curl of the lower eyelid, occurs less commonly but is more distressing because of the irritation of the eyelashes on the globe. Entropion that does not resolve spontaneously may require surgical correction (Fig. 16-43).

The incidence of ectropion or scleral show reported for subciliary incisions with skin and muscle dissection varies considerably.[125,126,219-221] Heckler et al[130] have reported a 6% temporary incidence after a skin-muscle approach to the orbital floor. Manson et al and Dufresne et al[222-224] have noted a 10% incidence of temporary ectropion or scleral show using a skin-muscle flap to approach the orbit. They noted that with time, sufficient resolution occurred that patients did not request corrective surgery. Wray et al[126] have compared the incidence of ectropion following subciliary exposure of orbital fractures with the conjunctival approach and found an extremely high incidence of postoperative vertical lid shortening in the former. After subciliary incisions, ectropion developed in 19 of 45 eyelids, 15 of which were transient and 4 of which required operative intervention. A prospective study by Lacy and Pospisil[225] has reported on 55 skin-muscle dissections through the eyelid to perform surgery for zygomatico-orbital trauma. Ectropion occurred in 18% of their cases, being transient in all but two. They again noted an increased incidence in older patients and in those with edematous lids during surgery. Bähr et al[226] have confirmed that orbits operated on after the onset of traumatic edema develop more complications. They found that ectropion developed in 3 of 16 patients (18.8%) after a subciliary incision with skin-muscle dissection to approach the orbit. However, the ectropion was permanent in only one patient. Antonyshyn et al[181] have found a scleral show frequency of 16.6% with this same approach. Appling et al[227] have compared a subciliary incision with skin-muscle dissection with transconjunctival approaches to the orbit and noted a 12% rate of transient ectropion and a 28% rate of permanent scleral show after the subciliary approach. No transient ectropion and only a 3% incidence of permanent scleral show were found with the transconjunctival approach.

INFRAORBITAL NERVE DISORDERS

Occasionally, a patient who has had treatment of a zygomatic fracture will complain that the upper teeth, especially the anteriors, feel numb or different, and even painful to heat, cold, or light touch. De Man and Bax,[52] in a study of 273 isolated ZMC fractures, found that 80% suffered from dysesthesia on admission. Nordgaard[49] found sensory disturbance in 96% of 100 patients immediately after fracture. Jungell and Lindqvist[51] found that 81% of patients with ZMC fractures had paresthesia of the infraorbital nerve. The figure was even higher (94%) in those who required surgical treatment. Most patients had regeneration, but 42% of patients had some degree of persisting sensory disturbance. Only 12% of patients had total loss of sensation. Similar statistics have been reported by Altonen et al,[20] who observed that 42% of patients had some permanent changes and 10% had marked deficits. Zachariades et al[228] have found that 27% of their patients have alteration of infraorbital sensation 6 months after ZMC fractures. Additionally, they found that inpatients who did not undergo surgery because of minimum or no displacement, all recovered sensation.

An interesting finding with nerve deficits after ZMC fractures has been that fewer deficits remain in patients treated with rigid fixation of their fractures. Champy et al,[111] de Man and Bax,[52] Zingg et al,[87,88] and Taicher et al[229] have stated that reduction and fixation are important factors in the recovery from sensory disturbances of

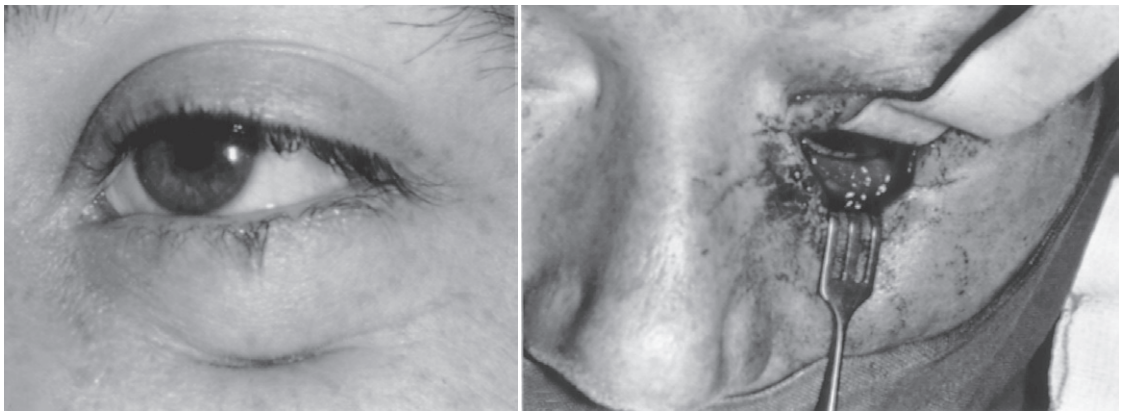


FIGURE 16-44 Patient who had a silicone implant used to reconstruct his orbital floor 13 months previously. **A,** For the past several months, he had intermittent swelling and drainage from this sinus tract. **B,** At surgery, the implant was found to be surrounded by chronic inflammatory tissue and was removed. The sinus tract was excised to gain access to the orbital floor. He had no further problems following implant removal.

the infraorbital nerve. They maintain that fixation of the fracture line by a miniplate, mainly in the frontozygomatic area, achieves the fastest recovery rate of neurosensory dysfunction. The proposed method whereby recovery improves is that improved stability prevents continued compression on the nerve after reduction.

Tajima[53] has indicated that full recovery should occur within 5 months. Jungell and Lindqvist[51] have found that most recovery occurs early, within the first 2 weeks. However, Lund[28] has noted sensory disturbances in almost 50% of patients more than 2 years after their injuries. In cases of persistent dysesthesia, anesthetization of the superior alveolar nerves by local infiltration should be attempted. If symptoms are not alleviated, the clinician should suspect a disruption of the infraorbital nerve within its canal where the middle and anterior superior alveolar nerves take origin, with possible neuroma formation. Surgical exploration may be necessary when the altered sensation is bothersome to the patient.

IMPLANT EXTRUSION, DISPLACEMENT, AND INFECTION

The possible risks that always exist when an alloplastic material is used are infection, displacement, and extrusion of the implant. Infection usually occurs early and may result in the need for implant removal. These complications are uncommon but do occasionally occur. Aaronowitz et al[230] have reported a 3.9% early complication rate (within 1 month of surgery) when Teflon implants were used to reconstruct the orbital floor. These complications consisted of infections and improper placement of the implant, necessitating removal in all cases. They also found a 2.8% late complication rate, which included one patient with a cutaneous antral fistula. Correlation criteria were established to determine whether any preoperative or intraoperative findings correlated with the complication rate. The only positive correlation was an association between the concomitant use of antral packs and implants. Therefore, they recommended that this application be avoided. The association between antral packs and implants has also been noted by Spira.[231]

Polley and Ringler[232] have reviewed 230 Teflon implants used on the orbital floor over a 20-year period and found only one postoperative infection that necessitated implant removal. There were no other complications. The implants were not routinely sutured to the orbital floor in their series. Similarly, Morrison et al[195] have reviewed 311 cases of silicone implants placed over a 20-year period and found that at least 13% required removal for one reason or another.

Particularly distressing are the occasional occurrences of acute foreign body reactions to silicone[191] and Teflon[190] orbital floor implants many years after implantation. It may be that slight trauma to the implant precipitates this reaction, because one of the patients reported was subjected to a blow to the orbit that preceded the acute reaction. In these subjects, implant removal and at least partial removal of the inflammatory tissue allowed resolution of the process. Other series have shown complication rates ranging from 3% to 15% with the use of alloplasts other than Teflon.[233-238] When the implants become displaced or extruded, they should be removed (Fig. 16-44). It is usually not necessary to place another at the time of surgery; however, if enophthalmos or ptosis occurs, reconstruction of the internal orbit can be undertaken secondarily.

PERSISTENT DIPLOPIA

Binocular diplopia present initially after zygomatic fracture is generally a result of edema or hematoma of one or more extraocular muscles or their nerves and intraorbital edema or hematoma. In these cases, resolution of diplopia following fracture treatment (if necessary) usually occurs spontaneously within 5 to 7 days.[49,239] Occasionally, muscle entrapment is the cause of diplopia but such entrapment should be apparent with the use of a forced duction test.

Persistent diplopia occurs in a small percentage of patients after what appears to be appropriate treatment, ranging from 3% to 15% in reported series. The cause of persistent diplopia is not known, but it has been thought to result from scar contracture and adhesions in ocular muscles or between them and other structures.

Neural injuries from the trauma or from surgery may also produce persistent diplopia. It should be pointed out that few of these patients complain of their diplopia, and blurring of vision may be found only in upward and lateral gaze. If the diplopia is bothersome, the patient should be referred to an ophthalmologist for evaluation and possible treatment with exercises and/or surgery.

ENOPHTHALMOS

Enophthalmos may be present, even after what appeared to be proper treatment at the time of the operation. Few patients are aware of enophthalmos and it therefore seldom presents a clinical problem unless severe. The incidence of enophthalmos varies considerably from one report to the next, depending on how much globe retrusion is considered to represent enophthalmos. The usual figure is low, reported between 5% and 12%.[27,29,64] However, Altonen et al[20] have noted enophthalmos in 41% of patients. The reason for this high incidence probably stems from the 26% incidence of slight enophthalmos in their series. If one takes away the 26% who had slight enophthalmos, the figure becomes a more understandable 15%. In a comprehensive study of patients treated for complex orbital fractures, Antonyshyn et al[181] have noted moderate enophthalmos, characterized by more than 3 mm of difference in projection from the uninjured globe, in 3 of 49 patients, and severe enophthalmos (more than 4 mm of difference) in 4 patients. Thus, 14% of their complex orbital injuries had some degree of enophthalmos.

Enophthalmos has been thought to be caused by a decrease in volume of the orbital contents, increase in volume of the bony orbit, loss of ligament support, scar contracture, or combination of these. The most popular theories of the mechanism of enophthalmos have been bony orbit enlargement and fat atrophy. A study by Manson et al,[67] which evaluated patients demonstrating post-traumatic enophthalmos using quantitative CT, found that an increase in bony orbital volume was present in these cases. Others have demonstrated similar findings.[66,240] The study by Manson et al, however, did not find loss of soft tissue volume within the orbit, which could signify fat atrophy. It is probably unusual to have great losses of orbital soft tissue volume unless infection has occurred, producing post-traumatic fibrosis and atrophy of the periorbital fat. Thus, post-traumatic enophthalmos is usually caused by an increase in bony orbital volume (Fig. 16-45; see also Fig. 16-14). Even after restoration of the orbital rims and floor at the time of surgery, defects located posteriorly along the medial and/or lateral walls are common and frequently overlooked, and are probably the main reason for postoperative enophthalmos.[63,241,242]

Enophthalmos is difficult to correct secondarily; however, improvement is possible. The goal of surgery is to reduce orbital volume by reconstructing the internal orbit and, if necessary, placing a space-occupying material behind the globe, thus displacing the globe anteriorly (see Fig. 16-45). A space-occupying material placed in front of the globe worsens the enophthalmos and that placed along the axis of the globe only shifts the globe to the opposite side.

Several materials have been used to decrease orbital volume, such as glass beads,[243-245] silicone sheets or sponges,[246-249] Teflon beads,[250] cartilage grafts,[251-253] porous polyethylene sheets,[185] hydroxylapatite,[254,255] and metallic mesh or plates.[203,205] The advantage of using nonresorbing materials is that they maintain their bulk within the orbit; however, extrusion, migration, and infection are always possible. The implant or bone may need to be placed in several locations within the orbit to affect the anterior projection of the globe; therefore, access to almost the entire circumference of the orbit is often necessary. Usually, the orbital floor, medial wall, or posterolateral wall of the orbit requires an implant or graft posterior to the axis of the globe.[131]

BLINDNESS

Reduced vision and blindness have occasionally been reported after the treatment of zygomatic fractures. Ord[256] has reported that the incidence of postoperative retrobulbar hemorrhage and blindness following treatment of zygomatic fractures is 0.3%. Blindness has also been reported in patients following internal orbital reconstruction.[186,257-261] These complications are extremely rare occurrences, but they have devastating consequences.

There are several causes of reduced vision following trauma or fracture repair. Direct damage to the optic nerve from displacement of a fracture segment or from a fractured optic canal is rare but possible.[4,262-265] Postmortem investigations, however, have demonstrated that injury to the optic nerve resulting from optic canal fractures is rarely the result of osseous compression, laceration of the nerve, or hemorrhage into the nerve itself. More often, hemorrhage into the optic sheath or contusion of the nerve results in edema and compression.[266,267] The injury may lead to secondary compression of the vascular supply to the nerve where the nerve sheath is fixed to its bony surroundings. Another cause of blindness following zygomatic fracture or fracture repair is retrobulbar hemorrhage.

A major question that must be answered when blindness follows fracture repair is to determine what caused the blindness—the trauma or the surgery. The answer is important from a surgical standpoint and obviously of interest from a medicolegal standpoint. Unfortunately, one cannot always know the answer. If the patient was blind before surgery, the answer is obvious. However, most cases of blindness associated with zygomatic fractures have followed surgical intervention. It might therefore be concluded that blindness occurring after surgical intervention, which was not present before, is a result of the surgery. However, there have been reports in which blindness occurred days following injury, even when no surgery had been performed.[268,269] Spontaneous retrobulbar hemorrhage has also been noted following fracture but before fracture repair.[270,271] Thus, if the fracture had been treated, it might have been thought to be responsible for the blindness. Unfortunately, there is no ideal method of sorting out these problems.

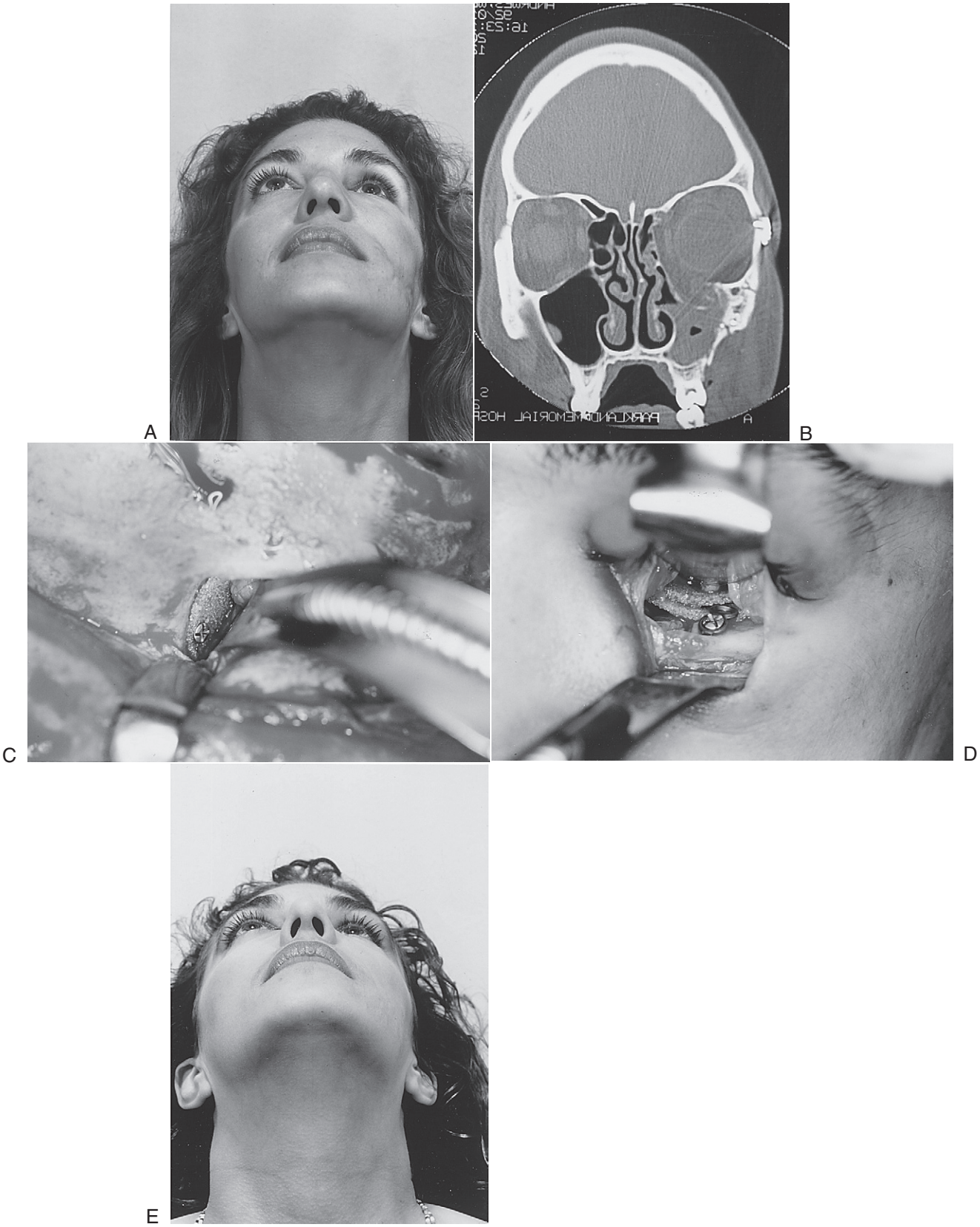


FIGURE 16-45 **A,** Clinical appearance 9 months after surgical correction of a left ZMC fracture (and facial lacerations). Note 3 to 4 mm of enophthalmos. **B,** Coronal CT scan showing large, untreated orbital floor and medial wall fractures. **C,** Intraoperative photograph of the left medial orbital wall exposed using the coronal approach. A porous polyethylene sheet was used to cover the entire medial orbital wall defect. Note that the sheet was stabilized with a bone screw. The sheet extended down to the orbital floor, covering that defect. **D,** Intraoperative photograph of the floor of the left orbital floor showing a sheet of polyethylene from the medial wall, covering a defect in the orbital floor. A second sheet of porous polyethylene was used along the floor and medial wall to occupy a space posterior to the globe to help correct the enophthalmos. **E,** Patient 6 months after surgery showing marked improvement, but slight residual enophthalmos remains.

RETROBULBAR AND INTRAORBITAL HEMORRHAGE

Intraorbital and retrobulbar hemorrhage can occur from the traumatic event or the surgery to repair the fractured zygoma. Reduced vision and blindness resulting from orbital hemorrhage have been reported in several cases of zygomatic fracture and fracture repair.* The cause of the reduced vision and blindness in these cases is unclear, but many have thought that these complications were a result of retinal artery occlusion. The occlusion of the retinal artery may be secondary to direct compression of the artery, sufficient stretching until the artery goes into spasm, or both. One explanation for the mechanism whereby stretching of the retinal artery is possible is that when hemorrhage into or around the muscle cone occurs, the eye protrudes. Because the muscles are fixed posteriorly to the tendinous ring, they are stretched along with the nerves and vessels of the globe. Another explanation for the reduced vision has been offered by Hayreh,[278] who postulated that increased intraocular pressure reduces the perfusion of the anterior head of the optic nerve in a progressive manner. Whatever the mechanism, the increase in intraorbital pressure from hemorrhage causes changes that can lead to blindness if not halted.

The signs and symptoms of retrobulbar hematoma include a tense proptosis (exophthalmos), periorbital swelling that may be in the process of increasing in size, retroorbital pain, dilation of the pupil, and ophthalmoplegia. Hueston and Heinze[279] have stated that "retrobulbar hemorrhage is not an emergency, total blindness is." If the process becomes static at this point, and if vision and retinal circulation are maintained, observation is indicated.[256,280] Fortunately, the vast majority of retrobulbar and intraorbital hemorrhages do not progress to produce visual impairment[281,282] and, when they do, most produce only transient and/or partial loss of vision.[283] Most ophthalmologists do not treat retrobulbar hemorrhages or treat them conservatively with the application of ice, sedatives, bed rest, and/or diuretics, such as intravenous mannitol. Observation for signs of visual impairment, however, is warranted. Gradual absorption of the hemorrhage occurs and full range of motion usually returns in several weeks but cases have been reported in which blindness occurred days after surgery because of the development of the hematoma.[186,276]

When the point is reached that the optic nerve or retinal artery becomes involved, the pupil becomes fixed and nonreactive to light. Funduscopic examination may reveal a pale edematous fundus, with blurring of the disc margins. The classic sign of arterial occlusion or spasm, the macula appearing as a bright red (cherry red) spot, is infrequently observed in reported cases.[284] These findings, associated with visual loss, constitute and should be considered a medical emergency because permanent loss of vision will occur in several minutes if the orbit is not immediately decompressed. Hueston and Heinze[279] have claimed that survival of the optic nerve head is at stake in this situation and 60 minutes of ischemia appears to be the limit for survival and recovery, although Rowe[33] has indicated that 15 to 20 minutes is a more accurate figure.

An ophthalmologist should be summoned immediately while orbital decompression is instituted. If one has placed an antral pack, this should be immediately removed. Orbital decompression can then be performed by a variety of approaches. If a transantral approach to reduce or fix the fractured zygoma has already been used, this approach can be used to decompress the orbit. The orbital floor should be carefully but quickly removed and the periorbita incised if it has not already been disrupted. This incision should provide an avenue for the escape of accumulated blood and cause an immediate increase in orbital volume. Careful and gentle suctioning by means of thin polyethylene tubing inserted into the periorbital tissue may also find pockets of hemorrhage.

If one has made an incision at the infraorbital rim for fracture reduction, it should immediately be reopened. If no blood is encountered below the periorbita, the periorbita should be incised if it has not already been lacerated from the injury, and blood should be evacuated. Careful blunt dissection through the periorbital tissue and dissection through the muscular septum between the lateral and inferior rectus muscles allows the intramuscular cone to be drained. Aspiration with a short piece of polyethylene tubing may help find areas of sequestered blood.[33]

If no periorbital incisions were made during fracture reduction and/or fixation, a 2-cm inferior lid incision should immediately be made. In contrast to the usual approach to the infraorbital rim, however, incision through the orbital septum is desirable when one is surgically decompressing the orbit. Blunt scissors should be inserted within the wound along the inferior aspect of the orbital floor and spread to evacuate accumulated blood. Dissection into the muscular cone, as just described, may also be necessary.

If any of these measures are not successful, as indicated by the expulsion of fresh hemorrhage and the beginning of relief of proptosis, access to the superolateral aspect of the orbit should be provided. This can be rapidly and safely achieved via an eyebrow approach with dissection through the periorbita.[285,286] Decompression via a lateral canthotomy and small curvilinear incisions above and below the lateral canthus have also been advocated.[186,287]

Decompression of the globe by perforating the anterior chamber of the eye has been suggested to be an effective treatment for managing retrobulbar hemorrhage[249,288]; however, many surgeons doubt its effectiveness.[280,281] In any case, it is not recommended as an emergency measure by nonophthalmologists.

Other measures that should be used in conjunction with the aforementioned are the following: control of the systemic blood pressure, if high; bed rest; possible use of diuretics (e.g., IV mannitol, 200 mL of a 20% solution; 500 mg of acetazolamide [Diamox] IV); and high doses of systemic steroids (e.g., dexamethasone, 3 mg/kg initially and then 2 mg/kg every 6 hours). Ophthalmologic follow-up is mandatory.

*References 6, 256, 265, 270, and 272-277.

MALUNION OF THE ZYGOMA

Malunion of the zygoma can be the result of improper reduction, improper fixation, or nonintervention when surgery was indicated. The last situation happens occasionally when the patient's medical condition precludes early operative intervention, treatment is not sought by the patient, or the patient declines surgery until later. The signs and symptoms are the same as those seen in a patient with a fresh zygomatic fracture, including flattening of the malar prominence, enophthalmos, altered pupillary level, and limitation of mandibular motion.

When confronted with this problem, one has two treatment alternatives—camouflaging the defect with an implant or transplant or repositioning the malpositioned bone. The advantages and disadvantages of a malar implant or transplant and osteotomy should be understood. In cases of malunion, when minor deformity is present and limited to flattening of the malar eminence, with little orbital involvement, a subperiosteal implant or transplant can be inserted to restore normal facial form. Another situation in which an implant or transplant is useful is when the zygoma is so comminuted that it cannot be mobilized and repositioned in one piece. Many implant or transplant techniques and materials are available for this purpose. There are advantages and disadvantages to the use of any material, and several have been used for malar augmentation. Bone has been less frequently used than alloplastics because of the difficulty in contouring bone and the unpredictable amounts of resorption that may occur. Most surgeons use silicone or porous polyethylene implants in a subperiosteal location. The implant techniques described are beyond the scope of this chapter. If necessary, a coronoidectomy can be performed, along with implantation to improve the range of mandibular motion.

When concomitant functional deficits are present along with cosmetic deformity, zygomatic osteotomy should be considered because it will correct both problems simultaneously. If zygomatic refracture or osteotomy is selected as the appropriate treatment modality, preoperative CT is warranted. The areas of fracture, position of the globe, orbital volume and shape, and defects in the orbital floor or walls should be thoroughly investigated. Restoring a severely malpositioned zygoma to its proper position following malunion and simultaneously correcting existing orbital defects is a difficult and challenging undertaking. The ZMC must usually be refractured or, more appropriately, osteotomized to allow repositioning. Fixation is always necessary, and restoration of normal globe position may require internal orbital reconstruction of the bony orbit. Several techniques for zygomatic osteotomy have been used in the past and all can produce good results with proper attention to detail. However, preoperative planning and intraoperative repositioning are the most important steps. Software for computer planning and intraoperative navigation have greatly improved outcomes for such malunions.

Various soft tissue access incisions allow visualization of the osseous anatomy for osteotomy. Some surgeons refracture the ZMC using standard incisions, such as the eyebrow, inferior lid, and intraoral. Another approach that greatly facilitates zygomatic osteotomy is the coronal approach used in conjunction with subciliary and intraoral approaches (see earlier, "Coronal Approach"). With this approach, the zygoma can be totally freed from all external soft tissue attachments, facilitating osteotomy and repositioning. Although there may be concern that the zygoma will resorb or become infected after extensive stripping of soft tissue, this is a rare occurrence in craniofacial surgery, in which large segments of osseous tissue are completely stripped from their soft tissue attachments and replanted.[289] If the old fracture site is visible, it can be used as the line of osteotomy. However, the fracture along the orbital floor should be used only if it does not extend too far posteriorly toward the orbital apex. A sharp osteotome, thin bur, or saw can be used to perform the necessary bony incisions. Care is taken to protect the infraorbital neurovascular bundle within its groove along the orbital floor and at its exit from the infraorbital foramen on the anterior surface of the maxilla. Care is also taken to protect the orbital contents.

The most difficult osteotomy to make is that extending from the inferior orbital fissure, along the temporal surface of the maxilla to the zygomatic buttress area of the maxilla. This osteotomy is greatly facilitated by the use of the hemicoronal flap, which permits access to this difficult area. A sharp osteotome can be easily inserted from the temporal approach into the inferior orbital fissure and can be used to create a fracture down the infratemporal aspect of the maxilla. The osteotomy along the anterior maxillary wall can be extended inferolaterally until it meets the posterior osteotomy at the zygomatic buttress of the maxilla. Some also use an intraoral approach in the maxillary vestibule to help complete this osteotomy. This is a helpful optional incision because it may be necessary to place bone grafts into the zygomatic buttress area, especially if nonrigid forms of fixation are applied along the orbital rims. Before mobilization, the zygomatic osteotomies should be carefully checked to verify that all osseous incisions have been completed. If mobilization is attempted while some bony areas are still intact, aberrant fractures may occur.

After the zygoma has been mobilized, it is repositioned. If the malunion was long-standing, it may be necessary to excise bone in some areas in which callus and new bone have formed to permit proper reduction. The lateral orbital rim may need to have some bone subtracted for proper repositioning; however, this is not always the case. The most accurate method of repositioning involves the use of intraoperative navigation to help the surgeon place the zygoma into the most symmetrical location, as determined by preoperative computer planning. The zygoma is best stabilized with bone plates at the frontozygomatic and possibly infraorbital areas. If necessary, bone grafts are inserted into bony voids (Fig. 16-46). The internal orbit is reconstructed as described earlier, with care being taken to correct defects in the lateral wall of the orbit. When the zygoma has been comminuted by trauma, it may be necessary to onlay graft or implant over the malar prominence to reestablish normal contour. The zygomatic arch can be reconstructed with a strip of cranial bone or rib.

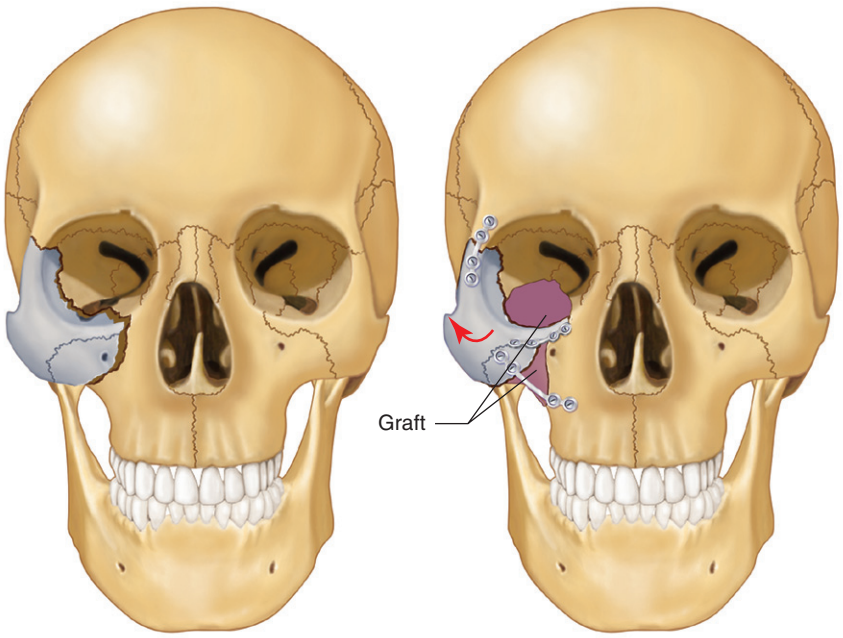


FIGURE 16-46 Osteotomy to reposition the malaligned ZMC. In most cases, the old fracture site is visible and may serve as the osteotomy site. *Left,* Note the medial rotation of the right zygomaticomaxillary buttress into the maxillary sinus. *Right,* Following repositioning and stabilization, it is usually necessary to bone-graft the area of the zygomatic buttress of the maxilla to rotate the body of the zygoma outward and upward. Internal orbital reconstruction may also be necessary.

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CHAPTER 17

Diagnosis and Treatment of Midface Fractures

Christopher D. Morris | Paul S. Tiwana

OUTLINE

Le Fort Classifications
Emergency Care
Facial Examination
Le Fort Type I Fractures
Anatomic Considerations of Fractures of the Maxilla
Treatment of Unilateral Maxillary Fractures
Treatment of Le Fort Type I Fractures
Le Fort Type II (Pyramidal) Fractures
Treatment
Palatal Fractures
Classification
Treatment
Naso-Orbital-Ethmoid Injuries
Anatomy
Diagnosis
Physical Examination
Classification
Treatment
Le Fort Type III Fractures
Treatment
Complications
Malunion of Midface Fractures
Treatment
Orbital Wall Fractures
Blowout Fractures
Medial Wall Fractures
Blow-In Fractures
Complications After Treatment of Midface Fractures
Bleeding
Cerebrospinal Fluid Rhinorrhea and Otorrhea
Ocular Complications
Neurologic Complications
Lacrimal System
Anatomy
Evaluation
Dacryocystorhinostomy
Emerging Surgical Techniques and Materials
Endoscopic Management of Midface Fractures
Advances in Computer-Based Imaging Techniques

The midface is important functionally and cosmetically. It serves an important role in vocal resonance within the sinuses of the facial bones as well as in the function of the ocular, olfactory, respiratory, and digestive systems. The face is also fundamental to interpersonal recognition and the perception of self-image.

The midfacial complex is constructed of a series of vertical pillars that primarily provide protection from vertically directed forces. These include the nasomaxillary (nasofrontal), zygomaticomaxillary, and pterygomaxillary buttress.[1] These vertical pillars are further supported by the horizontal buttresses—the supraorbital or frontal bar, infraorbital rims, and zygomatic arches.[2,3] Contrary to claims of a lack of sagittal buttresses, the midface does have support, however weak, from the maxillary walls, lateral nasal wall, and nasal septum. Clearly, these weaker buttresses of the midface tolerate frontal or laterally directed forces poorly.[4] Behind this buttress system sits the medial and lateral pterygoid plates inferiorly and the skull base superiorly. This framework results in a few anatomic sites of weakness, resulting in fairly predictable patterns of fracture.

LE FORT CLASSIFICATIONS

Rene Le Fort's cadaver studies in the early twentieth century defined the three weakest levels of the midfacial complex when assaulted from a frontal direction. He defined the three most common "linea minoros resistentiae," which are classified as the Le Fort I, Le Fort II, and Le Fort III fractures. These fracture patterns are characteristic of a unidirectional, low-energy injury rather than the multivector, high-energy mechanisms commonly observed today. However, this system is popular because it provides a simple, anatomically differentiated system for the general classification of midfacial injuries.[5]

The Le Fort type I fracture pattern results from a force directed above the maxillary teeth, resulting in a floating palate (Fig. 17-1). The Le Fort type II fracture pattern results from a force delivered at the level of the nasal bone, resulting in mobility of the midface through the orbits and midfacial region (Fig. 17-2). The Le Fort type III fracture pattern results from a force directed at the orbital level, resulting in a craniofacial dysjunction or separation of the entire middle third of the craniomaxillofacial skeleton from the skull base (Fig. 17-3).

EMERGENCY CARE

Initial evaluation of the severely injured midface can be an intimidating experience (Fig. 17-4). Emergency care should be immediately initiated, applying the principles of Advanced Trauma Life Support (ATLS). When dealing with midfacial injuries in the emergency setting, certain

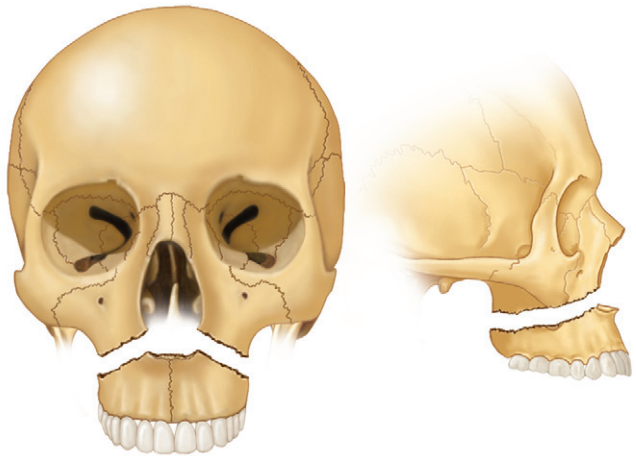


FIGURE 17-1 Le Fort I fracture.

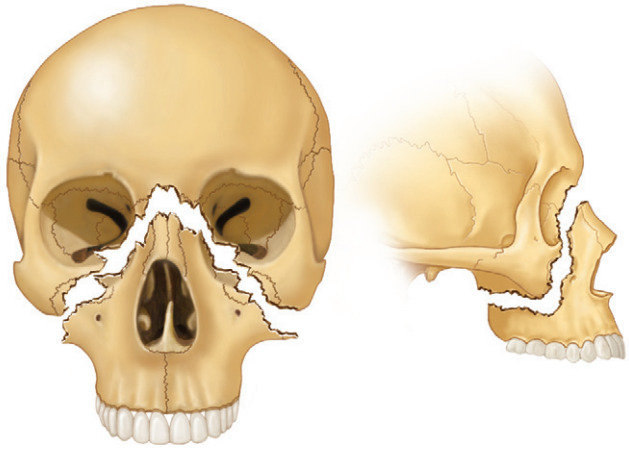


FIGURE 17-2 Le Fort II fracture.

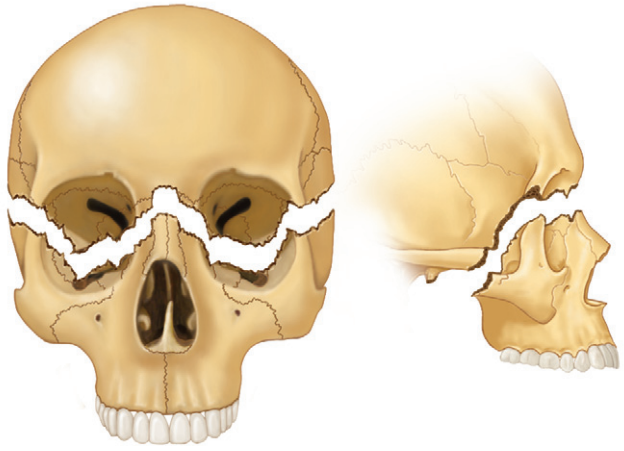


FIGURE 17-3 Le Fort III fracture.

components of the examination and treatment deserve special attention (Fig. 17-5).

The airway is immediately evaluated for obstruction. The oral cavity may be full of secretions or debris, which may contribute to supraglottic obstruction and aspiration. The oropharynx must be manually cleared of any fractured teeth, dentures, and/or blood clots. If stable, the patient may be placed in a lateral decubitus position

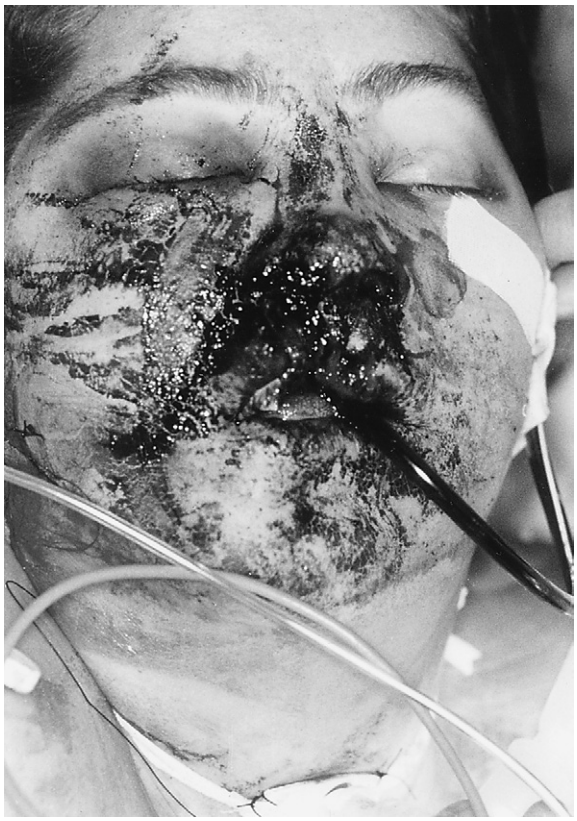


FIGURE 17-4 Compound comminuted midfacial fractures.



FIGURE 17-5 Axial CT reconstruction demonstrating panfacial fracture.

and mild Trendelenburg position to allow optimal drainage. If oral or nasal bleeding is encountered, these sites should be packed.[6] If bleeding is uncontrollable, a definitive airway should be established immediately.

Any patient with facial trauma is presumed to have a cervical spine injury and should be stabilized with a rigid collar until ruled out by appropriate examination. Any motion of the head can be lethal because of bone fragment impingement or laceration of the spinal cord. The presence of cervical spine injuries have been reported as 1.0% to 1.8% to as many as 9.6% of patients with facial fractures.[7-9]

The safety of orotracheal intubation by various techniques in patients with cervical spine injuries has been shown to be safe if precise in-line immobilization is provided.[10] A cricothyroidotomy is also an appropriate option for establishing an emergency definitive airway in this setting.[11] If nonurgent airway control is needed in the setting of cervical spine fracture, an awake fiberoptic intubation is likely the safest option because no atlanto-occipital extension is required with this technique.[12]

A lower airway obstruction, such as laryngeal or tracheal fracture, requires an emergency tracheostomy. Also, if reconstructive procedures of the midfacial injuries will be limited by the presence of an endotracheal tube, a tracheotomy is also indicated.

After definitive airway control is obtained, the ATLS protocol may continue. In regard to midfacial fractures, it is uncommon for hemorrhage from this region alone to result in hemodynamic instability. However, it is important to be cognizant of blood loss, especially if multiple large vessels are violated, large grossly open wounds are present, or continuous blood loss continues from this highly vascular region. As noted, packing of the cavities or wounds with selected clamping of blood vessels is usually effective immediately.



FIGURE 17-6 Extensive facial edema and periorbital ecchymosis.

FACIAL EXAMINATION

After initial stabilization, a complete facial examination is performed. This requires clinical and radiographic components;, however, the radiographic examination may be delayed until the patient's condition is fully stabilized.

The face is systematically evaluated for the presence of any lacerations or obvious deformities of the skull and asymmetries are noted. Otorrhea or rhinorrhea is assumed to be cerebrospinal fluid (CSF) until proved otherwise. No packing of the ear or nose should be performed if a CSF leak is suspected. This practice is potentially helpful for initially preventing a retrograde infection that might result in meningitis.

The craniomaxillofacial skeleton is palpated bimanually in a systematic manner and any discontinuity or irregularity is noted. The frontal area and supraorbital rim is examined first, with a logical progression downward, including the lateral and infraorbital rims, although extensive edema in this area may make the examination difficult.

Periorbital edema and ecchymosis are often initial signs of orbital trauma (Fig. 17-6). The globe may protrude because of gross edema, making a complete examination difficult. Visual acuity is grossly examined and extraocular muscle function evaluated. The presence and direction of diplopia or strabismus are noted. Pupil size, shape, and reaction to light are recorded. The location and extent of subconjunctival hemorrhage are also recorded. A fundoscopic examination is carried out to evaluate for intraocular hemorrhage. If lid lacerations are present, they should be closed promptly to prevent contracture. A through and through laceration requires a three-layer closure. A 6-0 plain absorbing suture may be used on the conjunctiva, with care to prevent protruding knots that may injure the cornea. The orbicularis oculi muscle is closed with 5-0 resorbable suture; the skin is closed with a 6-0 nonresorbable suture. Exact approximation of the gray line of the lid margins is obtained with 6-0 resorbable suture (Fig. 17-7).

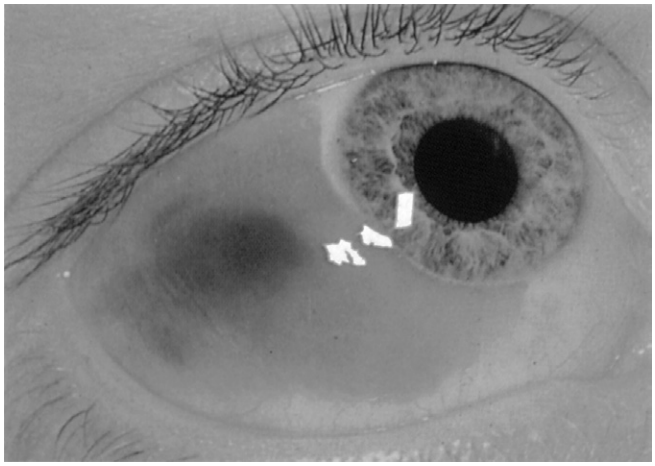


FIGURE 17-7 Subconjunctival ecchymosis with no lateral limit should instill a high index of suspicion for osseous orbital fractures.

Ocular injury is present in most midfacial fractures and is common in midfacial trauma. Al-Qurainy et al, in a prospective study on orbital injury with midfacial fractures of any type, have reported that 90% of patients had an ocular injury to some degree, 16% had moderate ocular injuries, and 12% had severe injuries. Almost 15% of patients had a decrease in vision.[13] In patients with naso-orbital-ethmoid fractures, ocular injury and subsequent loss of vision were reported in 30% of patients.[14] Isolated orbital wall fractures have also been reported to

present with an almost 30% incidence of ophthalmic complications.[15] Current evidence supports the practice that facial fractures involving the orbit should be referred for ophthalmologic evaluation.[13-18]

Crepitation to palpation is indicative of orbital emphysema. This examination finding is most commonly observed when injuries result in communication with the ethmoidal or maxillary sinuses and requires no treatment. The patient should, however, be instructed to not blow his or her nose to prevent expanding subcutaneous emphysema. The attachment of the medial canthal ligament is evaluated by palpating the insertion of the medial canthal ligament for crepitus or instability and by lateral traction on the lateral canthus. Bimanual examination may also be performed by application of a Kelly clamp intranasally and a finger on the central fragment; this is followed by an attempt at lateral displacement of the central fragment.[19] Physical findings of medial canthal ligament disruption include rounding of the lacrimal lake, epiphora, and increased intercanthal distance.

The zygomatic arches, nasal bones, maxilla, and mandible are then sequentially evaluated. Mobility of the maxilla is assessed by firmly grasping the premaxilla and attempting to displace it in three dimensions. Ecchymotic areas, especially of the palate, are common findings with fractures of the maxilla. The pharynx is examined for lacerations or retropharyngeal bleeding. If responsive, the patient should also be questioned about any salty, metallic-tasting discharge, which is an indication of CSF drainage.

The mandibular opening is evaluated for fracture or displacement of the zygoma, which may obstruct the forward movement of the coronoid process. The buccal vestibule is palpated with the index finger and crepitation or displacement of the lateral antral wall; zygoma can be easily appreciated by this maneuver. The occlusion and quality of the dentition are recorded, because these factors may significantly influence in the method of treatment.

Once the patient is sufficiently stabilized, radiographic evaluation may proceed. The preferred radiologic modality for midfacial injuries is a maxillofacial computed tomography scan (CT).[20-22] A maxillofacial CT scan will provide 2- to 3-mm axial cuts with coronal reformatting. If desired, sagittal or three-dimensional formatting may also be provided. The CT scan allows evaluation of bone, providing detailed information about fracture patterns. CT scans also provide characterization of soft tissues, including the extent of edema, presence of foreign bodies, formation of a retrobulbar hematoma, or entrapment of the extraocular musculature. Plain films, although necessary in the absence of CT scanning, provide little diagnostic information. The plain films obtained in the history include the Water's, submentovertex, anteroposterior, and lateral skull views.

As a general principle, early management of these fractures is preferable following stabilization of the patient's condition and diagnosis of midfacial fractures. After 7 to 10 days, it may become more difficult to mobilize the maxilla and achieve an ideal reduction, particularly in patients in whom there is impaction of the fractured segment. In our opinion, in the patient with substantial midface injury, earlier rather than later repair significantly enhances outcome.

The maxilla, palatine bone, and nasal bones form the bulk of the midface. The maxillary bones are integral in the formation of the three major cavities of the face—the upper part of the oral cavity and the nasal and orbital fossa. The maxillary sinus, which is small at birth, expands inferiorly within the maxilla with maturity until it forms the major bulk of the midface. This factor adds to the distinct weakness of the region. Because of the many articulations between the surrounding bones, it is difficult at times to categorize fractures patterns. However, the classic Le Fort I and II classifications of midfacial fractures will be discussed here and the Le Fort type III and naso-orbital-ethmoid (NOE) fracture patterns will be discussed separately (Fig. 17-8).

LE FORT TYPE I FRACTURES

Le Fort type I fractures are caused by a force delivered above the apices of the teeth. The fracture occurs at the level of the piriform aperture and involves the anterior and lateral walls of the maxillary sinus, lateral nasal walls and, by definition, pterygoid plates. The nasal septum may also be fractured and the nasal cartilage may be buckled. Sagittal fracture(s) of the palate may also be present. The pull of the medial and lateral pterygoid muscles may contribute to displacement of the fractured segment in a posterior and inferior direction, resulting in an open bite deformity. This fracture may present as an impacted, immovable, or free-floating maxillary segment.

Le Fort I injuries, on initial examination, may not be clearly evident. Examination should include firmly grasping the maxillary arch with the finger and thumb facially and palatally and attempting displacement of the maxilla in three dimensions, as well as compression and expansion of the maxillary arch. Malocclusion and mobility may be noted. Hypoesthesia of the infraorbital nerve may be caused by the rapid development of edema. A unilateral maxillary fracture may also occur, with the fracture coursing through the palatal suture line or adjacent to it. Palatal ecchymosis is usually noted and may present in conjunction with a malocclusion or displacement of the fractured fragment.

ANATOMIC CONSIDERATIONS OF FRACTURES OF THE MAXILLA

The internal and external pterygoid muscles together have been suspected as being responsible for the posterior and inferior pull seen in fractures of the maxilla. However, unlike the mandible, the midface is more subject to traumatic rather than muscular displacement.

The blood supply to the maxilla is via the internal maxillary arteries. Together with the superior and posterior alveolar arteries, they supply the hard and soft palates. Anteriorly, the nasopalatine artery reaches the incisive foramen and supplies the mucoperiosteum of the anterior palate.

Neurosensory supply is via the second division of the trigeminal nerve. This nerve exits the infraorbital foramen and supplies the lateral nasal, superior labial,

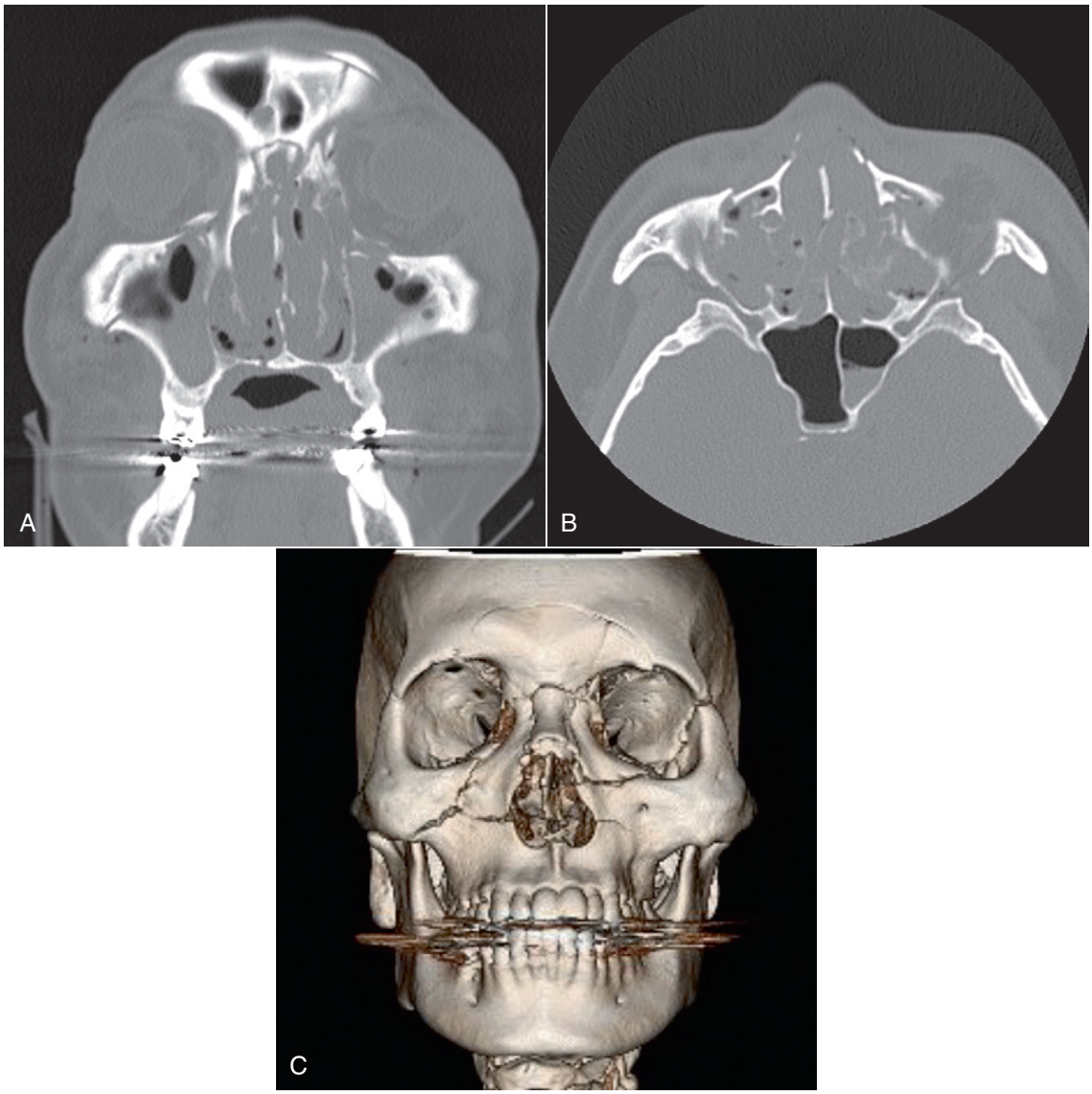


FIGURE 17-8 CT scans in the appropriate view can stand alone as a diagnostic radiographic tool. **A,** Coronal. **B,** Axial. **C,** Three-dimensional.

and inferior palpebral regions, as well as the labial mucosa and anterior teeth.

TREATMENT OF UNILATERAL MAXILLARY FRACTURES

The fractured segment is reduced by digital pressure and a maxillary arch bar is applied loosely to the teeth in the mobile segment and firmly to the stable dentition in the unfractured maxillary segment. MMF is then applied between the maxillary and mandibular arch bars and the reestablished pretraumatic occlusion is used to reduce the mobile maxillary segment. Open reduction and internal fixation by miniplates are completed through a vestibular incision and MMF is removed. The patient is kept on a soft diet for 2 to 3 weeks while the fractures heal. MMF may also be left in place if there is concern for patient compliance.

An occlusal splint is an excellent option for accurate reduction of the fractured maxillary segment. Alginate impressions are taken of the maxillary and mandibular arches and the correct occlusal relationships are reestablished on study models. An interocclusal splint is then constructed. Arch bars may be placed before or after placement of the interocclusal splint, depending on the mobility of the maxillary segments. The maxillary arch bar is secured by interdental fixation and the interocclusal splint is secured to the maxillary arch bar. The mandible is then passively guided into the interocclusal splint. MMF may be used to stabilize the maxillary fragments during open reduction and internal fixation (ORIF), or may be used as a method of fixation if ORIF is not desired, but will require a 3- to 4-week period of MMF.

Impacted maxillary fractures may be impossible to mobilize with digital manipulation alone. A disimpaction forceps may be used in this situation for reduction of the impacted maxillary segment. Teeth in the line of fracture should be left in place unless excessively mobile or hopelessly nonrestorable.

TREATMENT OF LE FORT TYPE I FRACTURES

Early reduction of Le Fort type I injuries presents minimal difficulty but, beyond 7 to 10 days, increasing amounts of force are required because of the natural healing process. ORIF with restoration of facial contour is the preferred method of treatment. MMF is also an acceptable, although less optimal, method of treatment. This requires a treatment period of approximately 6 weeks, depending on the level of comminution.

Incisions for the open reduction are made in the buccal vestibule in a circumvestibular fashion, from the first premolar to the first premolar on the opposite side. Wide buccal pedicles of the U-shaped incision are retained for maintenance of the vascular supply. This approach allows visualization of the lateral antral wall and zygomatic buttresses. A Rowe or Hayton-Williams forceps can then be used to complete the reduction, if necessary (Figs. 17-9 and 17-10).

The advent of plate and screw fixation has transformed craniomaxillofacial fracture repair from obligatory long-term MMF and craniofacial suspension to rigid stabilization. Rigid stabilization provides an opportunity for primary bone healing and allows earlier function and optimized nutrition.

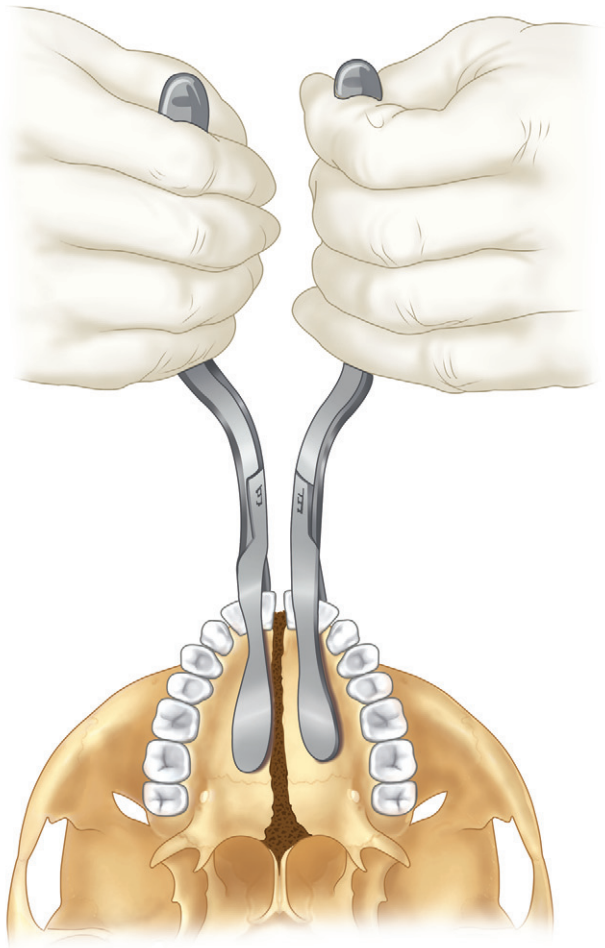


FIGURE 17-9 A Rowe disimpaction forceps is used when attempting to reduce a displaced maxilla.

The patient is first placed in MMF to reestablish the pretraumatic occlusal relationship. The maxillomandibular complex can then be oriented in three dimensions as a unit, with special attention to the position of the condyles. Ensuring that the condyles are seated properly in the glenoid fossa will limit subsequent development of an open bite. Stabilization of a fracture must prevent translational and rotational motions in the x, y, and z axes.[23] Four-point fixation along the pyriform and zygomaticomaxillary buttresses is routinely provided for stability of this fracture pattern. Occlusion should be immediately rechecked following release of MMF (Fig. 17-11).

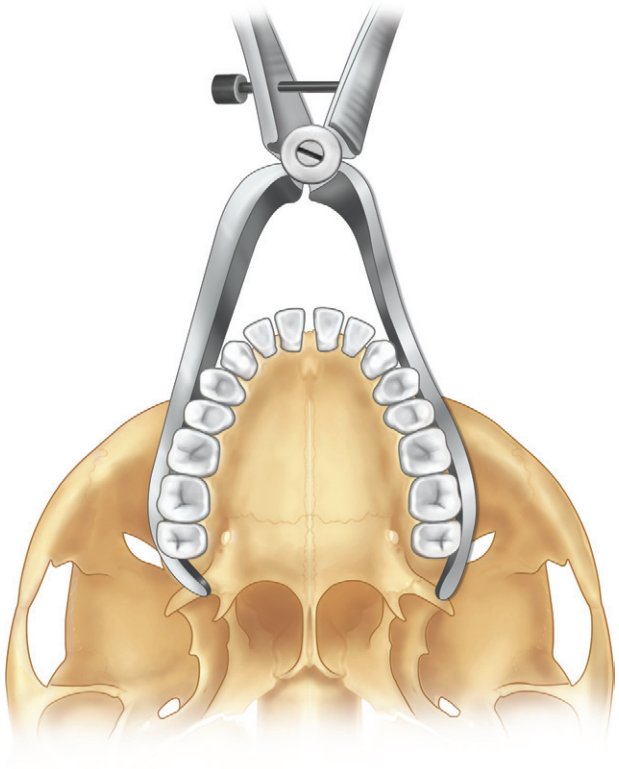


FIGURE 17-10 Positioning of the Hayton-Williams forceps when attempting to reduce a fractured maxilla.

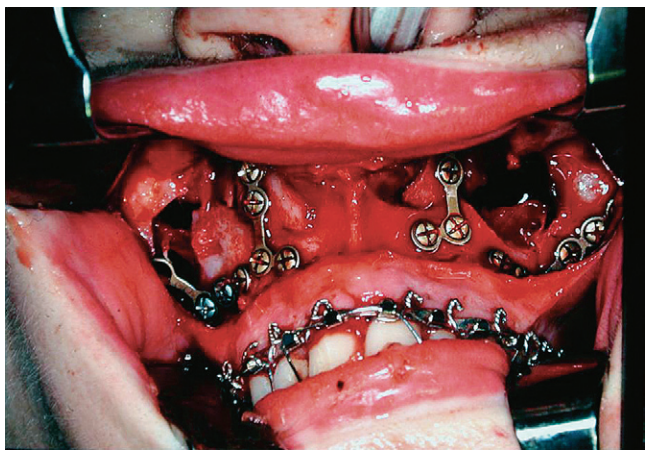


FIGURE 17-11 Four-point fixation—stabilization of the midfacial buttresses.

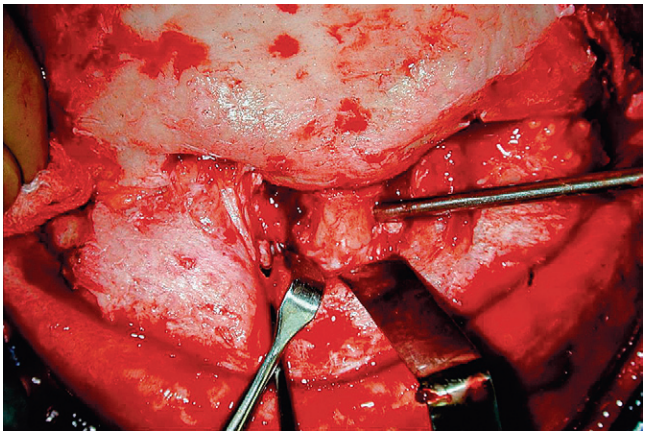


FIGURE 17-12 Le Fort II fracture.

LE FORT TYPE II (PYRAMIDAL) FRACTURES

The Le Fort type II fracture pattern is also referred to as a pyramidal fracture; the apex of the pyramid is the nasofrontal suture (Fig. 17-12). This fracture pattern involves the nasofrontal suture, nasal and lacrimal bones, infraorbital rim in the region of the zygomaticomaxillary suture, maxilla, and pterygoid plates. This fracture is typically higher than the Le Fort type I fracture posteriorly. As with the Le Fort type I pattern, the nasal septum may also be involved.

The physical examination is likely to reveal noticeable signs of injury. Edema is often present overlying the fracture sites. The classic raccoon sign caused by bilateral periorbital edema and ecchymosis may be noted. CSF rhinorrhea may be encountered as the result of a dural tear. Epistaxis is common. Hypoesthesia of the infraorbital nerve is also common because of direct trauma or rapid edema formation. Malocclusion is often present in the form of an anterior open bite. Grasping the anterior maxilla and attempting anteroposterior displacement facilitates evaluation of the nasofrontal suture and inferior orbital rims.

TREATMENT

ORIF is advantageous for treatment of these fractures. If the nasofrontal suture area is intact and continuous with the maxillary segment, bilateral intraoral exposure allows appropriate four-point fixation. However, the orbital floor, inferior orbital rim, or nasofrontal region often requires exploration and repair. In these situations, additional access is required. The basic incisions are the infraorbital, subciliary, middle to lower lid, and transconjunctival incisions. These methods of access will be briefly discussed.

Infraorbital Incision

The infraorbital incision, first described by Converse et al in 1944,[24,25] is made transcutaneously over the infraorbital rim in the natural crease, 4.5 mm inferior to the gray line. Advantages include excellent access to the floor and surrounding areas and ease of use. Disadvantages are potential poor healing, cicatricial distortion of the lower lid, and potential for development of lymphedema. More than 90% of the lymphatic drainage from the scalp, forehead, and upper lid passes lateral to the orbit.

Subciliary or Lower Blepharoplasty Incision

The subciliary or lower blepharoplasty incision is made 2 to 3 mm inferior to the gray line of the lower eyelid. The incision runs parallel to and along the length of the lower eyelid margin. The traditional method of pre–orbicularis oculi incision has fallen out of favor because of high rates of ectropion development, 38% to 42% temporary and 8% permanent.[26] The post–orbicularis oculi dissection, or skin-muscle flap, is when the incision is made through both skin and muscle and dissection occurs superficial to septum. This modification has become popular because of ease of use and has been shown to have a lower rate of early ectropion, 6% in one study, with resolution typically within weeks.[27]

Subtarsal or Mid–Lower Lid Incision

The incision is made 3 to 4 mm below the gray line. Dissection is the same as the subciliary. Use of this incision has been suggested to limit lower lid distortion and ectropion.

Transconjunctival Incision

The transconjunctival approach was popularized by Tessier in 1973.[28] This approach is made through the conjunctiva parallel to the gray line and can be made anterior or posterior to the septum.[29] The incision can also be extended laterally by lateral canthotomy and inferior cantholysis and medially by a transcaruncular incision to allow excellent access to the orbit (Fig. 17-13).

Access should be carefully planned on an individualized basis. Depending on the situation, various approaches are available and lacerations may be used. When multiple sites are affected, a coronal incision provides excellent exposure as well as access to cranial bone for bone grafting.

PALATAL FRACTURES

Isolated fractures of the palate are rare, but up to 8% to 13% of Le Fort fractures are complicated by concomitant palatal fractures.[30,31] Most patients will also have notable signs and symptoms of palatal fracture. Indications of palatal fracture on clinical examination include laceration of the lip and concurrent gingival and palatal lacerations. Often, a change in occlusion is also noted with the maxillary segment displaced anterolaterally.[3] Diagnosis is confirmed by a maxillofacial CT with axial and coronal cuts.

CLASSIFICATION

Several classification systems have been suggested[30,31] for palatal fractures. Hendrickson et al[30] have described six patterns based on the anatomic location of fracture (Fig. 17-14; Box 17-1).

Treatment

Surgical treatment planning depends on the type of fracture, presence or quality of the dentition, and

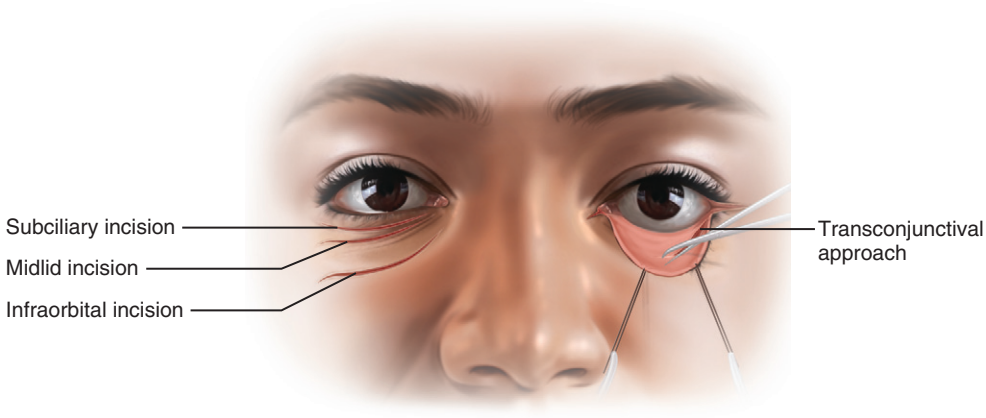


FIGURE 17-13 Various approaches to the orbit—subciliary, midlid, transconjunctival, and open sky, or use of existing laceration.

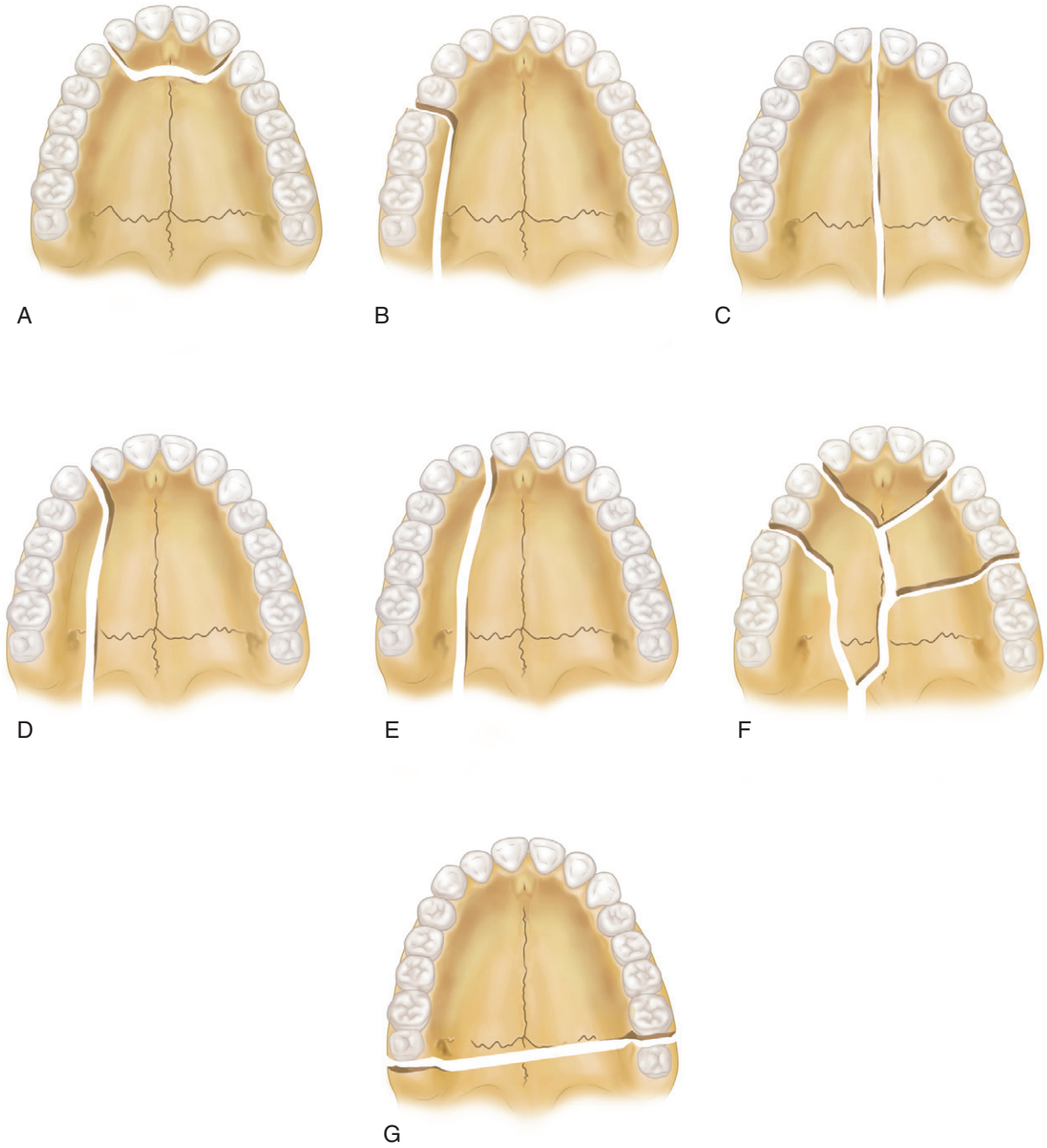


FIGURE 17-14 Hendrickson palatal classification system based on anatomic location of fracture.

BOX 17-1 Palatal Fracture Classifications

Type I: Alveolar fracture
Type Ia: Anterior alveolus; contains only incisor teeth and associated alveolus
Type Ib: Posterolateral; contains premolars, molars, and associated alveolus
Type II: Sagittal fracture, a split of the palatal midline; typically occurs in second or third decade because of a lack of ossification of the midline palatal suture
Type III: Parasagittal fracture; most common fracture pattern in adults (63%) because of thin bone parasagittally; fracture pattern differs from type Ia fracture by inclusion of maxillary canine
Type IV: Para-alveolar fracture; occurs palatal to the maxillary alveolus and incisors
Type V: Complex comminuted fracture; multiple fractured segments
Type VI: Transverse fracture, rare; involves a division in the coronal plane

concomitant facial fractures. Treatment incorporates the possible application of rigid internal fixation, arch bars, and palatal acrylic splints, depending on the clinical situation.[3,30-33]

Although there has been much discussion on the use of rigid fixation in palatal vault fractures, it is seldom clinically indicated. The first problem with this technique originates with the placement of transoral plates and screws to the palatal vault. This practice requires the mouth to be open and thereby excludes the application of ORIF and occlusal stabilization. This lack of occlusal control leads almost inescapably to error in the appropriate reduction of maxillary width and height. The second consideration is vascularization of the maxilla. Although techniques for making longitudinal palatal incisions are often described, vascular studies of the Le Fort I segment following osteotomy have shown that the blood flow in the severely fractured or osteotomized maxilla is dependent on the ascending pharyngeal and palatal mucosal blood supply.[34] Because management of midfacial injury often require an anterior vestibular incision, if treated open, any palatal flap raised under these circumstances must be approached with caution.

Treatment of the palatal fracture in dentate patients should center on occlusal reduction with MMF and a facial vestibular approach. Incorporation of occlusal splints can be extremely helpful in the comminuted palatal fracture and requires preoperative dental models to fabricate. The use of a palatal splint should be approached with caution. As noted, care must be taken to ensure that the palatal vascular supply is not compromised. In addition, postoperative surveillance of the palate for healing, fistula development, or necrosis of segments becomes more difficult because direct inspection of the palatal vault is obscured by the splint. If a concomitant mandibular fracture exists, open reduction and anatomic fixation of this injury first will allow the treatment of the palatal fracture to proceed with the appropriate occlusal template.



FIGURE 17-15 Traumatic telecanthus following NOE injury showing measurement of intercanthal and interpupillary width.

An indication for the use of rigid fixation for sagittal palatal fractures in the edentulous or near-edentulous patient exists if the patient does not have preexisting dentures or no preoperative Gunning-type splint was fabricated. In these rare patients, special care must be taken to reduce the fractures as anatomically as possible from the facial vestibular approach. Additional stabilization of the palate can then be gained from transmucosal locking plate and screw fixation, as described by Pollock, which attempts to preserve the critical vascular supply.[35]

NASO-ORBITAL-ETHMOID INJURIES

The naso-orbital-ethmoid (NOE) injury, often referred to as an NOE fracture, represents a significant diagnostic and reconstructive challenge.[36,37] This region houses the lacrimal apparatus, medial canthal ligament, and anterior ethmoidal artery. CSF rhinorrhea is common following NOE fractures.[38] In one series, Cruse et al have reported that central nervous injuries are present in 51% of cases and 42% have CSF drainage[14] (Fig. 17-15).

Assessment of these injuries requires close attention to the soft tissue and osseous structures and an accompanying CT scan, with both coronal and axial views.[39] Physical examination is likely to demonstrate a severely fractured nose, often with comminution and posterior displacement. The nasal bridge is widened and the nasal complex splayed. Epistaxis is common. Traumatic telecanthus (see Fig. 17-15) may occur because of disruption of the medial canthal ligament. The average intercanthal distance for a white adult is 28 to 35 mm, which is approximately half of the interpupillary distance. Halving the interpupillary distance is a useful tool on the preliminary physical examination because severe periorbital edema is preset in most cases. Traumatic telecanthus is suspected when the intercanthal distance is greater than 35 mm; a measurement more that 40 mm is diagnostic for this type of injury.[40] Epiphora following trauma to this area is likely the result of damage to the

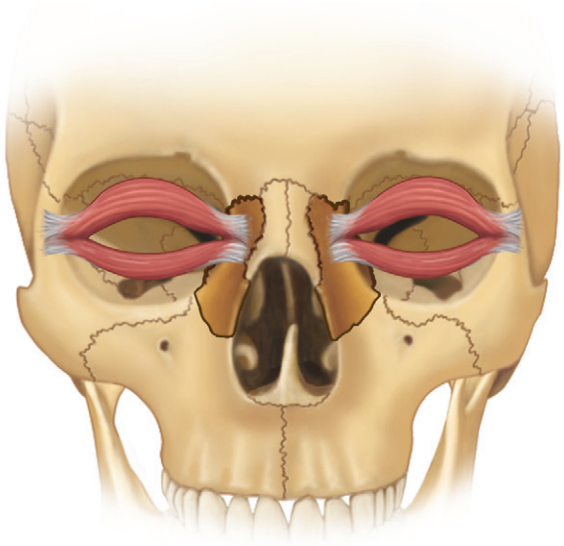


FIGURE 17-16 The central nasoethmoid complex is highlighted for identification.

lacrimal apparatus. Narrowing of the palpebral fissure, obliteration of the caruncle, and flatting of the base of the naso-orbital valley are more likely to be noted following the resolution of edema.

NOE fracture repair requires a broad knowledge of anatomy and should be based on anatomic reconstruction; the repair should be done early, when possible. Inadequate reconstruction or delay in treatment may result in a multitude of suboptimal results, generally including midface retrusion, blunted palpebral fissures, ocular complications, nasal deformities, and cerebrospinal fistula formation.[14,41-44]

These injuries may occur with other midfacial fractures, may be isolated or bilateral, and may have different patterns on either side of the midline, depending on the mechanism and velocity of impact.[40,44-49] Repair of these fractures requires surgical approaches that provide wide exposure and allow an anatomic repair. Attention to the anatomy of this region, in particular the intercanthal distance, is essential to a satisfactory outcome.

ANATOMY

The NOE region is made up of the cranium, nose, orbit, and maxilla (Fig. 17-16). The frontomaxillary buttress provides structural support to this region and serves as the stabilization point for reconstruction. The associated lateral buttresses are the frontal bar superiorly and the zygoma and inferior orbital rims inferiorly. The medial portion of the buttress contains the perpendicular plate of the ethmoid, lacrimal ones, and lamina papyracea. These bones are thin, fragile, and form a so-called crumple zone that is predisposed to medially displaced comminuted fractures[46] Reconstruction of the bony architecture is necessary in this area to control orbital volume.

The nasal bones are anterior to the medial orbit and connect to the frontal bone superolaterally and the lacrimal bone and frontal process of the maxilla medially. The ethmoid sinuses, located centrally and posterior to the nasal complex, are also vulnerable to injury. Because of the proximity of the ethmoid sinuses to the cribriform plate, the collapse and posterior displacement of these structures may result in intracranial involvement and potential for CSF rhinorrhea, pneumocephalus, and olfactory dysfunction. Fractures of the frontal sinus are also common because the floor of the frontal sinus is a component of the medial orbital roof. Ductal drainage leaves the frontal sinus inferoposteriorly, travels through the ethmoid bone, and exits at the middle meatus of the nose. Complications because of disruption of this system may be noted following NOE fractures.

The medial canthal tendon is a fibrous extension of the tarsal plates. It divides into two limbs, anterior and posterior. The anterior limb is larger and stronger, attaches to the frontal process of the maxilla, and functionally pulls the medial commissure of the eyelid forward and down.[50] The posterior or deep limb is comparatively thinner, inserts into the posterior lacrimal crest of the lacrimal bone, and functions to maintain the eyelid's position in relation to the globe. The posterior limb is also intimately associated with Horner's muscle, which is responsible for the flow of tears through the lacrimal sac. A superior limb develops from an extension of fibers from the anterior and posterior limbs, which serves to encompass the lacrimal sac and functions to add the posterior and superior vector of the medial canthal tendon. The normal anatomic position of the medial canthal tendon is responsible for medial eyelid function, position, appearance, and lacrimal drainage. The degree of displacement varies significantly; the method of repair depends on whether an injury results in complete avulsion or continued attachment to variously sized bony fragments. In any situation, precise reconstruction of the osseous components and medial reattachment or stabilization of the medial canthal tendon are required to prevent telecanthus, enophthalmos, and dysfunction of the lacrimal system. When this condition is improperly managed on initial reconstruction, the ensuing deformity can be debilitating and extremely difficult to manage secondarily.

DIAGNOSIS

Assessment of these injuries requires close attention to the soft tissue and osseous structures. An accompanying CT scan should be obtained, with both coronal and axial views.[39]

PHYSICAL EXAMINATION

A thorough visual inspection is followed by manual palpation of the supraorbital rims, nasofrontal junctions, lateral nasal complex, inferior orbital rims, and nasal complex as a unit. Mobilization of the nasal complex and its relationship to the nasofrontal junction are examined. Mobilization of the nasal complex in three dimensions is attempted to determine mobility, possible osseous impaction, and the extent of the fracture.

The attachment of the medial canthal ligament is evaluated by palpating the insertion of the medial canthal

ligament for crepitus or instability and by lateral traction on the lateral canthus, with evaluation of the medial canthal attachment. Bimanual examination may also be performed by application of a Kelly clamp intranasally and a finger placed externally on the central fragment; lateral displacement is then attempted.[19] Indications of medial canthal ligament disruption include rounding of the lacrimal lake, epiphora, and increased intercanthal distance.

Measurement of the intercanthal and interpupillary distance is also completed at this time and compared with normal individuals of white, black, and Asian ethnicity to determine whether displacement is present and if it is unilateral or bilateral. Another excellent method of determining pretraumatic intercanthal width is to obtain preinjury photographs.[51] Periorbital edema and ecchymosis will be present and must be taken into consideration when completing this examination.

Nasal tip position and telescoping of the nasal cartilage under the osseous segments of the nasal complex must also be determined. This is important for later reconstruction. A complete intranasal examination may be assisted by applying cocaine or oxymetazoline to shrink soft tissue and performing a careful visual examination of the nasal septum, concha, nasal mucosa, and intranasal position of osseous structures. The presence of CSF rhinorrhea can be determined with this examination; this is an indication of the extent of the injury into the ethmoidal sinuses and to the cranial base by way of the cribriform plate.

Imaging

Axial and coronal CT scanning are required in any patient suspected of having NOE injuries; 1.5- to 2-mm cuts are usually satisfactory for determining the extent of this injury. Correlation of the clinical and radiographic examination facilitates a proper diagnosis. Attention to anatomic structures in regard to location, displacement, size, and comminution is critical. Treatment outcome is based largely on proper identification of the bony segment and the status of the attachment of the medial canthal tissue.

CLASSIFICATION

A commonly used classification system developed by Markowitz et al identified NOE fractures based on their relationship to the central fragment at the site of medial canthal tendon attachment.[52] The fractures are typically noted to be unilateral, bilateral, simple, or comminuted and are likely to have with different fracture presentations bilaterally. They may occur as an isolated injury or in conjunction with other major facial fractures.

Type I

The simplest form of NOE fracture involves only one portion of the medial orbital rim, with its attached medial canthal tendon. It may occur in a bilateral or unilateral form. When the bilateral complete type I fracture occurs, there is no medial canthal tendon displacement and transnasal wiring is not required. Stabilization of the osseous segment is all that is usually necessary (Fig. 17-17*A*).

Type II

Type II NOE fractures may occur in a bilateral or unilateral form and may be large segments or comminuted. Most commonly the canthus remains attached to a large central segment. Reduction is usually best accomplished by control of the specific segment of bone that is associated with the canthal tendon (see Fig. 17-17*B*).

Type III

This fracture includes comminution involving the central fragment of bone where the medial canthal tendon attaches. The canthus is rarely avulsed completely but, on occasion, the fragments of bone are so small that reconstruction is not possible. In this circumstance, transnasal wiring of the canthus is required, as is osseous reconstruction (see Fig. 17-17*C*).

Variants of type I, II, and III fractures may occur on one side or the other in conjunction with each other. In any case, the type of injury and its severity will guide treatment.

TREATMENT

Treatment of the NOE fracture begins with a specific diagnosis and careful treatment planning. The fundamentals of surgical repair are early surgical intervention, wide exposure, careful anatomic osseous reduction, and internal rigid fixation. Special attention to the medial canthal tendon and its attachment is necessary to obtain optimal aesthetic results.

Surgical Approaches

The coronal incision is used most frequently in the management of fractures of the NOE region. This incision provides wide exposure of the superior and medial orbital compartments. The nasal bones are also evaluated and fractured segments removed for later use in reconstruction. Care should be taken to limit disruption of the nasal lacrimal duct and lacrimal sac and the inferior extent of this incision. Determination of nasal lacrimal duct patency will be discussed later in this chapter. A lower eyelid incision may also be necessary to gain access to the inferomedial orbital components. Further stabilization may be necessary by an internal approach to gain access to the nasomaxillary buttress for stabilization of the inferior portion of the NOE complex.

Systematic Management

Systematic treatment of NOE fractures is necessary for a predictable outcome. A systematic approach of eight key steps in the sequencing of NOE fractures was described by Ellis.[37] These steps include surgical exposure, identification of the medial canthal tendon and tendon-bearing bone fragment, reduction and reconstruction of the medial orbital rim, reconstruction of the medial orbital wall, transnasal canthopexy, reduction of septal fractures, nasal dorsum reconstruction and augmentation, and soft tissue adaptation.

The type I fracture is best managed by three-point rigid fixation—reestablishing the relationships of the nasofrontal junction to the nasal complex, the nasal complex to the maxillary buttress, and the nasal complex to the infraorbital rim (Figs. 17-18 and 17-19). As noted,

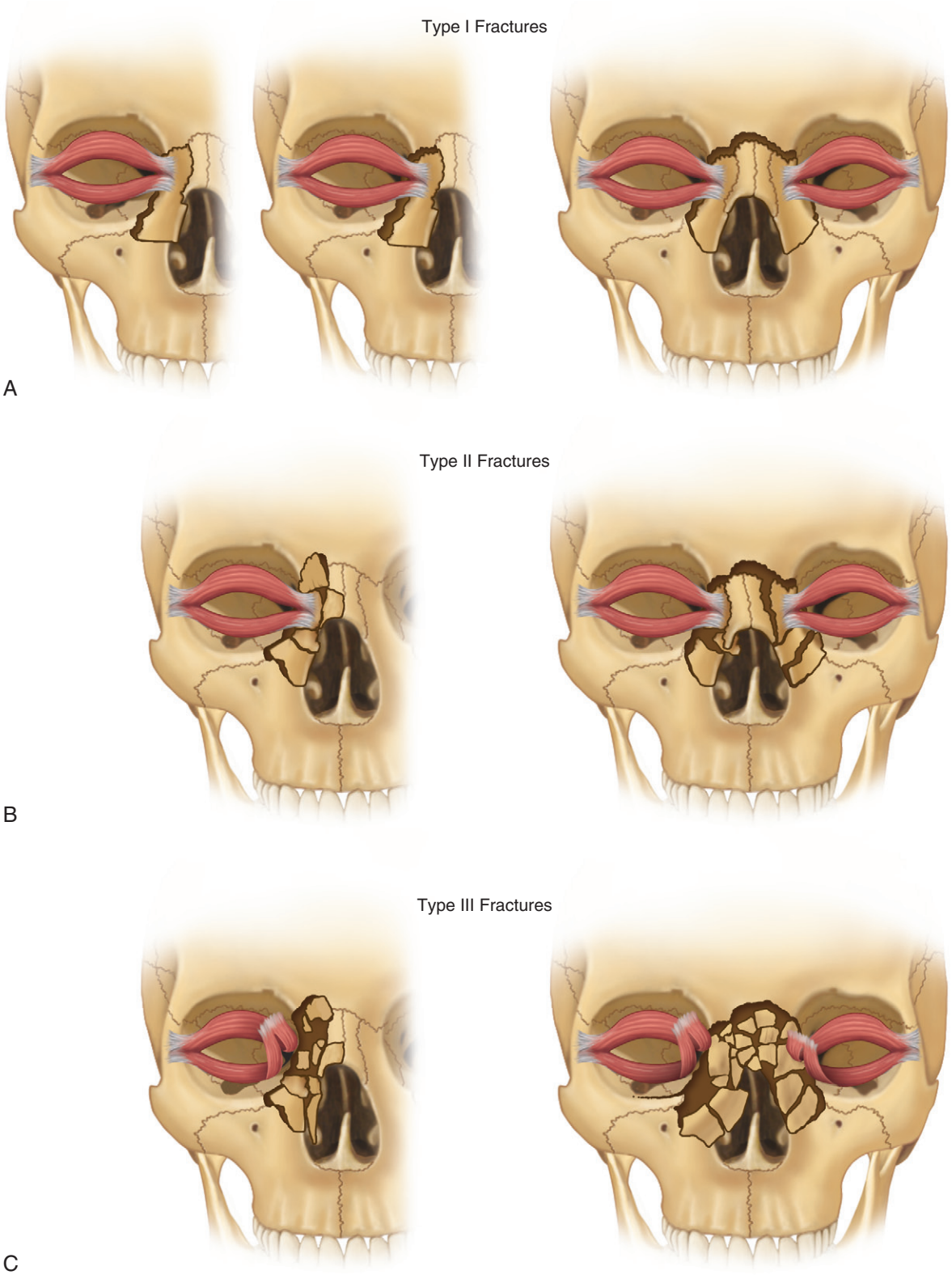


FIGURE 17-17 **A,** Incomplete type I fracture. **B,** Complete unilateral and bilateral type II fractures. **C,** Type III comminuted fracture involving attachment of the canthal tendon.

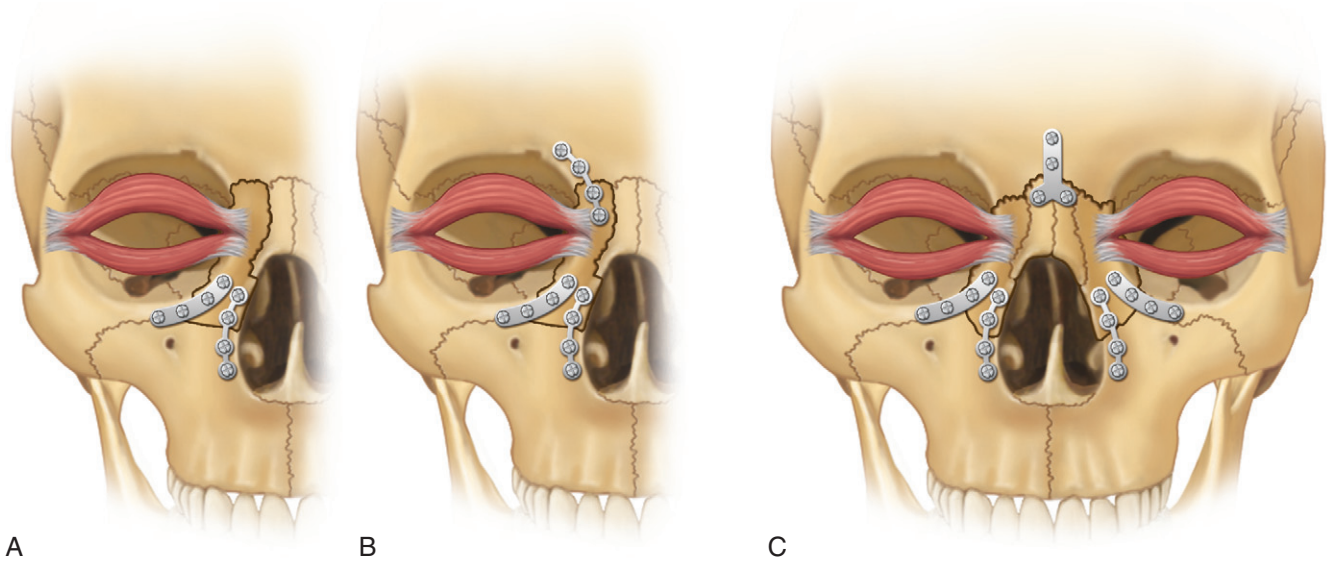


FIGURE 17-18 **A,** Basic fixation treatment of unilateral fracture requiring three-point fixation. **B,** Rigid plate fixation of unilateral fracture requiring three-point stabilization. **C,** Bilateral single-segment injuries with superior and inferior rigid fixation approaches.

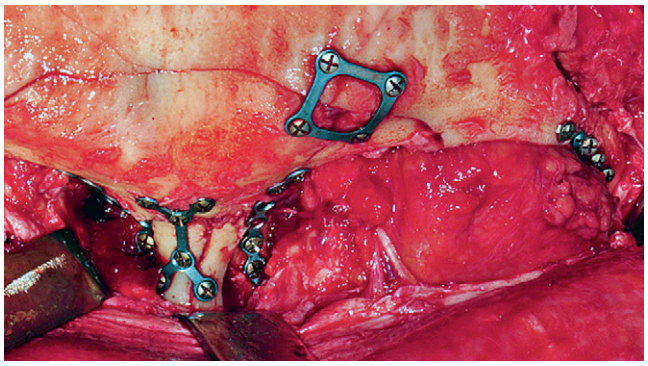


FIGURE 17-19 Three-point stabilization of bilateral single segment injuries via coronal approach.

in this type of injury, medial canthal tendon detachment is rare.

Type II fractures require a more extensive superoinferior approach because a degree of comminution is present by definition. Access through the coronal flap and infraorbital and intraoral incisions are often necessary. The small bony fragments must first be anatomically reduced with 28- to 30-gauge intraosseous wiring or with microplates. Rigid internal fixation then proceeds as for a type I fracture to reestablish the bony buttresses. Reattachment of the medial canthal tendon is then completed de facto by appropriate reduction of the large central fragment(s) to which the tendon is attached.

The type III NOE fracture involves extensive comminution and displacement of the osseous structures, with apparent avulsion of the medial canthal tendon unilaterally or bilaterally from its osseous attachment. The principles of access and repair follow the same principles as for type I and II fractures, although establishing pretraumatic osseous and soft tissue contour is more challenging because of the level of comminution. Severe comminution of the nasal region may require a dorsal nasal cantilever-type bone graft to reestablish dorsal nasal support and nasal tip projection. This bone graft can be placed through the coronal incision and stabilized with intraosseous screws into the nasal process of the frontal bone (Fig. 17-20). Cranial bone grafts can be used for this technique and may also be needed for reconstruction of the facial buttresses. Other autogenous sites recommended for the procedure include the rib, ilium, and mandible.[53] Canthoplasty, unilateral or bilateral, may be required. Direct transnasal wiring of the medial canthal tendon in a position slightly posterior and superior to the normal anatomic position will help overcome the forces of migration, relapse, and telecanthus (Fig. 17-21).

Transnasal Wiring Technique

Transnasal wiring or transnasal medial canthopexy is performed when necessary. This step follows surgical exposure, identification of the medial canthal tendon or tendon-bearing bone fragment, reconstruction of the medial orbital rim, and reconstruction of the medial orbital wall. This sequence of repair is important because reconstruction of the medial orbital rim is key to reestablishing the pretraumatic intercanthal distance,[52] and reconstruction of the medial orbital wall is required to maintain an appropriate orbital volume.[54-56]

A small incision is made approximately 3 mm medial to the medial canthus and blunt dissection is used to identify the fibers of the medial canthal tendon. Once the tendon is identified, a hole is made through the unstable central fragment and through bone on the contralateral side, one hole just posterior and superior to the lacrimal crest and the other superior to the lacrimal fossa. A double 26-, 28-, or 30-gauge wire is then threaded through the two holes transnasally with a curved needle or awl. The wire is secured to a screw or small plate on the contralateral supraorbital rim. This orientation provides the posteriorly and superiorly directed pull typically

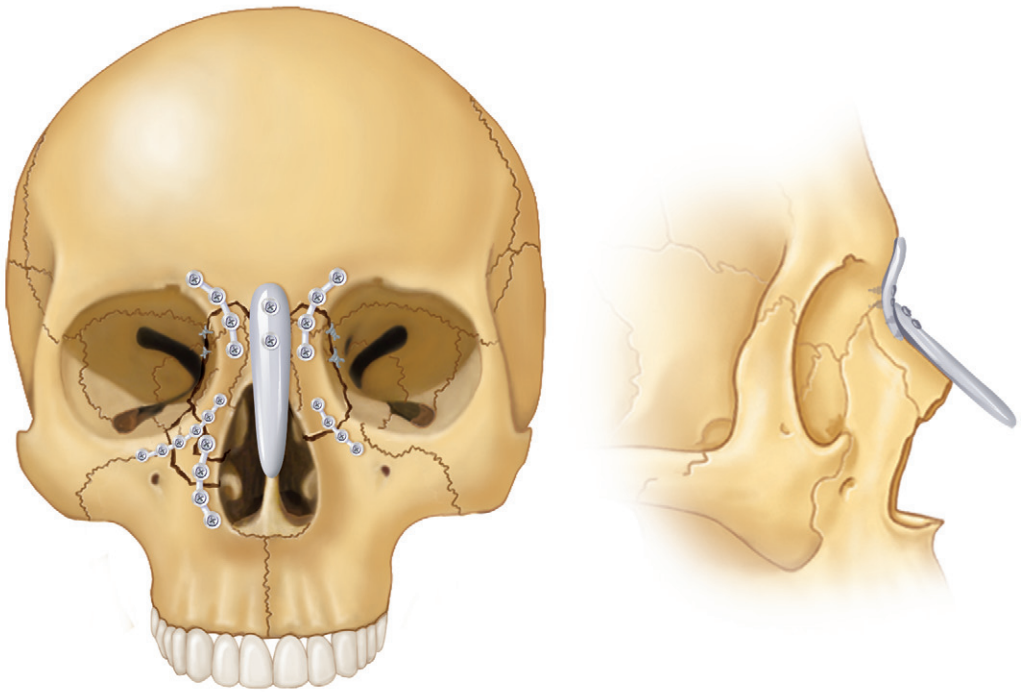


FIGURE 17-20 Comminuted NOE fracture with wire and plate fixation and dorsal basal bone graft placed with lag screw technique.

exerted by the medial canthal tendon. The same procedure may be used when the medial canthal tendon is completely detached from the central fragment or, if the traumatic disruption prevents identification of the ligaments, the tissue surrounding the tendon may be used in a similar manner (Fig. 17-22). Transnasal wires should be intentionally overreduced to prevent notable telecanthus and widening of the nasal dorsum.[40]

If a bilateral canthopexy is required, a separate mattress wire is sutured through one tendon and the wire is passed transnasally to be secured to the contralateral supraorbital rim by a screw or small plate. Both canthopexies are carried out in the same manner but should not be secured to each other; this is to prevent a complete loss of anatomic position if one side becomes loose.[37]

At the conclusion of these approaches for nasal complex reconstruction, final repositioning of the nasal bones is accomplished with Asch forceps and intranasal splints. External splinting can be beneficial, even to the extent of providing nasal support for proper soft tissue draping and reestablishing the correct adaptation of soft tissue into the medial canthus area.

LE FORT TYPE III FRACTURES

Because of the complex nature of midfacial injures, classification is often difficult; fractures classified as Le Fort III may actually be combinations of Le Fort I and II and zygomatic complex fractures.[57] However, in one series, pure Le Fort III fractures accounted for approximately 9% of midfacial fractures.[58]

The Le Fort III fracture pattern is a craniofacial dysjunction. Symptoms include a classic dish face deformity and mobility of the zygomaticomaxillary complex. CSF leakage, edema, periorbital ecchymosis, traumatic telecanthus, and epiphora may be observed. The clinical examination can be supplemented and diagnosis confirmed by CT, with coronal, axial, and sagittal views (Fig. 17-23). Plain films are suboptimal in this setting.

TREATMENT

There is some controversy regarding the optimal timing of the repair of midfacial injuries, particularly when significant edema is present. As a general principle, treatment should begin once the edema from the initial insult has begun to subside but should not be delayed for more than 10 to 14 days.[37] In our experience, earlier repair generally provides a more optimal, long-term soft tissue drape. However, concomitant neurosurgical trauma and other issues may delay repair beyond the surgeon's control. The Le Fort III fracture is essentially a complicated combination of bilateral zygoma and NOE fractures, and the same principles apply when treating this fracture pattern.

There are two general schools of thought regarding the sequence of repair. Gruss et al have proposed a method of reconstruction whereby reconstruction begins with the outer framework and progresses to the inward facial structures, from stable to unstable areas.[59,60] Repair begins with rigid fixation of the mandibular fractures. In the case of a bilateral mandibular condyle fracture, at least one condyle (ideally both) must be treated by ORIF. Once that is accomplished, the maxilla can assume its correct superior position and anteroposterior location. A stable outer framework of the midface is established with reduction and fixation of the zygomaticofrontal, zygomaticotemporal, and nasofrontal sutures and the appropriate reduction of the maxilla to the midface inferiorly. In turn, the proper occlusion must be established

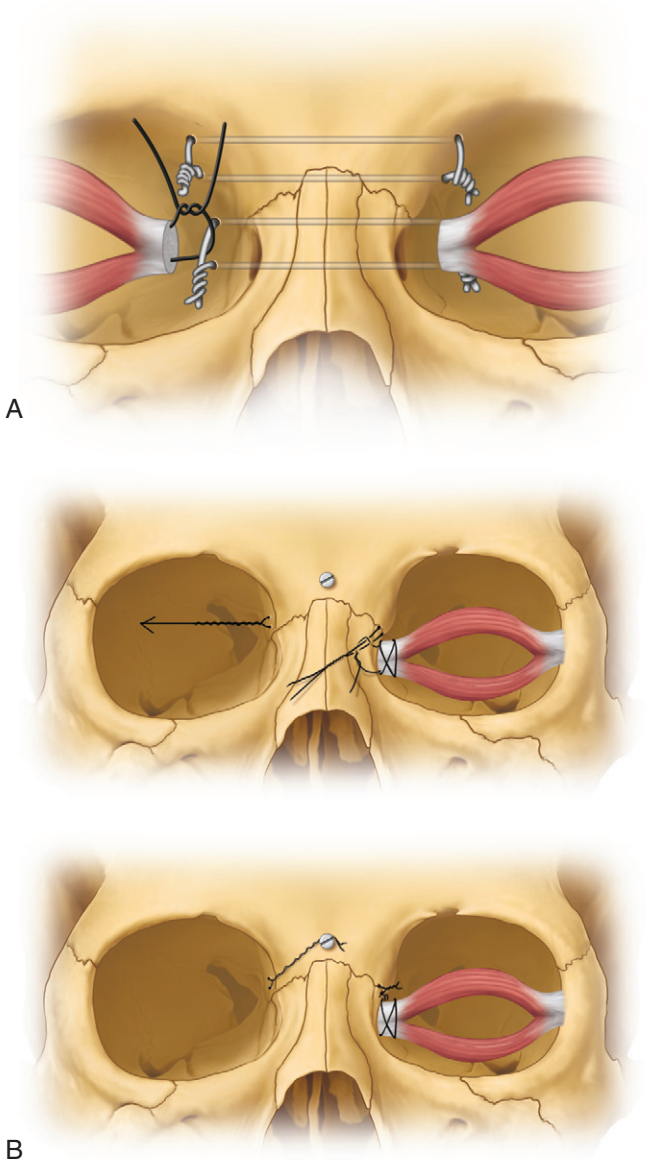


FIGURE 17-21 **A,** Wire support stabilization of avulsed medial canthal tendons. **B,** Transnasal reduction of medial canthal tendon with transnasal wire fixation to raise tendon superiorly and posteriorly.

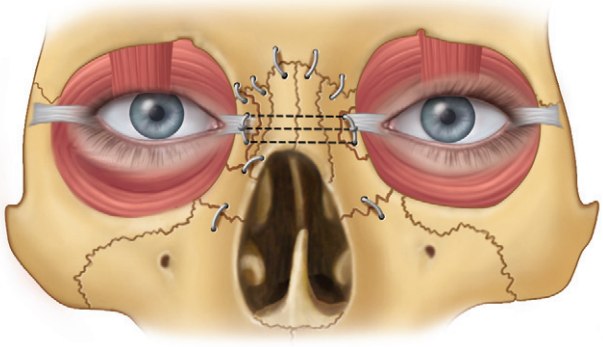


FIGURE 17-22 Wiring technique for an NOE fracture.



FIGURE 17-23 Axial CT of a Le Fort III fracture.

to complete the anteroposterior and lateral positioning of the midface. Once the outer framework has been established, one can proceed to reconstruct, in order, the nasal skeleton and floors of the orbits, correct any lacrimal system disorders, and reestablish the proper positioning of the medial canthal ligaments. Therefore, one works from the lateral superior and inferior direction medially to correct the Le Fort type III deformity.

A second school of thought, popularized by Markowitz and Manson,[2] focused on reestablishing facial width at the NOE complex and proceeding in laterally. A method described by Kelly and Manson outlines this approach.[61]

Marciani and Gonty have summarized the four factors contributing to positive outcomes following reconstruction of craniomaxillofacial trauma. These are early definitive treatment, anatomic and functional repair of NOE injuries, wide exposure of fractured segments, and anatomic repositioning and stable fixation in all planes.[62] It must be emphasized that there are a number of effective ways to approach these complex types of facial injuries. Thoughtful review of the different viewpoints regarding the management of these injuries has revealed that there is no one specific method that will definitively manage all types of injuries. Focused preoperative consideration of the injury pattern, with special consideration to areas of stability that will assist in establishing a stable framework, are likely to yield the best results.

Surgical Approaches

The coronal approach allows excellent exposure of the NOE complex, lateral orbital rims, and zygomatic arch. It is aesthetically acceptable because the incision is hidden in the preauricular areas and behind the hairline. In patients with a receding hairline or the probability of baldness, this approach should be considered.

The standard high preauricular incisions are extended superiorly and joined by a coronal incision across the skull, behind the hairline (Fig. 17-24). The flap is dissected anteriorly in a subgaleal plane superficial to the

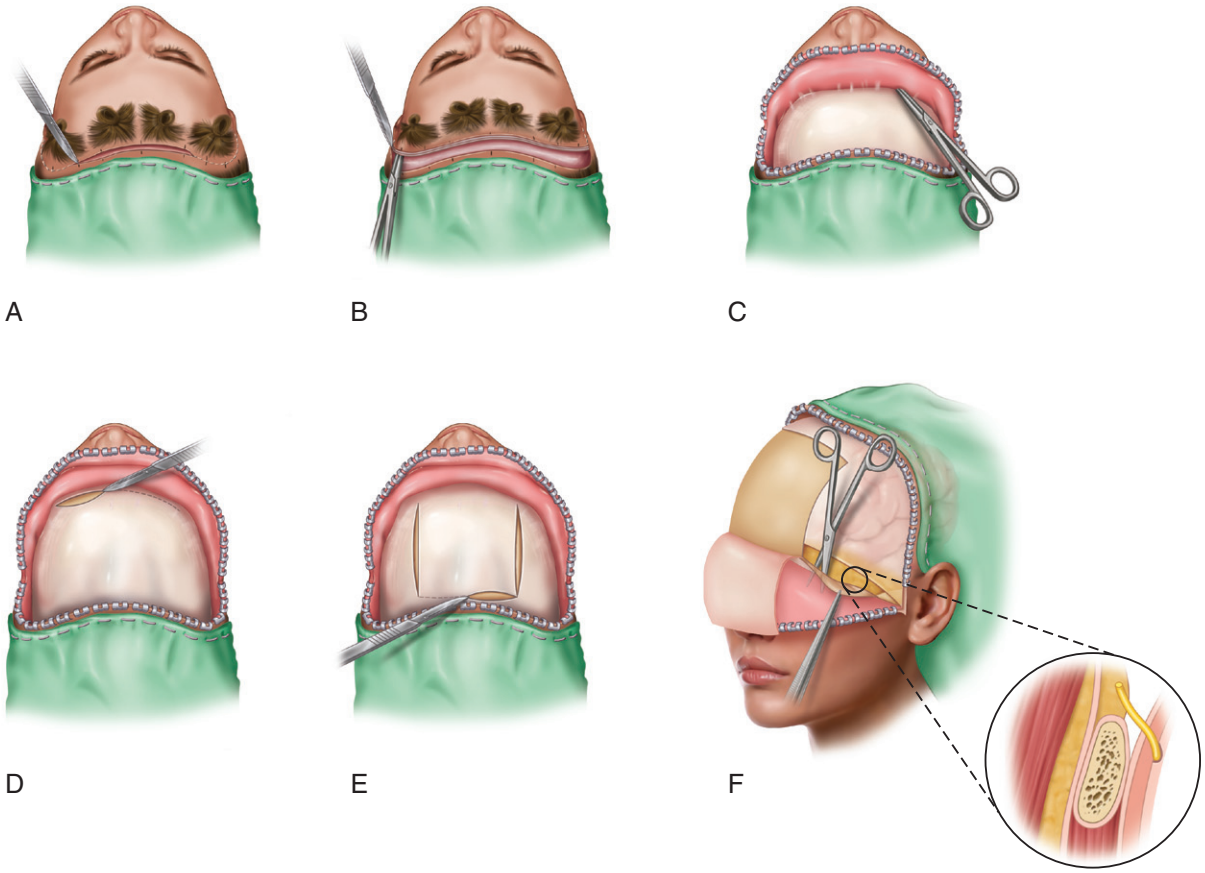


FIGURE 17-24 Coronal flap sequence.

pericranium. The periosteum is incised superiorly to the supraorbital ridges and the dissection is carried out subperiosteally. The temporalis fascia is also incised superiorly to the supraorbital rims, extending from the preauricular incision medially to join the superior dissection. This technique allows reflection of the superficial flap containing the temporalis branch of the facial nerve and thereby prevents injury.

The zygomaticofrontal, zygomaticotemporal, and nasofrontal sutures are well exposed. Resecting the inferior bony margin encasing the supraorbital nerve can facilitate exposure of the medial canthal tendon. This bony margin is easily removed by means of sharp chisels. The release of the nerve allows a continued inferior extension of the flap. The infraorbital rim and floor of the orbit must be exposed via an infraorbital approach. These incisions, in conjunction with an intraoral approach, provide wide open exposure of the fracture sites.

Following reconstruction, the flap is replaced. A closed suction drain may be placed after hemostasis is achieved.

Care should be taken to provide adequate resuspension of the facial soft tissues of the malar, infraorbital, and temporal surface of the orbit to prevent facial sagging and drooping of the eyebrows. A lateral canthopexy is also suggested if extensive lateral dissection has been used.

The scalp is closed in layers with 2-0 slowly resorbing sutures through the galea and staples through the skin. Non–hair-bearing incisions are closed in layers in a routine manner with appropriate sutures. Drains are removed on the second or third postoperative day and staples are removed in 10 to 14 days.

COMPLICATIONS

Complications following midfacial trauma are fairly common. A retrospective study of 20 patients requiring secondary reconstruction for periorbital deformities following initial midfacial trauma repair has concluded that the primary reason for orbital complications is a malpositioned zygoma.[63] Other notable complications include paresthesia of the infraorbital nerve, orbital dystopia, enophthalmos, diplopia, malunion, and lacrimal system dysfunction. These are discussed in the following sections.

MALUNION OF MIDFACE FRACTURES

Malunion of the midface resulting from improper reduction or fixation, postponement of treatment, or excessive comminution may result in suboptimal postrepair function and aesthetics. This may require correction by appropriate osteotomies and bone grafting. Although nonunion is rarely seen with the adjunctive use of plates

and screws, it can be prevented by judicious use of bone grafts during the initial reconstruction, if warranted.

TREATMENT

Malunion can be treated by augmenting the bony depression or by performing an osteotomy. Consideration should be given as to whether the resulting defect is primarily functional or aesthetic.

If a functional deformity exists, a corrective osteotomy should be considered. If corrective osteotomies are planned, they should be performed as early as possible, before the fractures have healed and obscured the proper position of the fractured segments. An example is a malposed zygoma, resulting in enophthalmos, diplopia, or obstruction of mandibular opening caused by obstruction of the coronoid process.

If the deformity is primarily cosmetic in nature, such as a flattened paranasal prominence or malar prominence caused by deficient projection of the zygoma, an onlay graft of autologous or alloplastic material may be acceptable.

Corrective Osteotomies

Diagnosis and treatment planning of secondary deformities from midfacial trauma can be assisted greatly by the use of three-dimensional CT scans. Other effective diagnostic aids include using the contralateral side of the patient's face (if uninjured) as a reference, an exophthalmometer for evaluation of exophthalmos, or a facial moulage for evaluation of three-dimensional deficiency. More promising is the development of computerized surgical navigation techniques and preoperative planning software.[64]

Possible osteotomies include the subcranial or modified Le Fort III osteotomy.[65] Permutations include a focused osteotomy of the Le Fort I level, nose, and zygoma, depending on the area and degree of secondary deformity. The details of these procedures are beyond the scope of this chapter. However, judicious surgical technique is necessary for successful mobilization of the craniomaxillofacial skeleton while preserving visceral function.

The position of the soft tissues may also need to be addressed following secondary corrections. This includes maneuvers such as redraping of the infraorbital tissues or temporalis muscle with the assistance of a suspension sutures to minimize defects after healing.

Bone Grafting in Midfacial Reconstruction

If the fracture results in significant comminution or avulsion, bone grafting may be necessary. However, if tissue avulsion makes primary closure impossible or if the bone graft would be open to the external environment or mucosal secretions, a mechanically stable, plate-maintained reconstruction and closure should be attempted, with secondary reconstruction of the soft and then hard tissues at a later date. This will allow initial anatomic positioning of the maxilla and hopefully limit subsequent midfacial collapse and deformity.

Untreated maxillary fractures are likely to result in midfacial elongation and retrusion because of the pull of the medial pterygoid muscles. Closed reduction of the comminuted maxilla has been used historically and may be an appropriate option if the maxilla is stable against a superior and posterior directed vector of force following the application of intermaxillary fixation. The classic deformity resulting from skeletal suspension of the maxilla is midfacial shortening and retrusion, resulting in overclosure.[66] This occurs because although intermaxillary fixation will direct closure in a horizontal direction, there is nothing to direct the vertical position of the maxilla.[67]

Rigid fixation is the most favorable method of fixation for management of facial fractures in most cases; stable areas of solid bone should be used for anchorage. The source of bone graft material is based on the preference of the surgeon. Cranial bone, rib, and iliac crest are the most commonly used autogenous grafts. Calvarial bone has been shown to be the most resistant to resorption and is also easily adapted to midfacial defects[68] (Fig. 17-25). The frontal bone offers straight grafts and the parietal and occipital areas offer a variety of convex grafts. Cranial bone grafts can be harvested to provide a large bone graft, which can be used as a full-thickness graft, or the diploic bone can be split and the inner table used for grafting purposes, with the outer table replaced to allow reestablishment of normal contour of the calvarium. Alternatively, the outer table can be harvested alone, which can also provide adequate bone for midfacial reconstruction and is associated with low morbidity. The diploic cavity between the inner and outer tables is consistently 2 mm thick; however, this space diminishes with advancing age, which should be considered when using this type of graft.[69] In our experience, the calvarium is the ideal autologous choice for midfacial reconstruction for many reasons and is often already exposed in the field through the coronal flap, minimizing further morbidity for the patient.

The rib cage is also a potential source of autogenous bone. The most common source is the fourth through eighth ribs. The upper ribs are larger. The most common postoperative complications have been pleural lacerations. Laurie et al[70] have reported such complications in 9% of a series of 44 cases. Persistent intercostal pain can also occur as a result of the injury. Although providing some structural integrity, resorption of rib grafts, when used to provide contour or onlay projection, remains a problem over the long term. Thus, rib grafting for midface trauma is rarely used.

The iliac crest is also a popular source of autograft because of the amount of bone available and the percentage of cancellous bone present. The posterior crest offers almost three times the amount of bone for grafting purposes and has been associated with less morbidity. The drawback is having to turn the patient midprocedure or deal with alternative positioning.[71,72]

By approaching the anterior crest from a medial direction, one bypasses the iliotibial band, comprised of the tensor fascia lata muscle and fascia lata. This is important because failure to reattach these structures may result in an inability to stabilize the upper thigh and lift and flex the leg when walking. By using the medial approach to the iliacus muscle, the postural muscles are preserved. The only gait disturbance is a result of postoperative pain

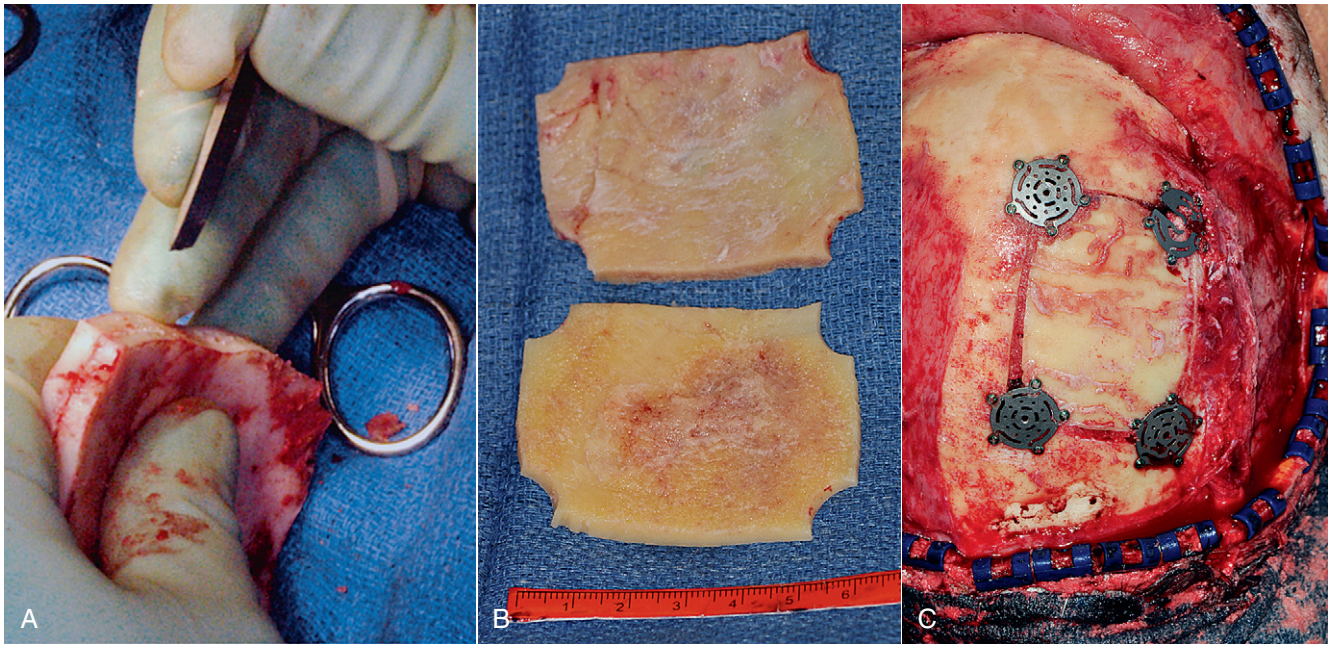


FIGURE 17-25 Cranial bone graft.

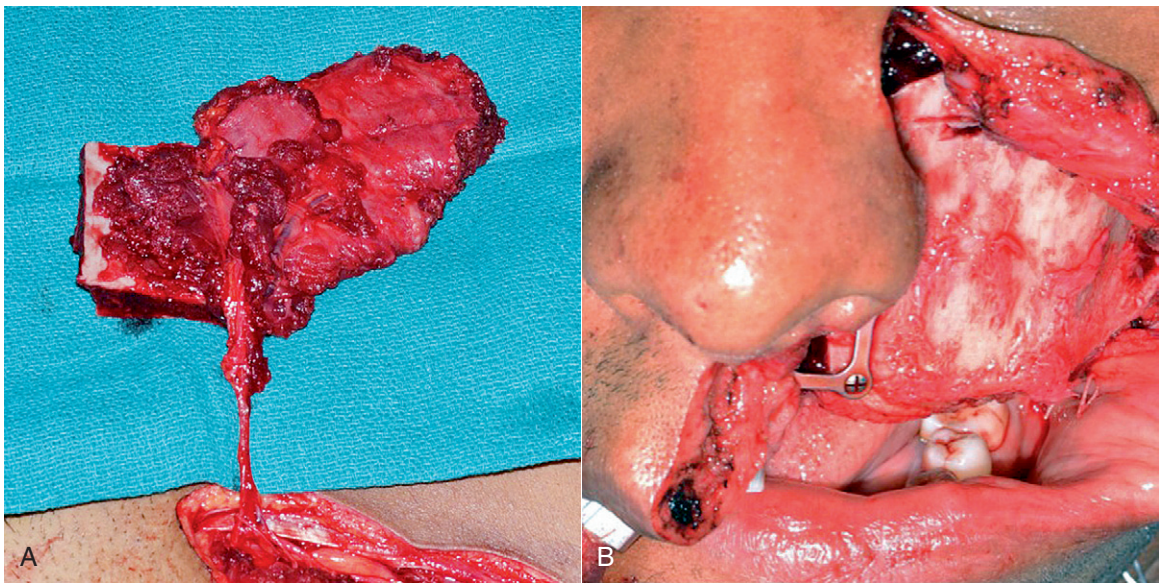


FIGURE 17-26 Microvascular free tissue transfer for midface reconstruction. (Courtesy Dr. Fayette Williams)

in the area. Anesthesia and paresthesia can occur following harvesting from the anterior and posterior iliac crest. Damage to the lateral cutaneous branch of the subcostal nerve (T12) and lateral cutaneous branch of the iliohypogastric nerve (L1) causes sensory denervation of the skin overlying the gluteus medius and gluteus minimus muscles. When approaching the posterior hip, damage to the superior and middle cluneal nerves will result in paresthesia over the region of the gluteus maximus muscle.

The use of microvascular free tissue transfer has dramatically increased the possibilities for immediate or delayed reconstruction of the severely avulsive midfacial injury. There are a number of flap variations and the decision regarding which flap to use should focus on the following: the adequacy and location of the proposed tissue bed; existing medical comorbidities; amount of tissue required; and whether bone (e.g., an osteocutaneous flap) is necessary for reconstruction[73] (Fig. 17-26).

The development of recombinant human bone morphogenetic protein (rhBMP-2)–assisted grafting has also been a transformative event in craniomaxillofacial reconstruction. Limited by not having structural integrity, these grafts are generally used with the assistance of autogenous grafts or allografts for bulk and/or structure. In addition, the use of a mesh or crib can be helpful in

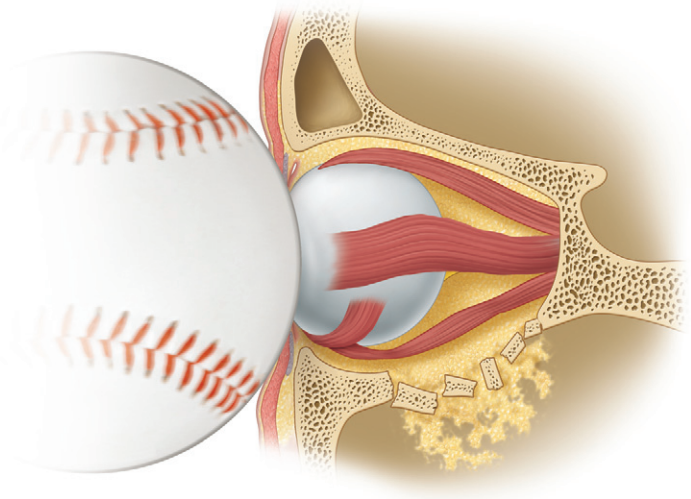


FIGURE 17-27 Blowout fracture of the orbital floor.

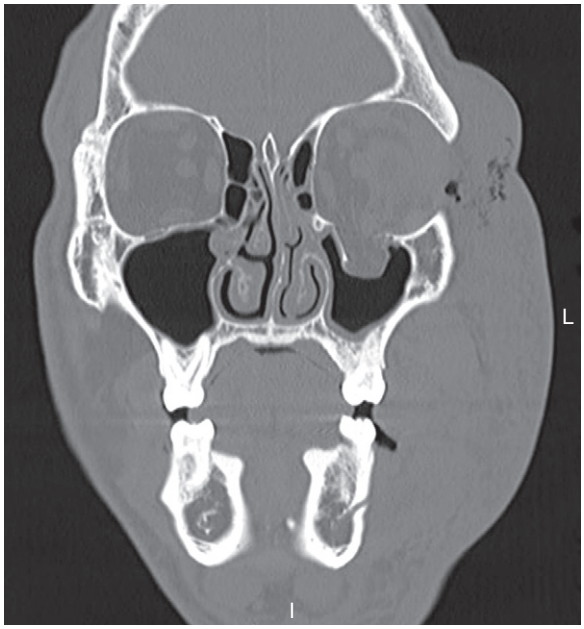


FIGURE 17-28 Coronal CT scan demonstrating a blow out fracture of the orbital floor.

providing contour and resisting soft tissue compression during consolidation.[74] If delayed reconstruction is planned, the receptor bed must be optimally vascularized with sufficient soft tissue, skin, and mucosa for closure. Soft tissue grafting or tissue expansion can be used to develop a healthy vascular bed and then secondary grafting can proceed. Before secondary reconstruction, the reepithelialized or grafted tissue should be allowed to mature to withstand manipulation during the bone reconstruction.

ORBITAL WALL FRACTURES

Fracture of the orbital walls can result in ophthalmic complications such as diplopia, enophthalmos, and vertical diplopia. Incomplete or improper reconstruction may fail to correct, or may even worsen, these conditions. The same level of care should be taken to reconstruct the orbital walls appropriately, as for the orbital rims.

Orbital wall fractures can be divided into two sections, anterior and posterior. The anterior section is composed of the orbital rim. The posterior section is composed of the thinner roof, floor, and medial and lateral walls. These fractures are commonly referred to as blow-in and blowout, fractures depending on the direction of the fracture.[75-77]

There have been two major theories proposed regarding the mechanism of blowout fractures. Converse and Smith[78] and Smith and Reagan,[76] who are known to have coined the term *blowout fracture*, described a hydraulic mechanism whereby hydrostatic pressure within the globe or orbital contents is transmitted to the orbital walls. An opposing theory has suggested that impact against the sturdy orbital rim transmits force to the more fragile orbital walls, resulting in a blowout fracture (Fig. 17-27). The increased incidence of blowout fractures in children is an indication that bone elasticity and orbital deformation play a role in orbital blowout fractures,[79] a concept is also supported by experimental studies by Fujino and Makino.[80] It is likely that hydraulic forces and bone conduction play a role in the mechanism of orbital blowout fractures.[81]

Regardless of the actual mechanism of injury, blowout injuries are further described as pure, for those that occur in the presence of an intact orbital rim, and impure, for those with a concomitant fracture of the orbital rim. Blowout fractures can occur on the floor, medial wall, or lateral wall (Fig. 17-28). Medial wall fractures accompany approximately 20% of orbital floor fractures.[82-84]

The incidence of orbital wall fractures has been reported to range from 4% to 70% of those who sustain orbital trauma. Isolated blowout fractures likely represent between 5% and 21.4% of midfacial fractures.[85-87]

BLOWOUT FRACTURES

Diagnosis

It is difficult to make a clinical diagnosis of an isolated orbital blowout fracture. Often, these fractures would not be clinically notable until several weeks later, when diplopia was noted.[88] The clinical examination is also initially obscured by significant edema, which may mask visual observation of enophthalmos or vertical diplopia and palpation of bony step deformities.

Extraocular movements should be assessed by the evaluation of cardinal movements. If there is any question about muscle entrapment, a forced duction test of all four rectus muscles is indicated. Limitations of motion may be the result of early postinjury edema and prolapse of orbital contents, but may allow normal range of motion prior to scarring and contracture[89] (Fig. 17-29). Damage to the infraorbital nerve may also be present in blowout fractures. A blowout fracture should be suspected if paresthesia of the infraorbital nerve distribution is present following trauma, with limitation of normal ocular motion and no notable fracture of the rim.[90]

Noncontrast CT scans with 1.5- or 2-mm axial, sagittal, and coronal cuts are the most appropriate for specific evaluation of the orbit. The indications for surgical intervention for an isolated, radiographically evident orbital blowout fracture is nonresolving diplopia within 2 to 3 weeks of injury or enophthalmos greater than 2 mm. Supporting evidence is provided by a positive forced duction test demonstrating ocular motility restriction not related to edema and CT evidence of orbital floor blowout injury. Other relative indications for repair include orbital floor defects larger than 1 cm^2 or clinically notable hypoglobus.

Once these criteria have been met, surgery should proceed in a timely manner. Emery et al[91] have provided a natural history of diplopia in isolated orbital blowout fractures. When diplopia was present on initial presentation, 55% had resolution of symptoms at 15 days, whereas others had gradual resolution of symptoms after the initial 15-day period, and 27% of this subgroup had some degree of permanent diplopia. Dulley and Fells, in a review of 103 patients, have found a 72% incidence of enophthalmos when the repair occurred at 6 months compared with a 20% incidence when surgery was performed at 2 weeks. Additionally, 40% of patients also needed additional ocular surgery, with limited success at resolving alterations in binocular vision.[92]



FIGURE 17-29 Clinical limitation of upward gaze because of orbital floor disruption.

Treatment

Surgical approaches to the orbit have been discussed earlier. The most commonly used are the subtarsal, subciliary, and transconjunctival incisions, with various modifications based on the location and presence of concomitant injuries.

Many materials have been suggested for orbital floor reconstruction. Autogenous grafts are useful for the management of large, significant floor or wall defects, the skeletally immature orbit, and secondary orbital reconstruction (Fig. 17-30). They are highly biocompatible but require donor site surgery. Donor sites usually suggested for reconstruction of the orbit include the anterior and posterior iliac crests and unicortical or bicortical calvarium. The anterior and posterior iliac crests have the advantage of providing different shapes and sizes, which is especially important if the posterior wall is absent or when correction of enophthalmos is being undertaken. The wedge-shaped floor implant necessary for forward projection of the globe requires an adequate thickness of graft material. Unicortical or bicortical calvarial bone may also be used for this purpose. The advantage of calvarial bone is that it is more resistant to absorption and, if selected properly, may closely mimic the shape of the orbital floor. The parietal and occipital regions of the calvarium seem uniquely suited to this application, and the grafts may be stacked to obtain the desired shape and thickness. Monocortical grafts provide an adequate graft, especially in the younger age group. The diploic space measures an average of 2 mm and allows a safe harvest site. Jackson et al,[69] Tessier,[88] and Powell and Riley,[93] reporting on large series of cases, noted few complications with this technique. Tessier et al, in a retrospective review of several surgeons over 25 to 50 years, have reported the incidence of dural tear as 25 in 9650 (0.26%) and never with injury to the underlying brain.[94] For smaller defects, the lateral mandibular cortex, lateral antral wall, and nasal septum are acceptable alternative donor sites.

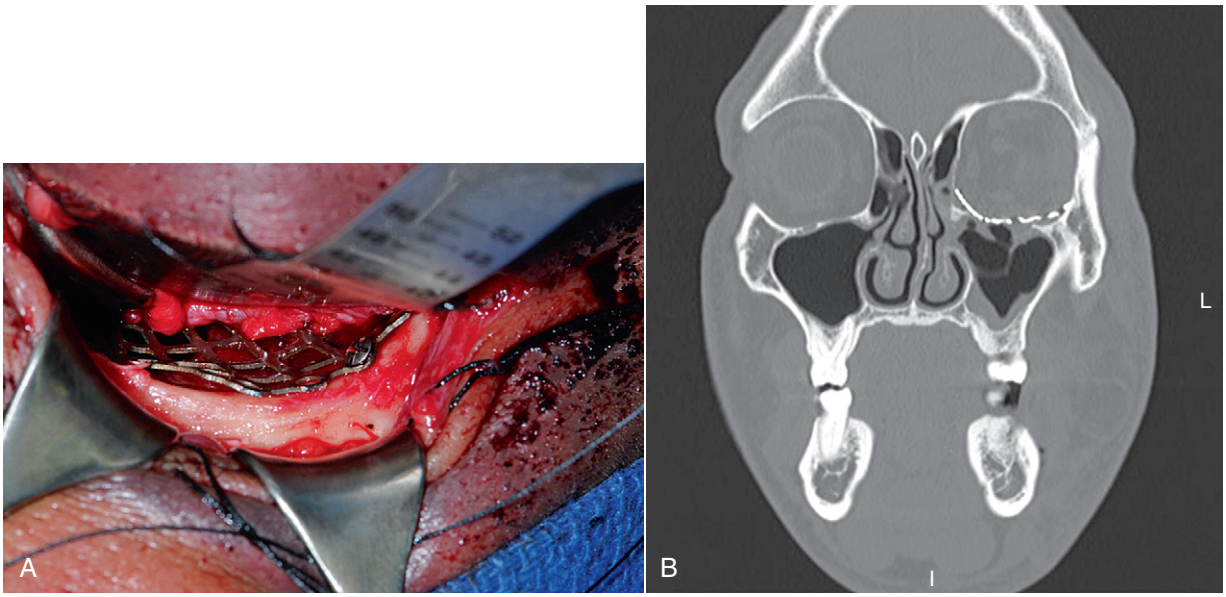


FIGURE 17-30 **A,** Orbital blowout fracture plated via the transconjunctival approach, **B,** Postoperative coronal CT scan.

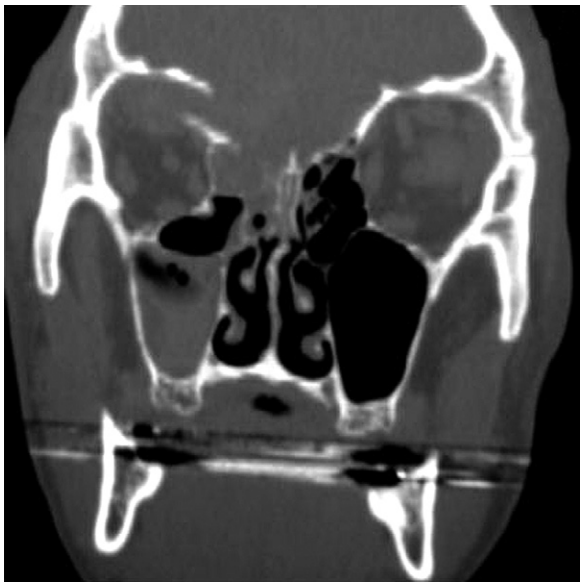


FIGURE 17-31 Axial CT scan of a blow-in fracture.

Many alloplastic materials are also available commercially and have become popular for routine use, including titanium mesh, polymeric silicone, polytetrafluoroethylene, polyethylene, and Gelfilm.[95-98] These materials limit the need for a donor site and most do not undergo resorption. On the other hand, these materials also have the potential to induce a foreign body response that might result in infection, extrusion, and/or displacement into the maxillary sinus.[99,100] Complications such as these are relatively rare with these materials but, if they do occur, are most likely to develop within the first few months postplacement. Consideration should be given to securing these materials in the orbit with screws or nonabsorbable sutures to limit the chance of migration until fibrous encapsulation has occurred.

Allogenic materials have also been used with success. These include lyophilized dura, allogenic bone, and cartilage.[101-106]

Tessier and others have stressed the importance of wide dissection of the periorbital tissue from the orbital walls, with particular attention to the floor defect. This is particularly important in late repair or treatment of enophthalmos.[88,107-111] Studies have suggested overcorrection of the floor defect, regardless of the method of repair. Multiple procedures may be necessary to achieve adequate correction, particularly with the repair of late enophthalmos.

Following completion of the orbital reconstruction, a forced duction test is mandatory to ensure that the positioning of the reconstructive material has not trapped inferior periorbital tissue.[110]

MEDIAL WALL FRACTURES

Diagnosis

The medial orbital wall is composed primarily of the thin lamina papyracea of the ethmoid bone. This is the second most commonly fractured orbital wall, comprising 20% of cases of disruption in orbital floor fractures.[82-84]

The diagnosis of medial wall fracture is made clinically by limitation in abduction of the globe, often in conjunction with globe retraction. This is caused by damage or entrapment of the medial rectus muscle. A forced duction test in this setting is mandatory. Medial wall fractures are most easily visualized on axial scans with maxillofacial CT. One study has shown that anteroposterior and coronal CT slices could not detect 100% of medial wall fractures.[82]

Treatment

The necessity to repair a medial wall fracture depends on the extent of the defect and degree of orbital tissue lost. A small defect does not require repair. If the defect is large and accompanies a floor defect, reconstruction may be required in a similar manner as orbital floor reconstruction.

The approach to the medial wall can be made by a Lynch incision, lateral nasal incision, coronal flap, existing laceration, or transcaruncular approach. The Lynch incision is a curvilinear incision made 12 mm medial to the medial palpebrae to prevent severing the insertion of the medial canthal ligament. This approach, as well as the lateral nasal approach, are prone to scarring and unaesthetic webbing of the incision sites. Other approaches, such as the coronal and transcaruncular approaches, avoid this complication. The transcaruncular approach is made by extension of the transconjunctival approach through the caruncle and has been shown to provide excellent access, with few complications.[112]

Dissection of the medial wall should take into account several anatomic landmarks. The anterior ethmoidal vessels lie 24 mm posterior and superior to the anterior lacrimal crest, with the posterior ethmoidal vessels approximately 12 mm behind them. The optic canal is 40 mm posterior to the anterior lacrimal crest.

BLOW-IN FRACTURES

The blow-in fracture, initially described by Dingman and Natvig in 1964,[113] is less common than the typical blowout fracture (Fig. 17-31). In a review of clinical findings in 41 patients with blow-in injuries, Antonyshyn et al[114] noted that 25% to 30% of them presented with proptosis because of a decrease in orbital volume and associated restricted ocular motility and diplopia. Other unusual findings reported include rupture of the globe, superior orbital fissure syndrome, and optic nerve injury.[115] Therefore, the blow-in fracture should be managed acutely. Particular attention must also be paid to orbital roof fractures. Separation of the orbit and dura in significant roof fractures must be preserved for several reasons. First, the orbital roof can become pulsatile with adhesion of the orbital contents and dura; second, in the young growing child, herniation of the leptomeninges can occur.[116]

COMPLICATIONS AFTER TREATMENT OF MIDFACE FRACTURES

BLEEDING

In the absence of a coagulopathy, severe hemorrhage from maxillofacial injuries is rare. The primary blood supply to the region is from the external carotid system.

Source control is usually easily accomplished. Buchanan and Holtmann have reported on 312 patients over 5 years and recorded epistaxis in 4% of facial fractures and 11% of midface fractures. Only 6 patients required packing or transfusion and only 2 required surgery for control of the hemorrhage.[117] In the patient with the avulsive midfacial injury, bleeding can be profuse. Direct control is usually possible through the wound.

Control of Bleeding

Midfacial bleeding usually manifests as epistaxis. Control of epistaxis requires an understanding of the vascular supply to the region. Direct and unobscured visualization is mandatory; phenylephrine hydrochloride (Neo-Synephrine) or a 4% cocaine solution assist by shrinking and anesthetizing nasal mucosa. If the bleeding is from the septal wall, the anterior and posterior ethmoidal arteries that supply the anterior portion of the septum are often the source. The nasopalatine, greater palatine, and superior labial arteries supply the posterior aspect of the septum.

Bleeding from the superolateral nasal wall emanates from the anterior and posterior ethmoidal arteries. Bleeding from the posterior aspect is likely from the sphenopalatine artery and the anterior aspect of the lateral wall is usually from the nasal branch of the facial artery (Fig. 17-32).

The patient's head should be maintained in a forward position to prevent pooling of blood in the pharynx and subsequent airway embarrassment. If a vasoconstrictor-soaked cottonoid pledget does not control the bleeding, cauterization may be attempted with silver nitrate. An anterior nasal pack may also be attempted. If this fails to control the bleeding, a posterior nasal pack or pressure balloon may be necessary.

If initial measures do not control epistaxis, angiography should be performed to determine the source of bleeding. Once the source is identified, embolization or ligation may be required.

To ligate the ethmoidal arteries, a 3-cm incision is made above the attachment of the medial canthal ligament and below the trochlear ligament. A subperiosteal dissection is carried out, exposing the confluence of the frontolacrimal and frontoethmoidal sutures. The frontoethmoidal sutures are followed posteriorly, taking care not to puncture the thin lamina papyracea. The anterior ethmoidal artery is identified and ligated with a vascular clip; the posterior ethmoidal artery can similarly be identified and ligated. Care must be taken during this dissection to stay anterior to the optic nerve, which is in close proximity (Fig. 17-33).

Ligation of the external carotid artery plays a limited role in the management of facial bleeding, generally because of collateral circulation from the contralateral external carotid arterial system, and this technique prevents subsequent attempts at arteriography and embolization.[118,119] Arteriography with selective embolization is a rapid and effective method to gain control of midfacial bleeding. In most cases, beyond those for which direct packing is useful, this technique has garnered universal acceptance because of the associated minimal morbidity and efficacy.

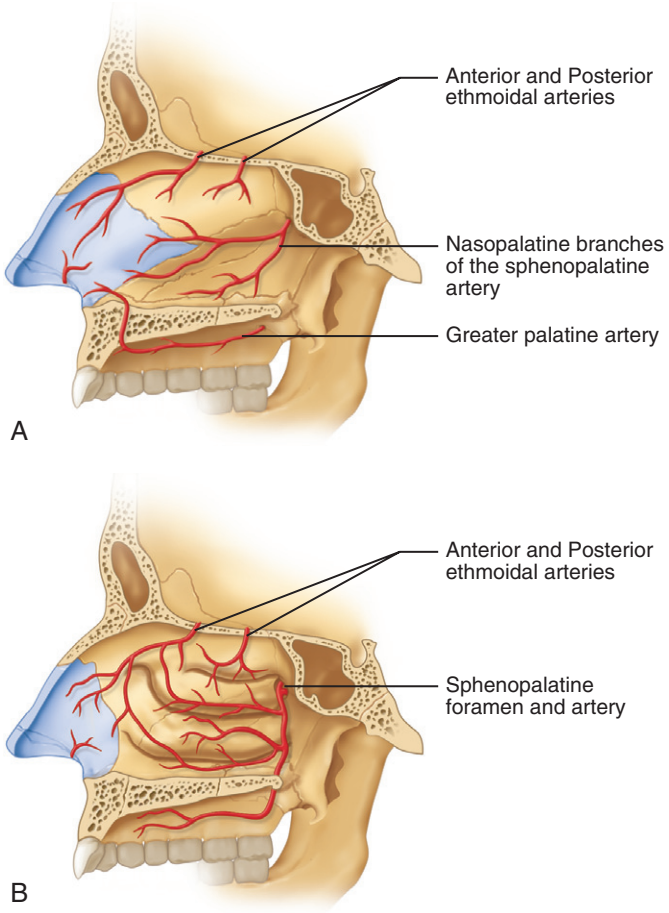


FIGURE 17-32 Anatomy of the medial wall **(A)** and lateral wall **(B)** of the nasal cavity.

CEREBROSPINAL FLUID RHINORRHEA AND OTORRHEA

Following trauma of the midface, CSF may emanate from a dural tear, resulting in rhinorrhea, otorrhea, or both. The disruption usually occurs lateral to the cribriform plate but may also result from disruption of the sphenoidal, ethmoidal, and frontal sinuses producing a dural tear and communication with the subarachnoid space.

CSF was first described by Willis in 1676.[117] CSF otorrhea emanating from the external auditory canal occurs in approximately 7% of basilar skull fractures.[120] Leakage is typically noted immediately following trauma but may occasionally be noted days to weeks after the traumatic event. This situation may occur when debris such as a blood clot or avulsed tissue obstructs free passage of the CSF, resulting in late leakage following clot lysis or an increase in intracranial pressure.

Diagnosis of CSF leakage is often complicated by mixture with nasal secretions, lacrimal secretions, and blood. Clear CSF should be collected in a vial, an absence of sediment and a glucose level of approximately 45 mg/dL is usually confirmatory. A more specific, albeit slower, laboratory examination is the CSF-specific beta-2 transferrin test.[116] CSF will also form concentric rings when poured on linen or soft filter paper. When bleeding is present, one should suspect the presence of a CSF leak

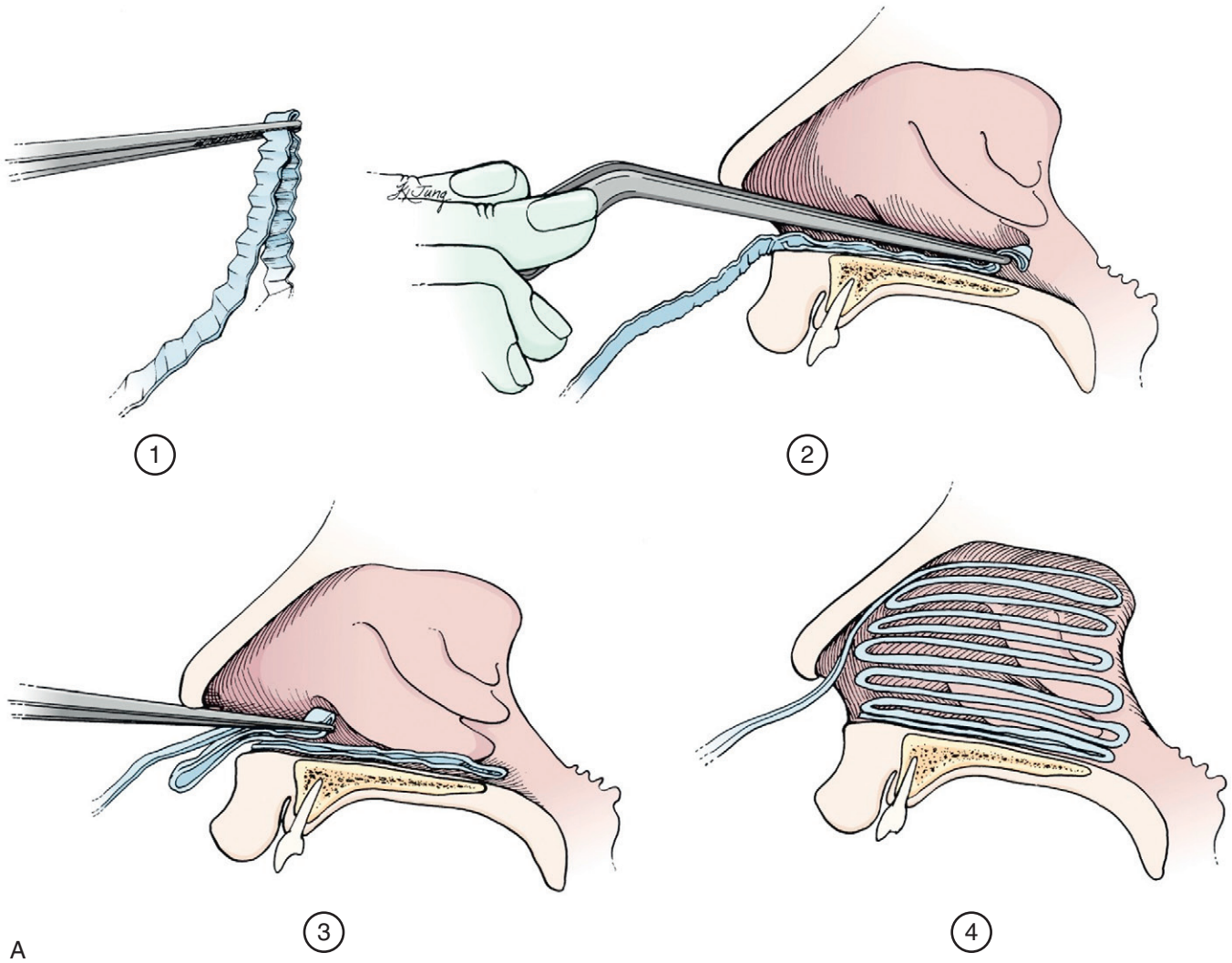


FIGURE 17-33 Correct technique for anterior packing of the nose. **A,** *1.* The gauze is gripped 4 to 6 cm from the end. *2.* The first layer is placed along the floor of the nose. *3, 4.* Subsequent layering of the gauze packing.

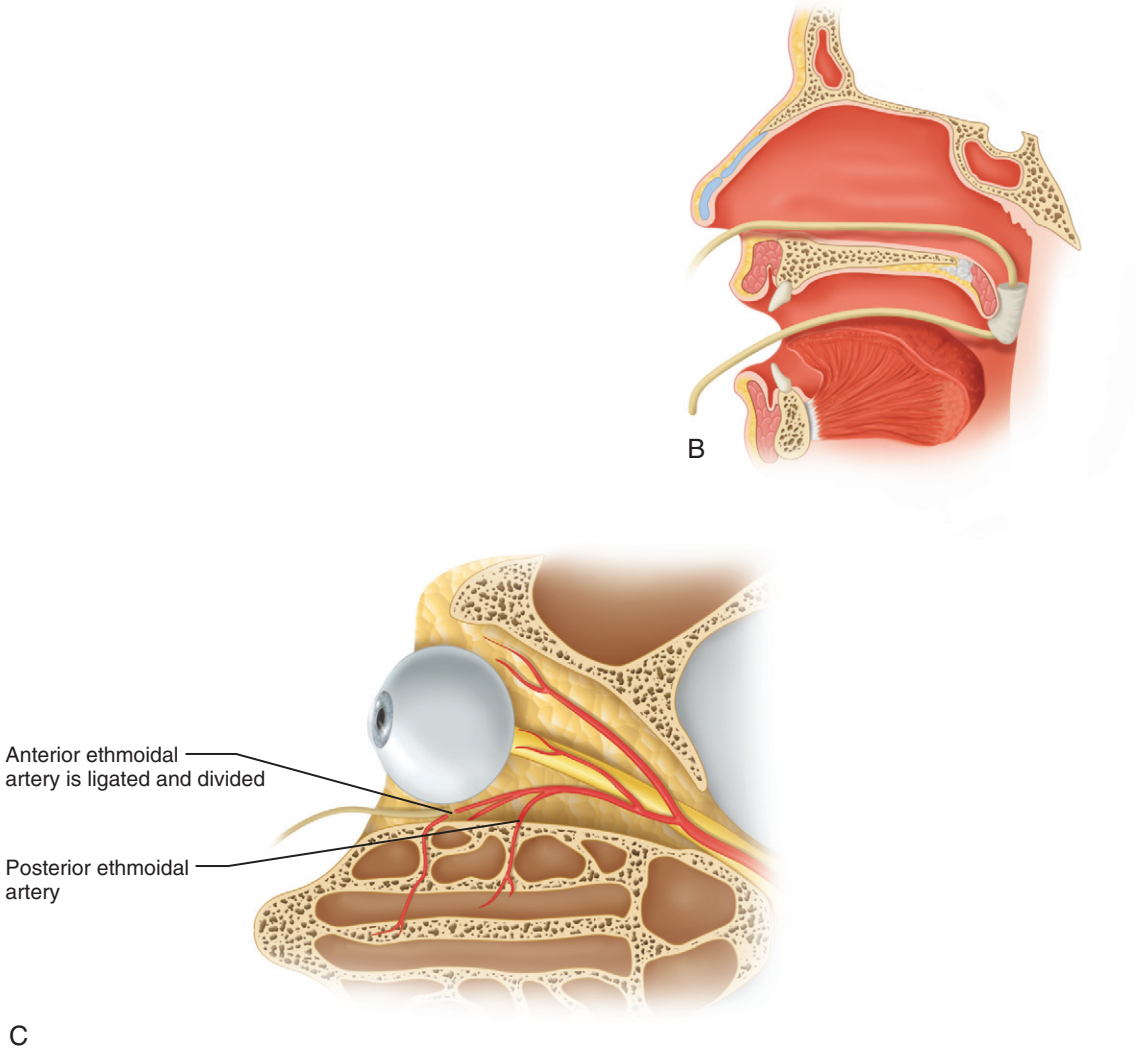


FIGURE 17-33, cont'd **B,** Posterior nasal packing. **C,** Ligation of the anterior and posterior ethmoidal arteries. (**A** from Roberts J, Hedges J: *Clinical procedures in emergency medicine*, ed 5, Philadelphia, 2010, Saunders.)

until proven otherwise. Once a provisional diagnosis is made, the patient should be placed in a semirecumbent position and instructed on how to minimize increases in intracranial pressure, including straining, sneezing and blowing of the nose.

Meningitis is a potential complication of skull base fracture with a concomitant dural tear. The absence of leakage does not imply the absence of a tear. The use of prophylactic antibiotics for potential meningitis is controversial and varies among institutions. Meningitis may develop in spite of antibiotic therapy because of opportunistic or resistant organisms, or the presence of a large bacterial load of nasopharyngeal and respiratory flora. Some studies have shown a decreased incidence of meningitis with prophylactic antibiotic therapy.[121-123]

The presence of a dural tear is not a contraindication to surgical repair of midfacial fractures. Early reduction and fixation will likely reduce the changes in intracranial pressure associated with mobile fractures that results in intermittent pumping of CSF through a dural disruption. Finally, if CSF leakage has not subsided in 3 to 4 weeks postreduction, surgical correction of the leak is indicated.[124]

Pneumocephalus may be noted on CT scans in any extradural, subdural, intracerebral, subarachnoid, or ventricular site. Pneumocephalus may be the result of fractures of the cribriform region, where the bone is very thin and the dura is bound tightly to the skull, or from any basilar skull or sphenoid fracture in which air in the ventricular and subarachnoid regions is common. Pneumocephalus is not immediately a cause for alarm but may indicate an accompanying CSF leak. It requires observation for subsequent infection and development of a cerebral abscess. The risk of meningitis secondary to a conservatively managed dural tear is significant and adequate follow-up is of paramount importance.

OCULAR COMPLICATIONS

Traumatic Diplopia

Among the most significant complications encountered in midfacial trauma and reconstruction are diplopia, enophthalmos and, on rare occasions, blindness. The most commonly noted ocular complication is traumatically induced diplopia.

Diagnosis. Traumatic diplopia occurs from 3.4% to 20% of the time in the presence of midfacial trauma

and may be temporary or permanent if not managed appropriately.[125-129] The condition is caused by the interference of extraocular muscles. This may be secondary to edema or hemorrhage with increased intraocular pressure, displacement of globe position, entrapment of muscle or fat in the line of fracture, bony displacement, and resultant alterations in extraocular muscle attachments. Also, adhesions between orbital contents such as the periosteum, muscle, or fat may form at the bony fracture sites, resulting in limitation of ocular motion. Damage or impingement of cranial nerves III, IV, and VI, may also result in diplopia.

Note that entrapment of the inferior rectus is not the major cause of diplopia. Tessier has maintained that diplopia is the result of an oculomotor imbalance and reported true entrapment in only 3 of 600 post-traumatic orbital repairs.[88]

An ophthalmologist should make the diagnosis of diplopia; the presence or development of symptoms of diplopia should initiate a prompt ophthalmologic referral. Putterman et al[130] have introduced a simplified method for evaluation and documentation of the degree of diplopia. The patient is initially positioned in primary gaze and the examiner shines a light into the eye. Then the patient is asked to move into each of the gaze positions and the patient states whether one or two lights is seen. This test is repeated several times in the first week and then weekly, preferably by the same examiner. Persistent diplopia greater than 30 degrees of primary gaze is an indication to proceed with surgical repair.

Binocular diplopia can become permanent unless treated. Binocular diplopia is most likely to be observed when midfacial trauma results in inferior displacement of the zygoma at the level of the zygomaticofrontal suture (i.e., above Witnall's tubercle) and results in displacement of Lockwood's suspensory ligament. Displacement or disruption of Lockwood's ligament is necessary for diplopia to occur. A disruption of the floor below Witnall's tubercle alone will not produce diplopia predictably without disruption of the suspensory ligament.

As noted, herniation of the orbital contents into the maxillary antrum may result in restriction in the primary upward and lateral gaze. Lateral gaze restriction may also occur with rupture of the medial wall by the same mechanism. Assessment is limited initially by edema, bleeding, or both. This usually takes 7 to 10 days to resolve sufficiently to determine whether the diplopia is transitory. The differential diagnosis includes edema, neurologic damage to the affected extraocular muscles, and entrapment. A forced duction test can assist in ruling out entrapment.

A forced duction test is performed by initially placing a drop of local anesthetic in the cul-de-sac of the lower lid and, using a pair of small toothed forceps, grasping the tendon of the rectus muscle in question and rotating the globe passively away from the restriction (Fig. 17-34). Limitation of motion can be caused by orbital edema, hemorrhage, or both and is not invariably an indication of muscular entrapment. Conversely, absence of resistance may indicate a neurologic deficit or possibly the presence of a large nonrestrictive floor defect. A positive result is usually the result of a trapdoor type of injury (Fig. 17-35).

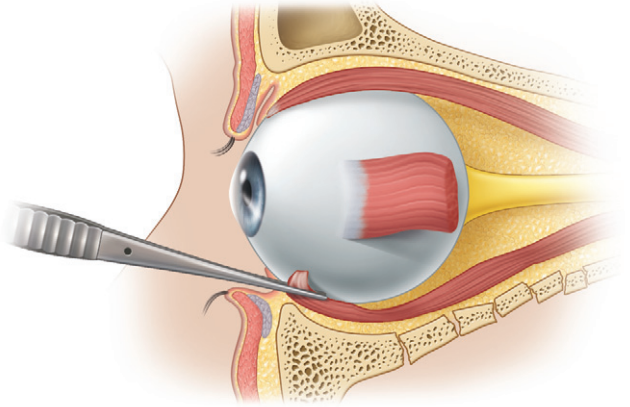


FIGURE 17-34 Performance of a forced duction test.



FIGURE 17-35 Clinical demonstration of a forced duction test of the inferior rectus muscle.

Timing of repair should take into account enough time to allow a workup, treatment planning, and resolution of initial edema. Waiting a period of at least 1 week allows this process to proceed smoothly and also provides an opportunity to observe spontaneous resolution of diplopia, as well as development of enophthalmos. Reduced vision and retrobulbar hemorrhage are also complications that have been described following injury and before surgical management.[131,132]

Treatment. The timing for treatment of diplopia has been controversial. It is clear that an inferiorly displaced zygoma, a large orbital floor defect with disruption of Lockwood's ligament and subsequent inferior repositioning of the globe, or a trapdoor injury with restriction of movement should be expediently repaired. The controversy is when to operate in the case of slow resolution of diplopia and a concomitant cosmetic deformity. Steroids should be administered for 5 to 7 days to help determine whether diplopia is caused by edema or hemorrhage of



FIGURE 17-36 Bird's eye view of patient with enophthalmos.



FIGURE 17-37 Axial CT scan displaying post-traumatic enophthalmos.

the orbital muscle or fat.[133] A maxillofacial CT scan of the orbit can help determine entrapment versus contusion of the extraocular muscles. A contused muscle will appear round as opposed to its normal flat appearance. In case of entrapment, a forced duction test is mandatory as the final indicator for immediate repair.

Objectives for reconstruction of the orbital floor include preventing the loss of orbital contents, providing a smooth floor, and reconstructing the floor or wall to mirror the contralateral side anatomically. With a trapdoor injury, the tissue is carefully dissected from the line of fracture without any other reconstruction of the orbital floor. If the defect is large enough to allow extrusion of the orbital contents, these should be elevated and the defect sealed, as discussed earlier.

Enophthalmos

Enophthalmos secondary to orbital trauma was described over 100 years ago.[134] The incidence of post-traumatic enophthalmos secondary midfacial trauma is unclear; a retrospective study by Gilbard et al[135] have reported an incidence of 22% but a more recent prospective study by al-Qurainy et al have reported an incidence of 8%[13] (Fig. 17-36).

Post-traumatic enophthalmos has been attributed to atrophy of the orbital fat, enlargement of the bony orbit, dislocation of the trochlea, cicatricial contraction of the retrobulbar tissue, unrepaired fracture of the orbital wall, and displacement of the orbital tissue. Tessier has pointed out that loss of orbital fat rather than post-traumatic fat atrophy plays the predominate role in traumatic enophthalmos.[107] As noted earlier, disruption of the orbital floor alone, with an intact suspensory system, does not result in globe displacement.[55] However, disruption of the orbital walls resulting in significant displacement of ligamentous attachments for suspensory ligaments of the globe causes a decrease in anterior support and subsequent enophthalmos (Fig. 17-37).

The axis described extends from the lateral orbital rim to the anterior portion of the lacrimal bone. Most fat along the orbital floor is extraconal, meaning that it is anterior to this axis. Displacement of this fat through an orbital floor fracture will seldom result in enophthalmos. However, because most of the lateral orbital wall is posterior to this axis, displacement of this osseous segment will result in an increase in orbital volume and resultant enophthalmos. Comminuted fractures to the lamina papyracea also lie behind the global axis, with the same effect. Intraconal fat is present behind the global axis, and loss of this structure will likely result in the development of enophthalmos.

Enophthalmos is more likely the result of an increase in bony orbital volume by displacement of the medial orbital wall, posterior floor, or lateral orbital wall than the anterior orbital floor. Treatment should therefore focus on repair of these areas and secondarily on the anterior orbital floor. Anterior orbital floor reconstruction should focus on changes in the vertical relationship of the globe and not on the correction of anteroposterior problems.

Physical Examination and Imaging. Physical examination proceeds in the usual fashion, with particular attention to the frontozygomatic suture and infraorbital rim region. A step deformity of the infraorbital rim may be evidence of an inferior and lateral displacement of the zygomatic complex. Frontozygomatic, zygomaticomaxillary, and zygomaticotemporal sutures are commonly displaced in low-velocity injuries and should be evaluated for position and displacement. The zygomatic arch may be inferiorly displaced because of the dual effect of masseter pull and gravity on the fractured segment. A complete examination of the globe for gross injuries, changes in visual acuity, and changes in extraocular muscle function by forced duction test are mandatory. A Hertel exophthalmometer may be used to determine the degree of enophthalmos; more than 3 mm of deficit is considered aesthetically unacceptable.

Pathologic Features. The most common cause of enophthalmos is the lateral and inferior repositioning of the body of the zygoma, resulting in increased intraorbital volume posterior to the axis of the globe. Reconstructive efforts must pay close attention to the position of the zygoma in three dimensions. Osseous reconstruction and rigid fixation for the zygomatic fracture and those areas posterior to the global axis will ensure a stable orbital volume and a satisfactory postoperative appearance.

Blowout fractures, isolated or in conjunction with zygoma or rim fractures, must extend behind the axis of the globe to create the volumetric expansion necessary for a resultant enophthalmos. Usually, this is the result of a concomitant medial wall component. Repair of these fractures should include elevation and securing of all herniated tissue back into the orbital cavity. It is essential to complete the dissection far enough posteriorly to ensure that any orbital floor disruption posterior to the axis of the globe has been addressed to prevent late enophthalmos.

Secondary Repair. Dulley and Fells have emphasized the importance of prevention in regard to enophthalmos, reporting a 72% incidence of postoperative enophthalmos when treatment was delayed longer than 6 months following orbital trauma compared with 20% when repair was performed within 14 days. Also, in the late repair group, 40% required additional surgeries.[92] In the secondary repair of enophthalmos, a similar physical and radiographic examination is indicated to determine the component of the orbital cavity that is primarily responsible for the increase in orbital volume. Access to the orbital cavity proceeds in the usual manner, with wide undermining of the periorbital soft tissues. The globe is freed, as determined by a forced duction test. When the culprit is a lateral and inferior displacement of the body of the zygoma, osteotomies are performed at the junction of the zygoma and maxilla. Bone grafts can then be used to elevate the body of the zygoma into the appropriate position. Rigid fixation is used to stabilize the segment. Wedge-shaped grafts may be positioned with the thicker edge to the posterior globe and may also be used to project the globe into a more anterior and superior position. Cranial bone grafts or preformed alloplastic wedges may be used for this purpose, providing a sufficient bulk of material to be held in position by fixation to the infraorbital rim. As noted, secondary correction of this issue is difficult and planning a certain amount of overcorrection is often necessary, with multiple procedures, to achieve an acceptable result.

Blindness

The most devastating ophthalmic complication of midfacial trauma is blindness. The rate of occurrence has been reported to range from 0.03% to 3%.[136,137] It may be the result of ischemia secondary to retrobulbar hemorrhage or direct trauma to the optic nerve.

Prompt radiologic assessment by CT should be used in the setting of decreased visual acuity following trauma. Manfredi et al[138] have found a larger number of optic canal fractures in patients sustaining blindness from facial trauma. Kellela et al have found swelling of the optic nerve to be the most common CT finding in patients with post-traumatic blindness, followed by fracture of the optic canal.[139]

Retrobulbar Hematoma. The most common cause of blindness in the setting of midfacial trauma is retrobulbar hemorrhage. This condition occurs in less than 1% of midfacial trauma injuries.[140] Retrobulbar hemorrhage typically occurs within the first few hours post-trauma or after surgical repair[141]; however, it has been reported to occur hours to days following the initial injury.[132] The mechanism of injury was described by Hayreh et al[142] and Ghufoor et al,[143] who suggested that retrobulbar pressure results in occlusion of the ciliary arteries that are responsible for blood supply to the optic nerve head. The subsequent optic neuropathy is caused by ischemia.

Signs and symptoms of retrobulbar hemorrhage are pain, proptosis, and decreasing visual acuity. Other indications include ophthalmoplegia, increased intraocular pressure, and papilledema. Ophthalmologic findings in retrobulbar hemorrhage are inconclusive. The cherry-red macular spot of central retinal artery occlusion has been reported by Ord[136] and Nicholson and Guzak.[144]

Timely evaluation, diagnosis, and treatment must be carried out to increase the chance of preserving sight in this patient subgroup. Even though the incidence of retrobulbar hemorrhage is low, it is good clinical practice for all orbital trauma patients to undergo a thorough ophthalmologic examination. Treatment must be instituted as soon as the diagnosis is suspected. Hayreh et al[140] have recommended that treatment be instituted within 90 minutes following the presentation of symptoms.

If the physical signs are unaccompanied by decreased visual acuity, it is mandatory to admit the patient at frequent intervals for observation of visual acuity. There have been reported cases of delayed loss of visual acuity.[141] All patients with an ocular component of midfacial trauma should have regular follow-up and be instructed to return immediately if visual changes occur.

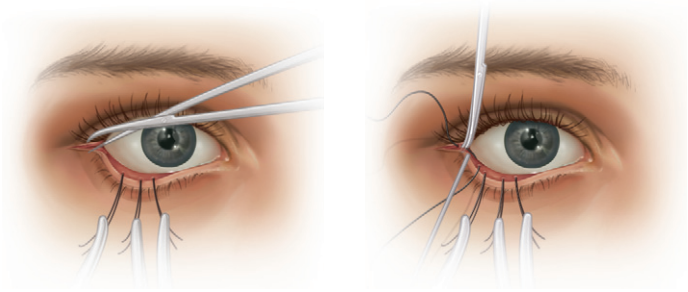
Following diagnosis, medical treatment should be instituted immediately. Intraocular pressure can be reduced rapidly by the administration of supplemental oxygen, 20% mannitol (2 g/kg IV over 30 minutes, with no more than 12.5 g in 3 to 4 minutes); 500 mg of acetazolamide sodium (Diamox) IV, and 1g methylprednisolone sodium succinate (Solu-Medrol) IV.[141,145,146] Medical treatment is aimed primarily at limiting the ischemic insult by dilating the intraocular vessels, reducing intraocular pressure, limiting inflammation and edema, and stabilizing cell membranes. If this does not improve symptoms, surgical decompression should be undertaken. Ord[136] has suggested a waiting period of 30 to 45 minutes. The sooner decompression is performed, the better the prognosis.

The aim of surgical evacuation is to gain access to the bleeding site rapidly and evacuate the developing hematoma. This can be performed by a lateral canthotomy; inferior cantholysis can be performed simply and safely in the emergency room setting. Local anesthetic is injected. The lateral canthotomy is then carried out by placing the medial blade of tissue scissors on the lateral orbital rim and cutting at a 45-degree angle posteroinferiorly. Inferior cantholysis ensures that the inferior crus of the lateral canthal tendon is completely incised, resulting in complete release of the lateral lower eyelid attachment (Fig. 17-38).

An adjunct to this procedure is the placement of an artery clip at the lateral canthus between the upper and lower lids, with advancement toward the lateral orbital rim. This serves to crush the tissue and thereby limit bleeding, and to guide the placement of the lateral canthotomy.[146]

Alternatively, to allow decompression of the expanded hematoma, a lateral brow incision can be followed by subperiosteal dissection; if bleeding is extraperiosteal or intraconal, the periosteum should be incised. The intercanthal space may be entered with fine hemostats after separation of the septum between the inferior and lateral rectus muscles.

Following open evacuation, drains may be placed for 24 hours. Proposed medical follow-up regimens include the following: 20% mannitol, 2 g/kg IV every 8 hours for 24 hours; acetazolamide sodium, 250 mg IV every 6 hours; and steroids, every 6 hours for 2 to 3 days. A topical beta blocker such as timolol maleate eye drops, 0.25% topical solution, 1 or 2 drops daily, has also been suggested to decrease production of aqueous humor.[147] Oral medication is then instituted. Surgical repair of the facial fractures should be delayed until the resolution of visual acuity, positively or negatively.



A B

FIGURE 17-38 Lateral canthotomy and inferior cantholysis.

Traumatic Optic Neuropathy

Treatment of traumatic optic neuropathy is controversial. If a patient loses sight at the time of trauma, it is unlikely to return. Penetrating trauma and subsequent loss of vision have a poorer prognosis than loss of vision secondary to blunt trauma. For the patient with traumatic optic neuropathy, three commonly used treatment modalities have been described—surgical decompression, high-dose steroids, and observation. Wang et al[148] have evaluated 61 patients by an algorithm based on the degree of reduced visual acuity; they used megadose steroids in all patients and optic nerve decompression for patients with no light perception or useless vision at 48 hours. In their series, in those with no light perception, 27% improved after treatment compared with 100% improvement in the light perception or better category. Mine et al have suggested that surgical decompression may benefit the patient whose vision initially could perceive hand motion or more.[149]

Superior Orbital Fissure Syndrome and Orbital Apex Syndrome

Hirschfeld first described the superior orbital fissure and orbital syndrome in 1858.[150] The contents of the superior orbital fissure include, from the apex downward, the lacrimal nerve, frontal nerve, superior ophthalmic vein, cranial nerve (CN) IV, superior division of CN III, nasomaxillary nerve, CN VI, inferior division of CN III, and ophthalmic vein (Fig. 17-39).

Symptoms of the superior orbital fissure syndrome are the result of depletion of its contents. Depending on the location and degree of involvement, they may include loss of sensation over the forehead because of involvement of the frontal branch of CN V, loss of the corneal reflex resulting from the involvement of the nasociliary

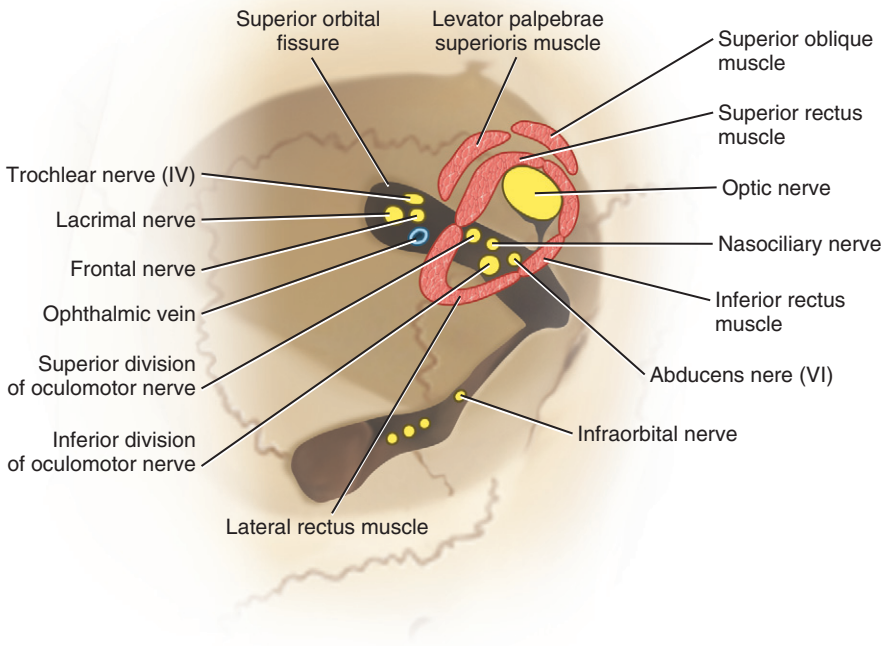


FIGURE 17-39 Anatomy of the orbital apex.

branch of the first division of CN V, a fixed dilated pupil resulting from blockage of the parasympathetic supply carried via CN III, and resultant unopposed sympathetic control of the pupil. Various presentations of external ophthalmoplegia may also be present secondary to involvement of CN III, IV, and VI.

Reflex and accommodation to direct light may be affected because of blockage of the efferent arc by paralysis of CN III and loss of the parasympathetic (motor) root of the ciliary ganglion via CN III. Persistent edema is noted because of venous obstruction of the ophthalmic vein. Proptosis may also occur as a direct result of pressure, antral and extraconal hemorrhage, or secondary to decreased muscle tone caused by compromised motor innervation of the extraocular muscles.

Clinical signs and symptoms guide treatment. If orbital compartment syndrome is suspected, surgical decompression is immediately indicated. This is most easily accomplished by lateral canthotomy and inferior cantholysis, as discussed earlier.

The orbital apex syndrome is a superior orbital apex syndrome with concomitant injury of the optic nerve. This is often the result of a fracture that propagates through the optic canal; it is managed similarly to traumatic optic neuropathy. Decompression of the optic nerve, as proposed by Murakami,[151] is controversial.

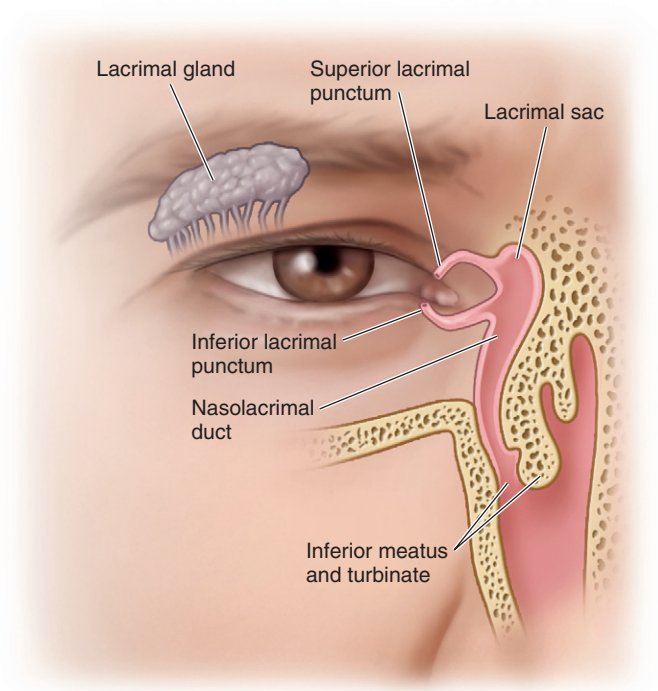


FIGURE 17-40 Anatomy of the lacrimal system.

NEUROLOGIC COMPLICATIONS

The most frequently reported neurologic complication of midfacial trauma is damage to the infraorbital nerve. Lund has reported a 37% incidence of infraorbital nerve disturbance in those who underwent open reduction of the lower orbital rim who experienced higher rates of nerve dysfunction.[152] Schmoker et al have found a 76% incidence of infraorbital nerve dysfunction in the immediate postinjury period, with a 43% incidence of long-term anesthesia.[153] This correlates with Waldhart's report of 70% of patients with orbitozygomatic fractures who experienced early paresthesia; 25% of cases were long term.[154] Haug et al have found infraorbital nerve paresthesia to be the most common complication in 50 patients with maxillary fractures, with an incidence of approximately 24%.[155]

Persistent paresthesia involving the inferior palpebral, lateral nasal, and superior labial regions demonstrates complete nerve involvement. The infraorbital nerve may be damaged at any point but usually as it exits the infraorbital foramen. Care must be taken following reduction of facial fractures or placement of orbital floor grafts to prevent compression on the nerve. If paresthesia does not resolve within 6 months, exploration of the nerve at the infraorbital foramen is indicated, particularly in case of closed reduction.

LACRIMAL SYSTEM

ANATOMY

The lacrimal system can potentially be disrupted by midfacial trauma, especially comminuted NOE fractures. The lacrimal system consists of a lacrimal gland situated in the anterior superolateral portion of the orbit and two lacrimal canaliculi that drain the eye via puncta situated in the medial aspect of each eyelid (Fig. 17-40). From the puncta, the ducts (usually measuring 1 cm in length) travel vertically and then medially to join the lacrimal sac. The sac measures approximately 12 mm and sits within the lacrimal fossa. It is protected laterally and inferiorly by the lateral limb of the medial canthal ligament and medially by the weaker medial limb of the ligament. The lacrimal sac empties into the inferior meatus via the nasolacrimal duct. The duct is approximately 20 mm in length, about 50% of which is incased in bone. The portion of the nasolacrimal system that is most prone to damage is the bony nasolacrimal duct,[156] and 80% of lacrimal secretions are handled by the inferior canaliculus. Therefore, a nonfunctional superior canaliculus will not usually result in epiphora.

The incidence of lacrimal system injury appears to be less than originally hypothesized. Gruss et al[157] have reported on 46 patients with NOE injuries and found that postoperative epiphora is primarily the result of lid malposition and not nasolacrimal obstruction. Of this group, 17.4% required dacryocystorhinostomy. Harris and Fuerste[158] have recommended primary silicone intubation of the disrupted distal lacrimal pathway to prevent future cicatricial obstruction. The tube is left in place for 4 to 6 months. A detailed history and workup are mandatory before one can undertake the reconstruction of the nasolacrimal system effectively.

It should be noted that disruption of the nasolacrimal system is not the sole cause of epiphora. Aging, with resultant pulling away from the puncta, paralysis of CN VII, disruption of the medial canthal ligament, and obstruction of Hasner's valve are all potential causes of epiphora. Epiphora may be secondary to trauma to the region or may be a coincidental finding. A history of

iritis, dacryocystitis, allergies, previous nasal surgery, or tumor resection can indicate a cause that is unrelated to the trauma sustained.

EVALUATION

The physical examination should include an assessment of the puncta for discharge and evaluation of the sac for enlargement, redness, or fistula. One should also look for lagophthalmos, ectropion, and patency of the puncta. The patency of the nasolacrimal system is determined by the Jones I and II tests.[159] The Jones I test is carried out by injecting 2% fluorescein dye into the conjunctival sac and, after 5 minutes, noting whether the dye emerges in the nose. A cotton applicator with 5% cocaine is placed under the inferior turbinate following shrinkage of this region. If no dye is noted on the applicator, the patient should be instructed to blow his or her nose. Alternatively, the head should be placed in a forward position to allow drainage to occur more freely, and not into the pharynx. A contraindication to the performance of a dye test is the presence of dacryocystitis.

If no dye is retrieved from the nose, the Jones II test should be carried out to determine the location of the obstruction in the system. The dye is flushed out of the sac and a cannula is inserted into the inferior canaliculus via an anesthetized punctum. The patient's head is bowed forward and saline is injected into the system. The appearance of fluid in the nose containing the dye indicates a partial blockage that was overcome by the injection. This problem is amenable to surgical correction via dacryocystorhinostomy. Similarly, dacryocystorhinostomy should be performed if there is reflux of fluid from the opposite punctum, indicating that the obstruction exists at or below the level of the nasolacrimal sac.

Intubation dacryocystography is a useful means of determining the exact location of an obstructed system and is an alternative to the Jones I and II diagnostic tests. It demonstrates the location of the disruption and location and size of the sac.

Ashenhurst et al[160] have introduced the technique of combined CT and dacryocystography for lacrimal problems following, for example, midfacial trauma. In this technique, the lacrimal system is injected with contrast and the midface is scanned by CT.

DACRYOCYSTORHINOSTOMY

History and physical examination proceed as described earlier. When the obstruction to flow is distal to the sac, a dacryocystorhinostomy (DCR) should be performed. This technique has undergone a number of variations. Functionally, the procedure bypasses the nasolacrimal duct by anastomosing the lacrimal sac with the nasal mucosa.

This procedure is typically performed under general anesthesia. The nose is packed with gauze impregnated with a vasoconstrictor. An incision is made in the skin overlying the medial canthal ligament, approximately 1cm medial to the inner canthus, extending inferiorly approximately 2 cm and commencing approximately 0.5 cm above the level of the attachment of the medial canthal ligament. A Bowman probe is passed through the lower punctum and canaliculus to enter the sac. This probe demarcates the position of the sac and the location for the formation of the nasal opening. The incision is carried medially and the orbicularis oculi muscle and fascia are incised to reveal the medial canthal ligament. The ligament is exposed and resected, the lacrimal fascia is opened, and the incision is continued inferiorly to expose the lateral and medial aspects of the sac. The sac is dissected free from its bony moorings and the bony ostium is made medial to the lower part of the sac with a 10-mm trephine bur. The lacrimal bone and part of the anterior lacrimal crest are removed. The opening is enlarged with Kerrison's forceps to measure at least 15 mm in length and 10 mm in width. The sac and nasal mucosa are incised longitudinally opposite the ostium. Releasing transverse incisions are carried out superiorly and inferiorly. The posterior nasal and sac flaps are sutured, as are the corresponding anterior flaps. Sutures (4-0 polyglactin 910) are used for the closure. The overlying tissue is closed in layers (Fig. 17-41).

Hollwich[161] has modified the classic technique by suturing the posterior flaps of the nasal mucosa and sac. The anterior mucosal flap of the sac is sutured to the overlying subcuticular skin. Busse has reported a success rate of 84.9% using this technique.[162]

There are a few important factors to keep in mind when performing this procedure. The nasal bone opening must be large enough, its borders must be smooth so that granulomas do not form, and daily lavage with Ringer's solution should be started on the postoperative day 2 and continued for approximately 4 weeks. In the case of an obstruction of the nasolacrimal system that was not diagnosed during the initial facial reconstruction, DCR usually can be performed safely 3 to 4 months after the initial reconstruction.

EMERGING SURGICAL TECHNIQUES AND MATERIALS

ENDOSCOPIC MANAGEMENT OF MIDFACE FRACTURES

Endoscopic surgical techniques have been suggested for smaller incisions, limited dissections, and subsequent decreased recovery times and postoperative pain.[163,164] Endoscopic surgery has also been suggested to limit potential complications of various traditional incisions. In the zygomatic complex region, these include alopecia, blood loss, injury to the facial nerve, and sensory loss to the scalp. In the inferior orbital floor and orbital rim, these include scarring, ectropion, vertical lid shortening, and eyelid edema.[165] Disadvantages include limited exposure, a steep learning curve with potentially longer operating times.[166,167]

These techniques have been suggested for fractures of the zygomatic arch, orbit, frontal sinus, mandibular angle, and subcondyle, among others.[168-171] Equipment used with endoscopic surgery necessitates the use of an endoscope with an overlying sheath to create an optical cavity for adequate visualization. Additionally, a video system composed of a camera, light source, camera converter, and monitor is necessary. Approach, technique, and applications for endoscopic surgery are similar to

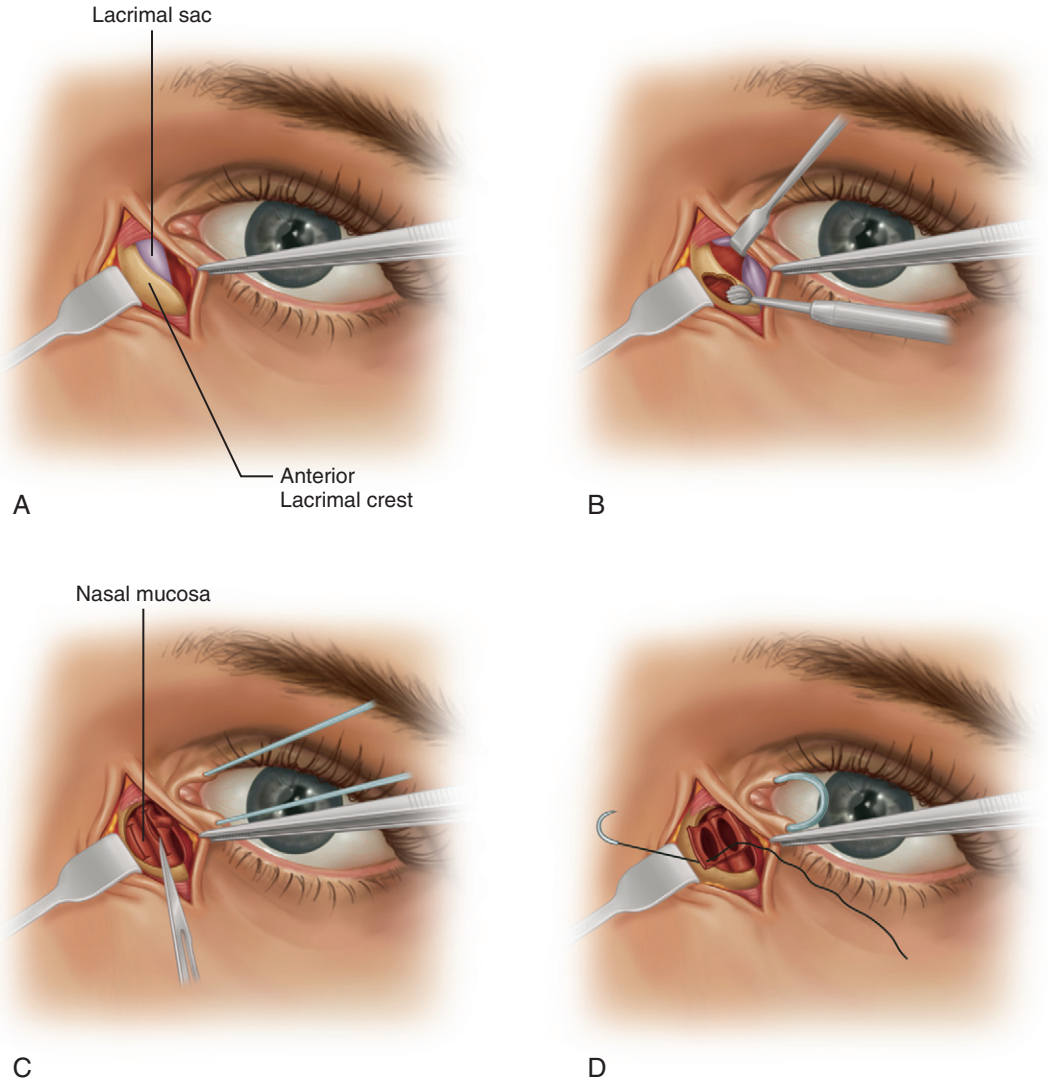


FIGURE 17-41 Technique for DCR.

those used in routine endoscopic craniomaxillofacial surgery.

ADVANCES IN COMPUTER-BASED IMAGING TECHNIQUES

Computer-aided craniomaxillofacial surgery can be divided into three interrelated categories—computer-aided presurgical planning, intraoperative navigation, and intraoperative CT–magnetic resonance imaging (MRI).[172] Advances in presurgical planning include the use of stereolithographic models, which can help guide preoperative plate contouring and precise positioning of plates and fractured segments. Computer-assisted surgical simulation without physical models can also be used to plan and evaluate surgical moves virtually. Three-dimensional CT scans can be used to mirror a contralateral unaffected side and to superimpose the image precisely over the fracture site to provide an accurate template reconstruction.[173] Tucker et al, in a recent study comparing postsurgical outcomes with three-dimensional surgical simulations on 14 patients undergoing orthognathic surgery, have found no statistical difference between computer-generated predictions and postsurgical results[174] (Fig. 17-42).

Intraoperative navigation has recently become available commercially for midfacial surgery and is a useful adjunct to certain common maxillofacial procedures. This technique requires the establishment of fiducial markers or laser surface scanning to align the three-dimensional scan with the navigational system for accurate intraoperative manipulation.[175] These systems have been shown to allow precise positioning of fractured segments secondary to facial trauma.[176]

Intraoperative cone beam CT has shown to be an effective way to allow immediate confirmation of appropriate reduction of fractured segments, particularly in complex facial fracture patterns.[177] This technology also limits the need for revisional surgery with a second general anesthetic in case of an initial suboptimal reduction. Tsiklakis et al have mentioned an 8- to 10-fold reduction in dosage when using cone beam CT in place of conventional CT.[178] This is particularly important



FIGURE 17-42 Stereolithographic model.

when considering the use of multiple scans for the evaluation of postoperative reduction.

Scolozzi and Terzic have recently reported on a small series of orbitozygomatic fracture repairs using a sequence of computer-based planning, bone repositioning using a navigational system, and intraoperative assessment of fracture repair by mobile C-arm cone beam CT, with satisfactory results.[173]

Further study is needed in regard to cost-benefit analyses and outcomes. However, in conjunction with traditional sound surgical principles, these advances in treatment planning, repair, and intraoperative evaluation will likely provide powerful adjuncts to future management of midfacial injuries.

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CHAPTER 18

Ophthalmic Consequences of Maxillofacial Injuries

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OUTLINE

Ophthalmic Assessment
- *History*
- *Clinical Examination*
- *Examination for Structural Disorders*

Minor Eye Injuries
- *Subconjunctival Hemorrhage and Bruised Eyelids*
- *Corneal Abrasion or Corneal Foreign Body*

Nonperforating Eye Injuries
- *Conjunctiva and Cornea*
- *Anterior Chamber*
- *Abnormal Depth of the Anterior Chamber*
- *Iris And Pupil*
- *Angle Recession*
- *Lens*
- *Ciliary Body*
- *Retinal Injury and Choroidal Injury*

Perforating Eye Injuries

Perforating Injuries of the Orbit

Retrobulbar Hemorrhage
- *Clinical Features*
- *Management*

Traumatic Optic Neuropathy

Disorders of Ocular Motility
- *Disorders of Central Control of Eye Movement*
- *Cranial Nerve Injury*
- *Ocular Motility Disorders Caused by Orbital Injury*
- *Blowout Fracture*
- *Fractures of the Orbital Roof*
- *Trauma to the Trochlea*

Displacement of the Globe
- *Proptosis*
- *Enophthalmos*
- *Vertical Displacement*
- *Horizontal Displacement*
- *Traumatic Herniation of the Globe into the Maxillary Sinus*

Injuries to the Eyelids
- *Eyelid Swelling and Hematoma*
- *Eyelid Lacerations*
- *Eyelid Avulsion*
- *Progressive Shortening of the Lower Eyelid*

Nasolacrimal Injuries
- *Canalicular Lacerations*

Indirect Ophthalmic Consequences of Injury
- *Traumatic Retinal Angiopathy (Purtscher's Retinopathy)*
- *Caroticocavernous Sinus Fistula and Arteriovenous Anastomosis*
- *Facial Palsy*
- *Papilledema*

Relationship Between Maxillofacial and Eye Injuries

The globe is protected from injury by a number of structures and mechanisms, including the prominence of the bones of the orbit and the natural reflexes of self-protection—namely, blinking, averting the head, and protecting the eye with the hand or forearm. Despite these factors, the eye may sustain injury, but the resilient structure of the globe allows it to withstand blows of considerable force without rupture.

Both prospective and retrospective studies of patients who have sustained midfacial fractures indicate that as many as 40% may sustain serious ocular injury that warrants ophthalmologic referral.[1-8] A recent study[9] has demonstrated that up to 91% of patients with orbital fractures who had an ophthalmic evaluation within 1 week of their injury sustained some form of ocular injury. Many of these were classified as mild but 45% were deemed to be moderate or severe injuries.

Some ophthalmic injuries may be clearly apparent. However, other potentially blinding complications can easily be missed unless they are actively sought. Inadequate care can result in blindness, with its attendant social and medicolegal implications. Examination of the eyes is mandatory for every patient who has sustained midfacial trauma severe enough to cause a fracture. This chapter reviews methods of ophthalmic examination and the ophthalmic consequences of injury and provides guidelines for ophthalmologic referral.

OPHTHALMIC ASSESSMENT

The assessment comprises the history, evaluation of visual function, and examination for structural disorders.

HISTORY

The following data are recorded:

1. The time, place, and circumstances of the injury
2. The exact nature of the injury
3. The nature of the object that caused the injury (e.g., a kick is more likely to damage the eye than a head butt)
4. The velocity and vector of the traumatic force
5. Whether glasses were worn (the glasses may have protected the eye or may have given rise to a glass foreign body entering the eye)
6. The antecedent visual status

How good was the vision before the injury? This information may be important, especially if legal claims are involved. If patients were in the armed forces, they would have had good vision in each eye. If they had visited an optometrist or ophthalmologist in the past, a record of visual acuity may exist.

CLINICAL EXAMINATION

Assessment of Visual Function

At the time of initial assessment, visual acuity, which is a measure of the resolving power of the eye, is determined in every case of fracture of the midface if possible. Distance acuity is assessed with the patient at 6 m (20 feet) from a Snellen chart. The test letters are constructed so that the edges of the lines composing the letter subtend a visual angle of 1 minute of arc when they are a certain specified distance away. The complete test letter subtends a total angle of 5 minutes of arc at the eye, for an eye with 6/6 (20/20) vision.

Visual acuity is recorded as a fraction. The numerator denotes the distance of the patient from the chart and the denominator the line that he or she sees at this distance. For example, the top letter of the Snellen chart subtends 5 minutes of arc at the eye when read from 60 m (200 feet). If the patient can read only the top letter of the chart, the visual acuity is 6/60 (20/200).

One eye must be fully covered while acuity is determined. If the patient cannot read at 6/6 (20/20) and yet does not have glasses for distance, acuity is measured with the patient looking through a pinhole; a device for this purpose can be easily improvised with a card and a pin. If acuity improves, the most likely cause of the poor acuity is a refractive error. Occasionally, acuity improves in patients with cataracts or opacities in the vitreous.

When visual acuity is less than 6/60 (20/200), the distance at which the top letter can be read is recorded (e.g., 3/60 or 10/200). When the chart cannot be read, the patient is asked to count fingers (CF), and the distance at which this task is achieved is documented (e.g., CF, 0.5 m). If acuity is less than this, the perception of hand movements is recorded as HM or the perception of light only as PL.

In some patients with multiple injuries, it may be possible to assess only the visual acuity for near vision. The reduced Snellen letters subtend the same angle at the eye at 0.33 m as the full Snellen letters at 6 m.

For convenience, the clinician can carry a means of assessing visual acuity for near vision in her or his pocket. For older patients, near acuity must be determined with the use of reading glasses or a pinhole (Fig. 18-1) because the eye's ability to accommodate declines with age. If a formal means of visual acuity assessment is not available, the clinician can estimate visual acuity using a newspaper or paper currency.

Visual Acuity in Children

It is equally important to assess visual acuity in a child who has sustained a facial injury. The most practical method for a child who is unable to read letters is the Sheridan-Gardiner test. Single Snellen letters are shown to the child from a distance of 6 m (20 feet), and the child points to the identical letter on a chart held by a second examiner or a parent who is sitting with the child.

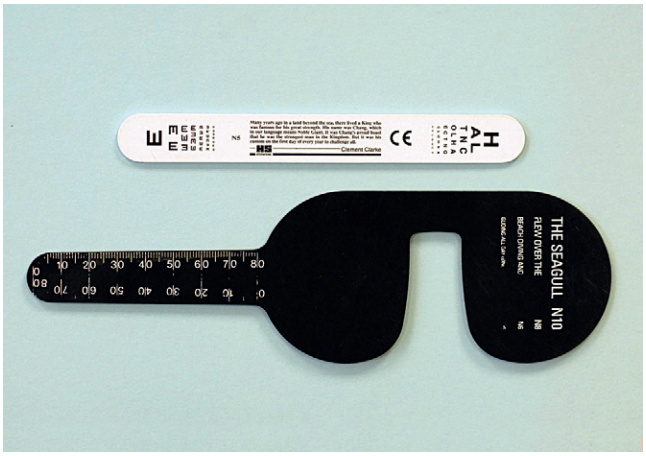


FIGURE 18-1 Pinhole occluder and portable visual acuity device for practical assessment of visual acuity in the injured patient. When presented at 0.33 m, each line of letters approximates to the near equivalent of 6/60 (20/200), 6/36, 6/24, 6/18, 6/12, 6/9, and 6/6, respectively. (Courtesy Clement Clarke International, London.)

Visual Fields

Visual fields are assessed in patients who have sustained severe head trauma, in those who are aware of a defect in their vision, and in those whose behavior indicates that a visual field defect may be present. Confrontation methods of visual assessment are most commonly used to screen for a visual field defect. However, more sensitive methods must be used if a minor defect is to be detected. We recommend the following strategy.

Testing Central Visual Function. The patient is instructed to look at a red object with each eye in turn, and he or she is asked to compare the color for each eye. A patient with traumatic optic neuropathy will be aware of color desaturation (i.e., the red will look duller with the affected eye). Unequal pupils preclude such light brightness assessment because an enlarged pupil increases apparent brightness. Next, the patient is told to look at the examiner's nose with each eye in turn and is asked whether any part of the examiner's face appears to be missing or blurred. This method is accurate for detecting a paracentral scotoma, which, for example, may be caused by a choroidal tear.

Binocular Visual Field Testing by Quadrants. This method is used to test for homonymous visual field defects. The examiner sits opposite the patient at a distance of 1 m (3 feet). The patient is asked to look at the examiner's eyes. Both hands are placed in the lower outer quadrants and then in the upper outer quadrants. The patient is asked to identify a small movement of the extended forefinger of each hand. The examiner moves each finger in turn and then moves both fingers together. The patient is asked to point at the moving finger (or fingers). A patient with a left homonymous hemianopia, in which the left field of vision in each eye is deficient, will not point to the moving fingers on the left. A patient with a visual inattention defect in that area will not perceive

movement when the finger is moved in the outer half of the visual field at the same time. Inattention hemianopia is indicative of unilateral diffuse occipital pathologic conditions.

Assessment of the Central Visual Field to Confrontation. Traumatic damage to the visual pathways is more likely to cause impairment of the central 30 degrees of the visual field than of the periphery. Therefore, a small target, such as a small red pin, should be used to screen for such defects. The examiner sits opposite the patient and closes one eye. The examiner asks the patient to cover her or his corresponding eye with the palm of the hand and to fixate on the examiner's open eye. The red target is introduced from the periphery to the center along a coronal plane halfway between the examiner and the patient. The patient is instructed to say "now" as soon as he or she becomes aware of the head of the pin. The examiner is specifically looking for a quadrantic field loss; therefore, the pin is introduced into the fields along the oblique meridians (if the examiner tests only in the horizontal and vertical meridians, she or he may miss the field defect).

To determine the sensitivity of the technique, the examiner checks the position and dimensions of the blind spot by placing the target in his or her own blind spot. The examiner's blind spot should correspond approximately to that of the patient.

Testing Peripheral Visual Field. The examiner tests peripheral vision using a large white pin. The patient is asked to cover one eye. Sitting opposite the patient, the examiner introduces the target from behind the patient and moves it in an arc of an approximate radius of 0.33 m (1 foot), centered on the patient's eye. The target should be identified as soon as it comes into the extreme peripheral field of vision. This technique can be modified for use in children. One eye of the child is patched. The child is given a toy to play with. The examiner stands behind the child and introduces the target into the periphery. As soon as it is seen, the child turns his or her head to look at the target.

Subjective Visual Field Assessment. Occasionally, all these tests may be normal, but the patient still complains of impaired vision. The examiner sits opposite the patient. The examiner closes one eye and covers the corresponding eye of the patient. The patient is asked to fixate on the examiner's pupil, and the examiner places the red pin close to the patient's face in each quadrant of the patient's visual field adjacent to the examiner's eye. The patient is asked to compare the colors in each position. In particular, in cases of traumatic chiasmatic damage, the patient is aware of color desaturation in the upper temporal fields, but no other detectable visual field defect may be noted with any of the other methods used.

When a visual field defect is detected, more accurate charting of the defect by perimetry may be necessary to determine the pattern and extent of the defect.

Pupils

If the patient's visual acuity is reduced and shows no improvement with use of the pinhole, the pupils are tested to seek evidence of an afferent pupillary defect. This occurs in defects in the visual pathway anterior to the chiasm, gross retinal detachment, and traumatic optic neuropathy.

Direct and Consensual Pupillary Reflexes. The background illumination is diminished by switching off the lights or drawing the curtains. The patient is asked to fixate into the distance to relax his or her accommodation. A penlight source is used to illuminate the eyes from below, but not from the front, because such lighting could cause an accommodative reflex. The light is shined twice into one eye, and first the direct and then the consensual reflexes are observed. The procedure is repeated for the other eye. An obvious afferent pupillary defect, in which the pupil reacts poorly to direct stimulation but briskly to consensual stimulation, can be detected by this method.

Swinging Flashlight Test. This test is used to detect a subtle defect caused, for example, by incomplete optic nerve damage. The pupils are illuminated in the same manner, but on this occasion the light is shined into each eye for about 2 seconds and then swung rapidly to illuminate the other eye. For an incomplete right afferent pupillary defect, when the light is shined into the right eye, both pupils constrict. When the light is swung to the left eye, both pupils constrict further. When the light reilluminates the right eye, the pupils return to their previous resting position and dilate slightly. This technique can be used even in the presence of a unilateral third nerve palsy, in which one pupil is poorly reactive or nonreactive. The swinging flashlight test is performed and the size of the contralateral pupil is determined for both its direct and its consensual reflexes. Any difference in size indicates a relative afferent pupillary defect.

For example, a fracture at the right orbital apex may damage the right oculomotor and optic nerves. The right pupil would, therefore, not react directly or consensually because of the oculomotor nerve damage. However, the diameter of the left pupil will be smaller for its direct response than for the consensual response from illuminating the right eye.

EXAMINATION FOR STRUCTURAL DISORDERS

Examination of the Anterior Segment

Careful examination of the anterior segment of the eye is essential if clinical signs of ocular trauma are to be detected. Ideally, a slit lamp microscope should be used. Operating loupes with focal illumination provide a useful alternative. The anterior segment of the eye is examined carefully for any of the pathologic conditions described in the next section.

Ophthalmoscopy Through Dilated Pupils

This test is indicated for all patients with reduced visual acuity. Tropicamide 1% produces rapid pupillary dilation with little effect on accommodation and with a return to normal within 3 hours. The addition of phenylephrine 10% may be necessary for those patients with pigmented irides the examiner must check first for any history of cardiac dysrhythmia or systemically administered monoamine oxidase inhibitors.

The contraindications to pupillary dilation are as follows:

1. An iris-supported intraocular lens, which could be dislodged if the pupil were dilated
2. A history of intermittent blurring of vision and pain in the eye, suggestive of angle-closure glaucoma
3. A shallow anterior chamber

Although the optic disc can be assessed without dilating the pupil, the central and surrounding retina cannot be adequately examined.

The reader will no doubt be conversant with the normal appearance of the retina (Fig. 18-2) and the use of the direct ophthalmoscope. The following hints may, however, be of value:

1. The examiner looks through the ophthalmoscope from a 0.33-m (1-foot) distance, examining the red reflex initially. By this means, the examiner can identify any opacities in the media—for example, vitreous hemorrhage or traumatic cataract.
2. The patient is asked to fixate into the distance with the other eye. If the patient focuses for near vision, the examiner will have difficulty in focusing the ophthalmoscope.
3. If a bright light reflex gets in the way, the light is reflecting from the cornea. If the ophthalmoscope is rotated very slightly, the light reflex will diminish or disappear, because it will no longer be reflected back along the examiner's visual pathway.
4. To observe a scene through a keyhole, the eye needs to be placed close to the keyhole. The same principle applies to ophthalmoscopy—the closer to the patient's eye is the examiner, the wider the angle of view.
5. To examine the fovea, the patient is asked to look at the light.
6. To examine the peripheral retina, the patient is asked to move her or his eyes in sequence in different directions. When the patient looks up, the examiner is looking at the upper retina as it is brought down into view; the same principle applies to the other positions of gaze.

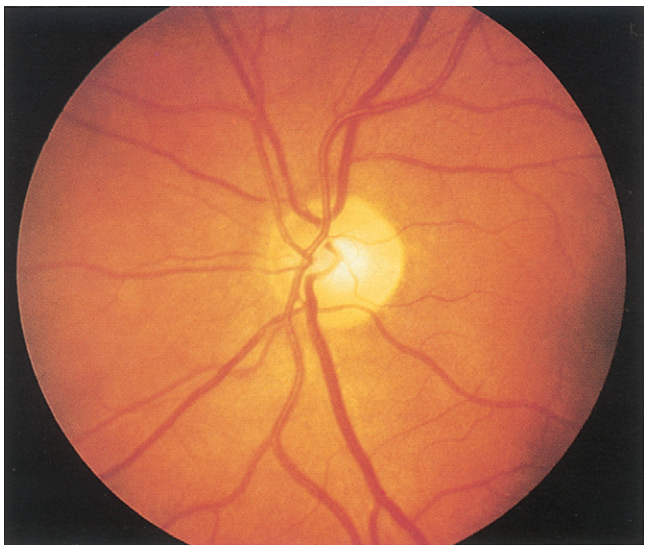


FIGURE 18-2 The normal optic disc and retina.

Examination of Eye Movements

Eye movements are commonly impaired following facial and head injury. It must be remembered, however, that an antecedent squint is not uncommon. Moreover, ptosis, blurred vision as a result of the eye injury, amblyopia, and a history of patching of the eye in childhood all may prevent the patient from experiencing double vision. Eye movements are therefore objectively assessed in all patients who have sustained an injury likely to be complicated by a motility disorder (e.g., a blowout fracture). Figure 18-3 indicates the primary directions of action of each of the extraocular muscles. The eye movements into each of these positions of gaze are examined.

The assessment of eye movements is a skilled procedure. The following strategy is suggested as a means of identifying patients who warrant referral.

The examiner sits directly opposite the patient and uses a penlight to examine the eye movements. The penlight is moved in a manner similar to that for peripheral visual field testing. The light is held at approximately 0.33 m (1-foot) from the patient. The examiner observes the exact position of the light reflexes on the cornea with respect to the pupil. The patient is asked to follow the light. The light is moved in an arc into each position of gaze, with the light constantly directed at the eyes. The symmetry of the light reflexes and symmetry of the positions of gaze are closely examined (see Fig. 18-3). The skilled observer is able to detect most motility disorders.

The cover-uncover test is performed while the patient fixates on the light in the primary position of gaze and in the positions of gaze in which double vision is experienced and a motility disorder has been detected. An eye occluder or a piece of a card is used. The examiner watches one eye and covers the other one. The eye that the examiner is watching should not move. If the eye does move to look at the light, it is a squinting or deviated

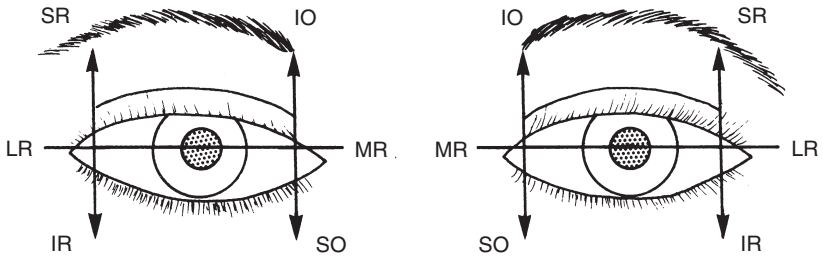


FIGURE 18-3 Actions of the extraocular muscles. *IO,* Inferior oblique; *IR,* inferior rectus; *LR,* lateral rectus; *MR,* medial rectus; *SO,* superior oblique; *SR,* superior rectus.

eye. The procedure is repeated for the other eye. This method provides an objective means of validating and quantifying the patient's subjective double vision.

Forced Duction Test. This test can be performed on a patient with a motility disorder in whom the differential diagnosis between entrapment and muscle weakness is in doubt. Topical local anesthetic (e.g., benoxinate) is instilled into both eyes. The conjunctiva in line with the muscle in question is grasped just adjacent to the corneoscleral junction (the limbus) with a pair of fine-toothed forceps and the globe is gently rotated. The procedure is repeated for the other eye to allow a comparison between both eyes to be made. The force required for rotating the globe is estimated in relation to the normal contralateral eye. Tethering of the globe is indicative of entrapment.

An alternative means of rotating the globe is to use a cotton swab (cotton-tipped applicator) soaked in local anesthetic and to rotate the globe by pressing the swab onto the eye and applying a tangential force. With practice, this method can be equally sensitive and is less likely to cause subconjunctival hemorrhage. However, it should be noted that in our experience, not all patients will tolerate the forced duction test.

Position of the Globe

In every case of facial fracture, the position of the eye should be carefully examined. The eyes may be displaced in any one of three dimensions.

Horizontal Displacement. This displacement is measured by comparing the distance from the center of the bridge of the nose to the center of the pupil on each side.

Vertical Displacement. Vertical displacement is most easily assessed with the use of a short, clear plastic ruler, which is held horizontally with reference to symmetrical landmarks, such as the eyebrows. Any vertical displacement of one eye with respect to the other is then measured with a second ruler. A vertical displacement of the globe may be misinterpreted as a squint; however, the light reflexes are symmetrical and there is no diplopia.

Anteroposterior Displacement: Enophthalmos or Exophthalmos. This displacement is most accurately measured with an exophthalmometer. These instruments, however, use the lateral orbital margin as the reference point, which precludes their use in patients in whom there is bone displacement or asymmetrical swelling of the soft tissue of the orbital margin. An alternative means of assessment in these cases is to examine the patient from above, comparing the positions of the corneas with respect to the supraorbital margins. With experience, a fairly accurate assessment can be made with this method.

MINOR EYE INJURIES

SUBCONJUNCTIVAL HEMORRHAGE AND BRUISED EYELIDS

Subconjunctival hemorrhage (Fig. 18-4) with bruised eyelids commonly follows midfacial injury. Blood may track forward from an orbital injury, or bleeding may take place locally. A clear demarcation line to the bruising of the eyelid suggests orbital hemorrhage. Such bruising is usually benign, but it may be related to severe ocular injury. Careful examination of the eye is required in every case.

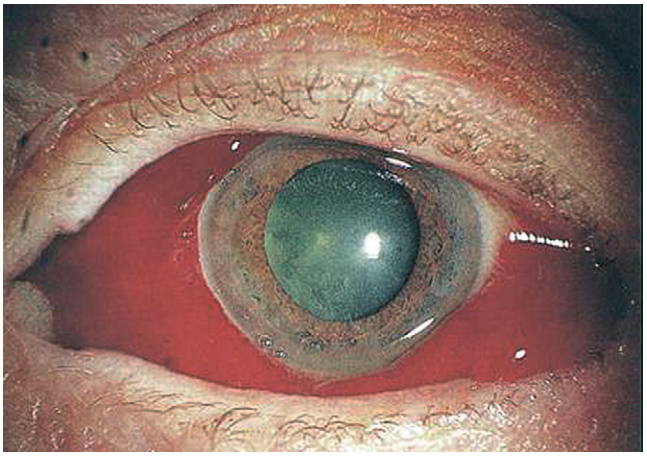


FIGURE 18-4 Subconjuctival hemorrhage.

CORNEAL ABRASION OR CORNEAL FOREIGN BODY

This injury causes severe pain, blurring of vision, photophobia, and lacrimation, except in the presence of corneal anesthesia. Loss of the corneal epithelium may be caused by direct injury to the eye or inadequate eyelid closure as a result of facial palsy, eyelid laceration, or injury during surgery. Alcohol-based skin preparations, incomplete eyelid closure, and accidental injury to the cornea during surgery all must be carefully avoided. When such a lesion is suspected, fluorescein stain allows a diagnosis to be made.

Treatment

After the administration of one drop of topical local anesthetic, which allows clinical examination and gives temporary pain relief, a medium-acting cycloplegic agent such as cyclopentolate (24 hours), which alleviates pain caused by ciliary spasm, and a topical antibiotic such as chloramphenicol are instilled. If a foreign body is present, it is removed. A corneal foreign body is removed with great care, preferably using binocular magnification. The physician can usually lift off a foreign body on the surface of the cornea by using a hypodermic needle held tangentially to the corneal surface, ensuring throughout the procedure that the patient is unable to move forward toward the needle. One drop of a topical nonsteroidal anti-inflammatory preparation is then instilled to alleviate pain.[10]

NONPERFORATING EYE INJURIES

A blunt injury severe enough to cause an orbital fracture may also damage the eye. Depending on the nature, direction, and force of the injury, any anatomic component of the globe may be disrupted. The effects of blunt injury can be divided into those resulting from distortion and those resulting from concussion. Both types of injury are commonly seen in the same eye.

A high-speed anteroposterior force results in marked distortion of the globe (Fig. 18-5). The eye is transiently deformed, with notable distention in the coronal plane and shortening of the anteroposterior dimension. The sclera is inelastic and the aqueous and vitreous cannot be compressed. The iris, ciliary body, zonule of the lens, and peripheral retina may be torn from their insertions and, in severe cases, the sclera may rupture. Distortion of the posterior segment of the eye can result in a tear of the choroid associated with subretinal hemorrhage and, in the most severe case, avulsion of the optic nerve from the globe.

The concussional component of the injury results from a coup-contrecoup effect. The cells of the cornea, lens, retina, and choroid all are susceptible to such injury and may transiently or permanently cease to function.

In this section, the results of injury to each component of the eye are discussed separately. However, almost any combination of injuries can occur, which can occasionally result in disorganization of the structures of the globe (Fig. 18-6).

CONJUNCTIVA AND CORNEA

Swelling of the conjunctiva (chemosis) is common in association with subconjunctival hemorrhage and resolves spontaneously. A tear of the conjunctiva is suggestive of a more severe blunt injury. In every case, internal injury to the globe must be sought.

Loss of the corneal epithelium (Fig. 18-7) is fairly common and causes the same signs and symptoms as a corneal erosion. The corneal endothelium is comprised of a monolayer of cells that probably do not replicate following injury. Their function is to maintain the clarity of the cornea by pumping water out of the cornea and into the anterior chamber. Damage to the corneal endothelium results from a combination of contusion, reactive inflammation, and raised intraocular pressure.[11] This condition may culminate in permanent edema if the endothelial cell population is reduced below a critical level. Recovery of corneal clarity can take place in some patients after a number of months. However, if corneal edema is persistent, a penetrating corneal graft may be required to restore visual function.

ANTERIOR CHAMBER

The anterior chamber comprises the space between the cornea and iris and is occupied by aqueous fluid secreted from the ciliary processes. Blunt trauma can result in bleeding in the anterior chamber, or hyphema (Fig. 18-8), and inflammation.

Hyphema

Hyphema, or bleeding in the anterior chamber, probably results in most cases from tearing of blood vessels at the root of the iris.[12] When the patient is upright, the blood settles at a fluid level, the height of which should be

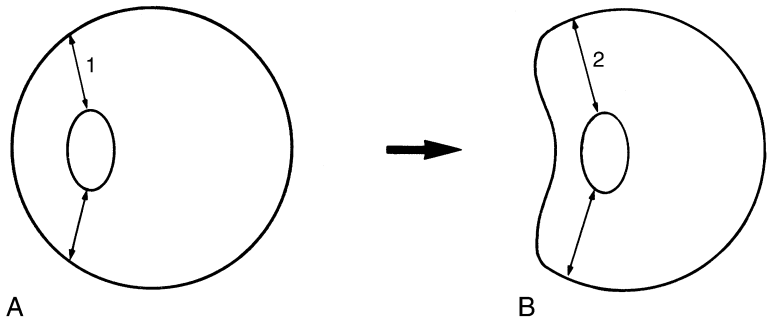


FIGURE 18-5 **A, B,** Distortion of the eye produced by anteroposterior injury to the globe. During the brief period of distortion, the coronal diameter is elongated and the zonule, iris, ciliary body, and vitreous adhesions to the peripheral retina are stretched *(arrows 1 and 2),* resulting in lens subluxation, angle recession or iridodialysis, cyclodialysis (rare), and retinal dialysis, respectively.

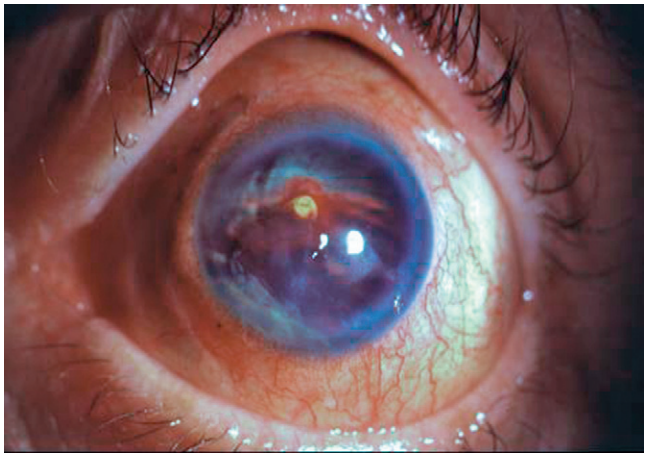


FIGURE 18-6 Disorganized anterior segment. The normal structures within the anterior chamber are not recognizable.

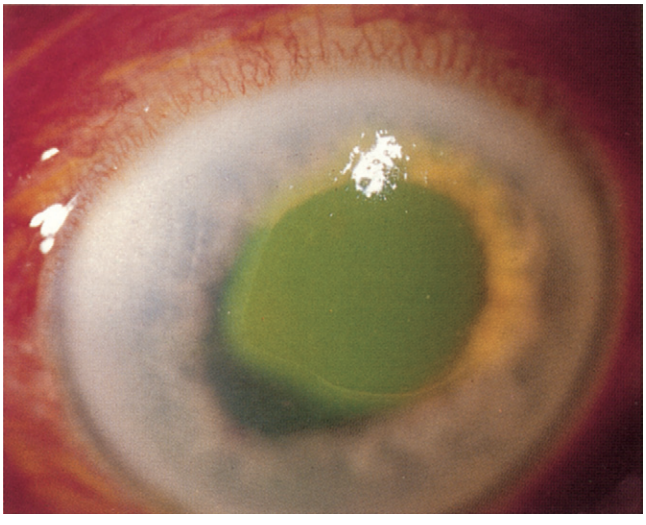


FIGURE 18-7 Corneal erosion stained with fluorescein dye. There is also a diffuse subconjunctival hematoma caused by the injury.

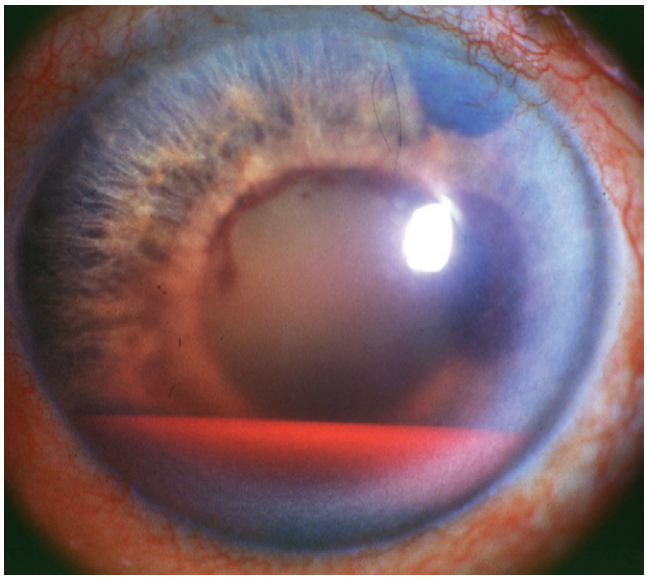


FIGURE 18-8 Horizontal fluid level of blood in the anterior chamber (hyphema) secondary to blunt eye injury. An iridodialysis is also present superiorly.

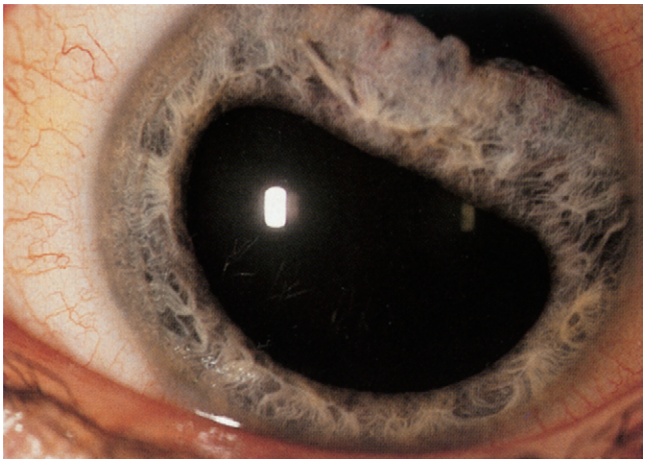


FIGURE 18-9 Iridodialysis. The superior iris has been ripped from its insertion by a blunt compressive eye injury.

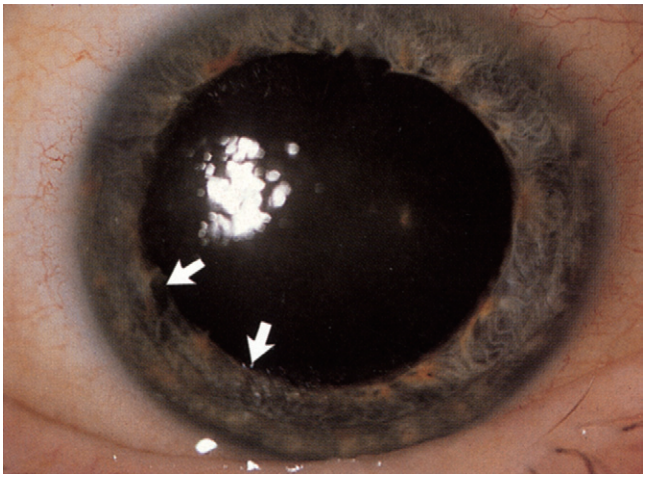


FIGURE 18-10 Traumatic mydriasis. *Arrows* indicate the points at which the pupil sphincter has been torn.

measured daily. The amount of bleeding is related to the long-term prognosis.[13] A history of blurring of vision after the patient lies down, which gradually clears spontaneously when the patient is erect, indicates that a small hyphema may have been missed. Most patients with hyphema are admitted to the hospital and observed. In most cases, spontaneous resorption of hemorrhage takes place but, in a small proportion, rebleeding can occur. Severe hemorrhage may be associated with impairment in the drainage of aqueous, which leads to a raised intraocular pressure. Under these circumstances, bloodstaining of the cornea may take place and surgical intervention may be necessary.

Traumatic Iritis

Traumatic iritis is common. Injury to the iris results in the release of protein and inflammatory cells into the anterior chamber, which can be observed by means of a slit lamp microscope. Traumatic iritis is treated by using topical steroids (e.g., dexamethasone, betamethasone) and a pupillary dilator (e.g., atropine). This treatment prevents the iris from sticking to the lens behind (posterior synechia).

ABNORMAL DEPTH OF THE ANTERIOR CHAMBER

On macroscopic examination, the anterior chambers are compared by viewing each eye obliquely from the side to provide an approximate estimate of depth. The slit lamp is used to obtain a more accurate assessment. Shallowness of the anterior chamber may suggest hemorrhage into the choroid, a choroidal detachment, swelling of the lens, or leakage of aqueous through a penetrating wound. Deepening of the anterior chamber suggests that the lens may have subluxated or dislocated posteriorly.

IRIS AND PUPIL

An iridodialysis (Fig. 18-9) results when the iris is ripped from its root. This condition is clearly apparent on direct ophthalmoscopy because a red reflex is seen through the tear and through the pupil.

Traumatic mydriasis (Fig. 18-10) is common following blunt eye injury. In this case, the pupil fails to react directly and consensually. Pupil sphincter rupture can be seen with the slit lamp. This injury produces a widely dilated pupil and is permanent. On the other hand, a mid-dilated pupil without a pupil sphincter rupture ordinarily recovers its function during the ensuing weeks.

A spastic miosis (small pupil) is common after less severe blunt injury to the globe. In such cases, the miosis may be accompanied by a transient spasm of accommodation brought about by axonal reflexes. This spasm may be followed within minutes or hours by a paralytic mydriasis.

Care should be taken to exclude other causes of pupillary dilation, such as an antecedent Adie's pupil and, in the unconscious patient, a palsy of cranial nerve III

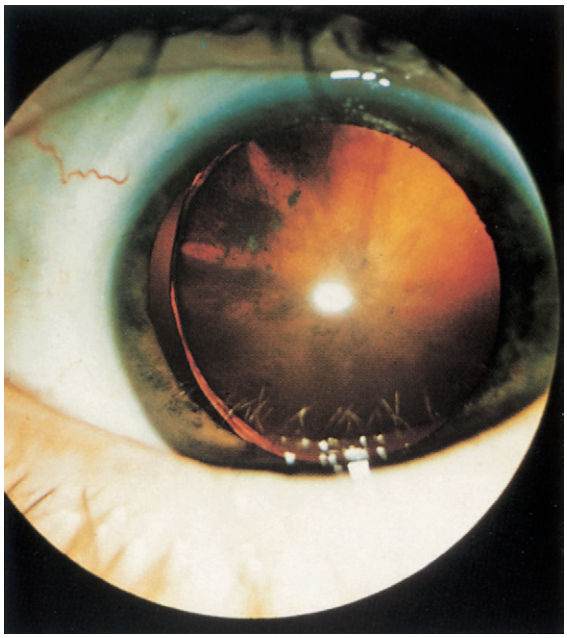


FIGURE 18-11 Subluxation of the lens. The edge of the lens is seen to be distinct from the dilated pupil margin.

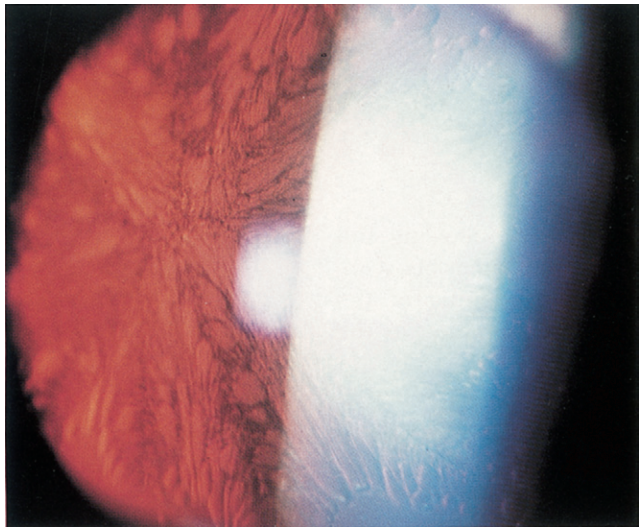


FIGURE 18-12 Traumatic posterior subcapsular cataract. This red reflex photograph illustrates the sunflower appearance that characterizes this type of cataract.

resulting from intracranial injury, with tentorial herniation of the cerebrum.

ANGLE RECESSION

In some cases, the iris may be partially stripped from its root without being torn completely. In severe injury, this tearing may take place for the 360-degree circumference of the eye. Lesser degrees of angle recession are more common.

This injury results in damage to the cells that line the surface of the trabecular meshwork. As the name implies, this structure is like a fine three-dimensional web or sponge through which the aqueous passes into the canal of Schlemm and then returns to the bloodstream. Subsequent fibrosis and scarring in the region of the angle can diminish the outflow of aqueous, thereby resulting in increased intraocular pressure and the development of secondary glaucoma.[14,15] Angle recession occurs at the time of injury and may be seen by gonioscopic examination (slit lamp examination with the use of a prism system to see into the angle) permanently thereafter. It can, however, be easily missed if this examination is not carried out. The medicolegal implications are obvious.

LENS

Subluxation of the lens (Fig. 18-11) is most easily diagnosed following full dilation of the pupil. The margin of the subluxated lens can then be seen. Additional clinical signs include a reduction in visual acuity, deepening of the anterior chamber, and wobbling of the iris, which is seen as a fine shimmer as the patient moves the eye (iridodonesis).

In severe cases, the lens may be dislocated. In this situation, it lies freely in the vitreous and an aphakic correction is required to allow the patient to see. Rarely, the lens may dislocate into the anterior chamber. This dislocation is an ophthalmic emergency because it may interfere with the flow of aqueous and cause an unusual form of acute glaucoma.

Both subluxation and dislocation of the lens result in a change in the patient's refraction. However, in such cases, the injury is likely to have been severe enough to give rise to other causes of visual impairment. In addition, the lens is likely to develop opacities (cataract) with time.

Small, discrete subepithelial and subcapsular lens opacities or a rosette-shaped posterior subcapsular cataract (Fig. 18-12) may develop shortly after severe concussive injury. In serious cases, the pupillary margin is impacted on the anterior surface of the lens and leaves a pigmented ring (Vossius' ring) deposited on the anterior lens capsule. The presence of such a ring indicates that the injury has been very severe and that the eye should be thoroughly examined for other pathologic conditions.

Rupture of the lens capsule may also take place. This rupture allows aqueous humor into the lens, which then becomes opalescent. Release of soft lens matter into the eye can result in severe inflammation. Lens protein is sequestered from the immune system during embryonic development and is thus perceived by the immune system as foreign. This situation can result in a severe inflammatory reaction in the eye, with raised intraocular pressure. Surgical removal of the lens is indicated in these cases.[16]

CILIARY BODY

The functions of the ciliary body are to produce aqueous humor, which provides nutriment for the eye and maintains the intraocular pressure, and brings about accommodations of the lens. When the ciliary muscle contracts, the zonule relaxes and the lens adopts a more biconvex shape, which causes the eye to focus for near vision.

Damage to the ciliary body can therefore result in impairment in the formation of aqueous and a consequent reduction in intraocular pressure. Moreover, injury

to the ciliary muscle gives rise to impairment in accommodation, which is a fairly common short-term sequela of eye injury.[17] The patient complains that he or she has difficulty in focusing, and clinical examination reveals that the near point of accommodation (the nearest point at which he or she can focus clearly) is farther away than before the injury. No specific treatment exists for either of these conditions. The intraocular pressure is normally restored during the few days following injury, and accommodation similarly recovers spontaneously in most cases.

Traumatic cyclodialysis refers to the tearing of the ciliary body from its root. This condition is unusual and results from severe blunt eye injury. A persistent, very low intraocular pressure may indicate that a cyclodialysis has taken place.

Changes in Intraocular Pressure

Reduced Intraocular Pressure. The intraocular pressure is commonly reduced after blunt trauma to the eye. The normal range of intraocular pressure is 12 to 20 mm Hg. A pressure between 5 and 10 mm Hg may follow blunt injury, presumably because of damage to the ciliary body. However, the intraocular pressure may be reduced to 0 to 2 mm Hg when the differential diagnosis includes cyclodialysis, scleral rupture, and severe diffuse injury to the ciliary body. A persistently low intraocular pressure may be associated with choroidal effusions.

Raised Intraocular Pressure. Glaucoma can be defined as a condition in which the intraocular pressure is sufficiently increased to cause damage to ocular structures, whether it be transient or permanent. Ocular hypertension, on the other hand, is a condition in which the intraocular pressure is increased without detectable damage to the eye.

The causes of increased intraocular pressure include angle recession, traumatic iritis, hyphema, blockage of the trabecular meshwork by lens debris, and ghost cell glaucoma, which is a condition that occasionally follows hemorrhage into the vitreous. Multiple ghost cells, which are erythrocytes without hemoglobin, are seen by histologic examination to occlude the trabecular meshwork.

Rarely, a marked acute increase in intraocular pressure may occur following eye injury. It causes severe pain, photophobia, and a sensation of seeing halos around lights. Clinical examination reveals a mid-dilated nonreacting pupil, circumlimbal injection (i.e., a red eye, particularly the area of the sclera close to the cornea), and hardness of the eye on palpation. Corneal edema prevents examination of the posterior segment by direct ophthalmoscopy. On the other hand, a gradual increase in intraocular pressure may cause no symptoms or a mild aching pain around the eye. In the chronic case, gradual cupping of the optic nerve head ensues, with progressive loss of the visual field (Fig. 18-13).



FIGURE 18-13 Pathologically cupped optic disc.



FIGURE 18-14 Retinal detachment. A severe blunt injury has resulted in intraretinal hematoma and retinal detachment.

RETINAL INJURY AND CHOROIDAL INJURY

Retinal Detachment

The distortional effects of concussive injury may cause retinal detachment (Fig. 18-14)[18,19] The vitreous gel is firmly adherent to the peripheral retina, and the acute coronal distention that accompanies anteroposterior blunt eye injury can result in the retina being torn. This condition causes a retinal dialysis (Fig. 18-15) or the formation of a retinal hole, because the distortional forces produce vitreous traction at the retinal periphery.

It has been suggested that almost all cases of retinal dialysis are secondary to trauma.[20] Approximately 10% of traumatic retinal detachments occur immediately, 70% within 2 years, and 20% more than 2 years after injury.[21]

Injuries to the globe may also cause changes in the vitreous (syneresis) in which the vitreous gel collapses and tearing of the peripheral retina results. The patient may complain of the sudden development of floaters—because of the condensation of the vitreous and vitreous hemorrhage—accompanied by a sensation of flashing lights in the periphery of the vision. Any patient having these symptoms should be carefully examined by a surgeon who specializes in retinal detachment.

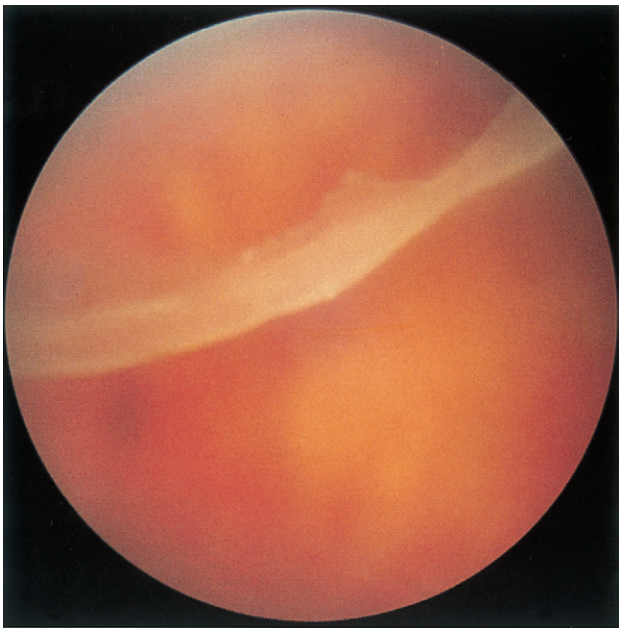


FIGURE 18-15 Retinal dialysis. This peripheral retinal photograph shows the torn edge of the retina, which is curled over.

The embryonic derivation of the eye is such that the retina is not firmly adherent to the underlying pigment epithelium. If a hole or tear develops, fluid derived from the vitreous may pass through the hole and gradually lift the retina off. As soon as the retina is detached, the part that is detached does not function.

Treatment of a retinal detachment is complex and not always successful in restoring normal vision. Therefore, it is important to prevent retinal detachment by sealing retinal holes by means of photocoagulation or cryotherapy. All patients who have sustained significant blunt ocular injury sufficient to cause any of the internal anatomic disturbances described earlier should be seen by a surgeon specializing in retinal detachment to screen for and treat peripheral retinal breaks and tears.

Exudative retinal detachment is a rare sequela to blunt eye injury. Extensive blunt injury to the retina can result in the accumulation of subretinal fluid, presumably because the pigment epithelium is damaged to such an extent that it is unable to pump the water through into the choroid. In these cases, the retina may flatten spontaneously, but the prognosis for the recovery of vision is poor.

Traumatic Retinal Edema

Traumatic retinal edema is also known as commotio retinae, or Berlin's edema (Fig. 18-16). If the injury involves the retina of the posterior pole, the patient complains of rapid loss of vision following the injury. However, if the injury is more peripheral, the patient may be asymptomatic because only the peripheral visual field is affected. Most patients report a gradual improvement in vision although in some cases, however, severe injury may result in a more prolonged recovery.[22]



FIGURE 18-16 Traumatic retinal edema.

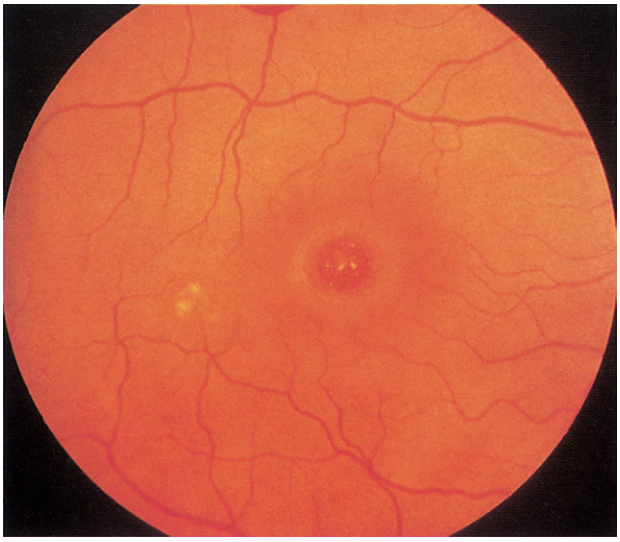


FIGURE 18-17 Macular hole. A central retinal hole has resulted following a blunt ocular injury.

Traumatic Pigmentary Retinopathy

Following a very severe blunt injury to the retina, photoreceptor disruption and damage to the pigment epithelial cells occur to such an extent that post-traumatic replication and migration of pigment epithelial cells give rise to a clinical pattern indistinguishable from that of retinitis pigmentosa. This condition is accompanied by little recovery of function.[23,24]

Retinal and Vitreous Hemorrhage

Rupture of the blood vessels may be accompanied by hemorrhage into the retina and into the vitreous, with loss of vision. Spontaneous recovery of vision accompanies resolution of the vitreous hemorrhage. Occasionally, fibrosis in the vitreous can result in a tractional retinal detachment.

Macular Hole

Macular holes (Fig. 18-17) are also observed following blunt trauma from detachment of the vitreous or as a sequela of retinal edema.[25]

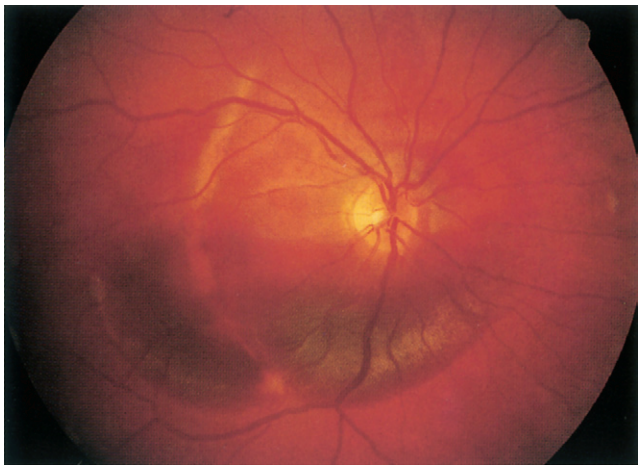


FIGURE 18-18 A traumatic choroidal tear has resulted in a subretinal hematoma, which is clearing to reveal the tear passing through the macula.

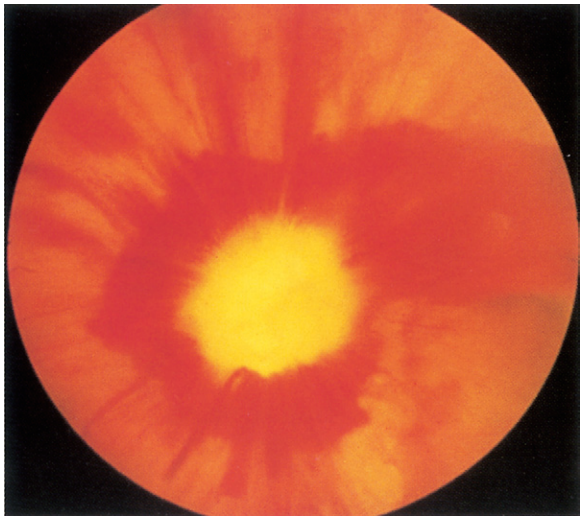


FIGURE 18-19 Optic nerve avulsion. Severe trauma has resulted in the optic nerve's being avulsed from the eye.

Choroidal Tear

Tears of the choroid (Fig. 18-18) characteristically occur circumferential to the optic disc and follow concussional injury in which the eye is severely compressed and distorted. The patient is aware of loss of central vision. This loss of vision is attributable initially to extensive subretinal hemorrhage. At this stage, the tear of the choroid cannot be seen. Over the ensuing weeks, as the hemorrhage resolves, the choroidal tear becomes apparent. A line of underlying white sclera, usually concentric with the optic disc, can be seen on ophthalmoscopy. If the tear does not pass through the fovea, the prognosis for spontaneous recovery of vision is good. However, a lesion beneath the fovea results in loss of central vision. A choroidal tear can be complicated by the development of abnormal new blood vessels beneath the retina, which may themselves bleed. In some cases, such a lesion may warrant laser photocoagulation. Therefore, all patients with a choroidal tear should be evaluated and followed up by an ophthalmologist.

Choroidal Effusion

In cases of ocular hypotonia (see earlier), the choroid may detach from the underlying sclera because of the accumulation of underlying plasma-like fluid. Treatment of the hypotonia (e.g., by closing a scleral rupture surgically) usually results in spontaneous reapposition of the choroid.

Scleral Rupture

It is important to recognize that a rupture of sclera may be silent and the only clinical sign is ocular hypotonia. If a scleral rupture is not repaired, persistent hypotonia or the ingrowth of fibrous tissue into the eye may develop. In addition, sympathetic ophthalmia, in which inflammation of the other eye occurs, may rarely complicate scleral rupture.

Surgical exploration is indicated for most cases of persistent hypotonia for which there is no alternative explanation, because repair of the scleral rupture is usually accompanied by restoration of intraocular pressure and prevents the complication of fibrous ingrowth.

Avulsion of the Optic Nerve

In very severe injuries, the optic nerve may be avulsed from the eye, with accompanying permanent loss of vision (Fig. 18-19).

PERFORATING EYE INJURIES

It is important to recognize that perforation of the globe may accompany orbital fractures, particularly in patients who have been involved in motor vehicle accidents. Any patient who has sustained multiple facial lacerations must be suspected of having a corneoscleral laceration until proven otherwise.

A detailed history of the nature of the circumstances surrounding the injury is necessary. Penetrating injuries still continue to be missed and are a major source of litigation. Perforation may be caused by a small, fast-flying missile—resulting in a retained intraocular foreign body—or by a sharp implement (Fig. 18-20).

With perforating eye injuries, the examiner ascertains the patient's visual acuity, when possible. The eye is inspected with great care, with the examiner taking every precaution to preclude pressure on the globe, which could result in the herniation of ocular contents. Therefore, the eyelids are retracted without direct pressure on the eyeball. The eye and adnexa are examined for the following:

1. Laceration or perforation of the eyelids
2. Evidence of a foreign body
3. Perforation of the globe
4. Asymmetry of the pupil, which could be a result of prolapse of the iris
5. Opacification of the ocular media caused by intraocular hemorrhage
6. A shallow anterior chamber
7. Prolapse of the iris, ciliary body, or vitreous

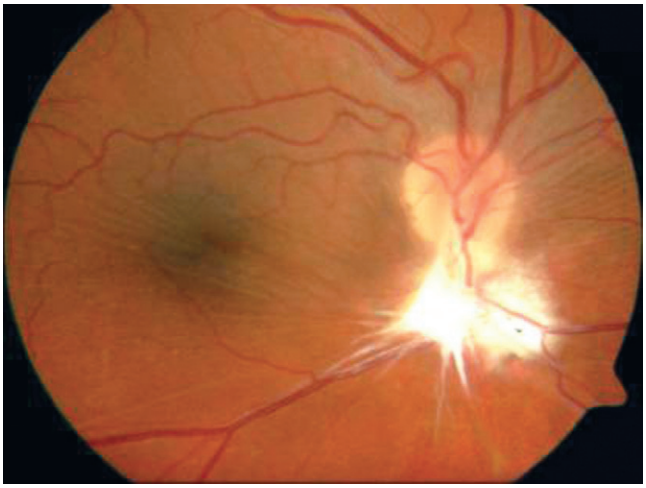


FIGURE 18-20 Impact site of an intraocular foreign body, which struck the retina just below the optic disc.

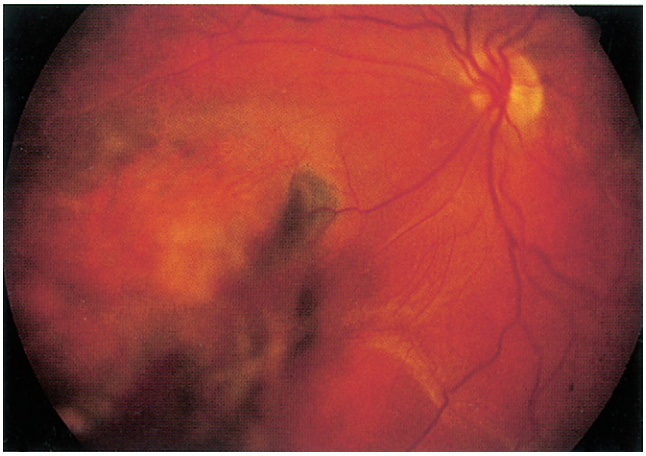


FIGURE 18-21 Chorioretinal necrosis and hematoma from an airgun pellet injury to the sclera.

Radiologic examination is also mandatory for all cases of suspected retained intraocular foreign bodies. All patients with confirmed or suspected perforating eye injuries should be transferred immediately to the care of an ophthalmologist for further evaluation.

A prognostic model, the ocular trauma score, has been developed to predict the visual outcome of patients following ocular trauma.[26] Poor initial visual acuity, globe rupture, endophthalmitis, retinal detachment, and the presence of a relative afferent papillary defect are all predictors of a poor outcome.

PERFORATING INJURIES OF THE ORBIT

Nonorganic intraorbital foreign bodies, such as airgun pellets, are usually inert, and it may be deemed safer to leave such a foreign body in the orbit than to remove it.[27] However, if an airgun pellet is immediately accessible, surgical removal is indicated. An airgun pellet may ricochet off the sclera as it enters the orbit. Therefore, full clinical examination of the peripheral retina is indicated in every case because necrosis of the choroid and retina occurs at the impact site (Fig. 18-21) and subsequent retinal detachment may ensue. This detachment can be prevented by appropriate and timely surgery.

A high index of clinical suspicion is necessary if there is a small perforating wound of the eyelid. A detailed history may provide useful information. A foreign body retained in the orbit may be radiopaque, but organic material, such as a thorn or piece of wood (Fig. 18-22), may be radiolucent and may require computed tomography (CT) for optimal imaging. Detailed scrutiny is required to exclude orbitocranial injury or globe perforation.[28]

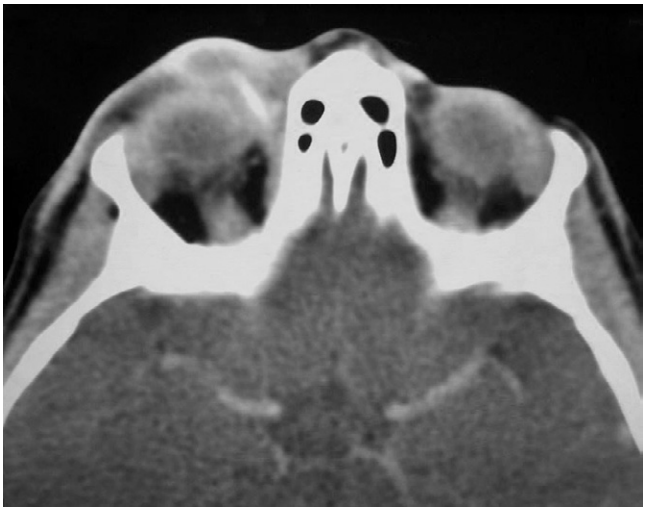


FIGURE 18-22 Splinter of wood within the nasal aspect of the right orbit.

RETROBULBAR HEMORRHAGE

Intraorbital hemorrhage is a common sequela to midfacial fracture. In most cases, this problem resolves spontaneously, with no adverse sequelae. Venous hemorrhage probably causes no problem, but arterial hemorrhage can lead to compromise of optic nerve function and central retinal artery obstruction. This complication is most likely in young individuals, in whom the orbital septum is inelastic and impermeable, thus preventing spontaneous decompression by anterior displacement of the globe and leakage of blood into subcutaneous tissue. Arterial bleeding may occur as a direct result of the injury, perhaps from rupture of the infraorbital artery or of the anterior or posterior ethmoidal arteries, or it may occur as a postoperative complication of surgical exploration of the orbit.

CLINICAL FEATURES

The patient complains of a severe aching pain accompanied by progressive loss of vision. Proptosis occurs and is accompanied by increased intraocular pressure, marked subconjunctival hemorrhage, and gross eyelid swelling. As vision is lost, the pupil becomes fixed and dilated.

MANAGEMENT

Arterial bleeding within the orbit is an ophthalmic emergency. The visual acuity, pupillary reaction, and intraocular pressure are recorded initially at 5-minute intervals. In the unconscious patient, reliance is placed on the pupillary reaction and intraocular pressure alone. In some cases, spontaneous decompression takes place and surgical intervention is not required. In this situation, progressive loss of vision, proptosis, and elevation of intraocular pressure are documented for the first 10 to 20 minutes, but the vision spontaneously recovers thereafter. No treatment may be required.

If, after 20 minutes, spontaneous recovery does not occur, the loss of vision is progressive, the intraocular pressure becomes notably increased (e.g., 30 to 40 mm Hg; normal range is 12 to 20 mm Hg), and the pupil reaction becomes sluggish, immediate therapeutic intervention is required. In the postsurgical case, the surgical wound is opened and blood may escape under pressure. The patient may be aware of an immediate spontaneous improvement in vision and the normal pupillary reaction returns. The source of bleeding is arrested.

In other cases, the source of bleeding may not be known. The patient may be treated medically with a slow IV injection of acetazolamide (adult dose up to 500 mg), which reduces intraocular pressure. When the site of the hemorrhage is not known, surgical treatment includes lateral canthotomy, in which the palpebral aperture is widened, and lateral cantholysis, in which a pair of sharp-pointed scissors is used to divide the lateral canthal ligament. This procedure allows the globe to move forward and facilitates the escape of blood from the orbit. It is usually accompanied by a temporary and sometimes permanent improvement in vision.[29] If imaging facilities are available, an immediate orbital CT scan is performed and any locule of blood is drained. When a CT scan is not available and visual function deteriorates further, division of the orbital septum superiorly and inferiorly, with insertion of drains, is carried out. Only rarely is this procedure accompanied by the release of hemorrhage; however, it does allow for prolapse of orbital fat and thereby gives rise to orbital decompression, which may be accompanied by an improvement in visual function. Orbital decompression via a transcranial approach has also been reported, although this is controversial.[30]

TRAUMATIC OPTIC NEUROPATHY

Traumatic optic neuropathy can occur following direct or indirect optic nerve injury and is a rare but potentially blinding complication of orbital trauma.[31] Optic nerve swelling within the optic canal following the injury can result in retinal ganglion cell loss. Both optic nerve decompression and high-dose steroid treatment have been advocated as a means of improving visual outcomes. However, because there is a significant spontaneous rate of visual recovery in this condition, the role of surgery and steroids remains unclear.[32-34]

DISORDERS OF OCULAR MOTILITY

Disturbances in ocular motility following trauma may arise from the following: (1) disorders of the central control of eye movement; (2) injury to cranial nerves III, IV, or VI; or (3) orbital injury with associated muscle injury or entrapment.

DISORDERS OF CENTRAL CONTROL OF EYE MOVEMENT

These disorders include horizontal and vertical gaze palsies, impairment or failure of convergence, and loss of fusion. They are more likely to occur following closed head injury.

Impairment or Failure of Convergence

This impairment may follow a closed head injury. The exact pathogenesis is uncertain but may be related to upper midbrain injury. When convergence is impaired, the patient complains of difficulty in reading and looking at near objects. Complete paralysis of convergence causes symptoms of double vision whenever the patient attempts to focus for near vision. In most cases, spontaneous recovery takes place.[17]

Loss of Fusion

Loss of fusion of the images from both eyes may result in double vision. It is an unusual but well recognized consequence of a closed head injury.[35] In this condition, the patient is unable to maintain single fused images when synoptophore, prism, or stereoscopic tests are used. Trauma is also a well-recognized cause of the breakdown of a preexisting latent squint that has received orthoptic treatment by patching in the past. The patient complains of constant diplopia. Both cerebral contusion, with or without skull fracture, and whiplash injury may cause this problem. Spontaneous recovery may or may not occur.[36] Because all tests of fusion require a subjective assessment by the patient, the results may on occasion be difficult to interpret, especially when compensation is involved.

Lateral Gaze Palsy

Lateral gaze palsy is a condition in which there is impairment of the deviation of both eyes in a horizontal direction. Lesions of the frontal cortex or the pons may cause this condition, with or without persistent conjugate deviation of the eyes. In cerebral contusion, the eyes are deviated toward the damaged side and spontaneous recovery of function usually occurs within 1 to 4 weeks.[37] Pontine lesions, on the other hand, manifest a range of other clinical signs, such as disturbance of horizontal conjugate gaze and horizontal jerk nystagmus combined with vertical nystagmus, and tend not to improve. Caloric tests and the doll's eye phenomenon provide methods of distinguishing between the two pathologic conditions. With cerebral lesions, these tests are normal, whereas with pontine dysfunction they are abnormal.

Skew Deviation

Skew deviation is a condition in which there is a vertical disparity in the positions of the eyes on eccentric gaze. This condition may result from a closed head injury. However, the sign is not of localizing value.[37]

Parinaud's Syndrome

Parinaud's syndrome results from midbrain trauma. The pupils are mid-dilated and fixed, convergence is not impaired, and there is paresis of upward gaze.[37]

Post-Traumatic Nystagmus

Post-traumatic nystagmus may occur as a result of trauma to the petrous part of the temporal bone, with associated damage to the labyrinthine system, or may be caused by brainstem trauma.[37]

CRANIAL NERVE INJURY

Traumatic damage to cranial nerves III, IV, and VI is a common sequela of a closed head injury.[38]

Cranial Nerve III Palsy

Cranial nerve III palsy typically occurs following a frontal blow to the head, such as in a motorcycle accident. The condition is usually unilateral. The signs include ptosis, fixed dilation of the pupil (caused by the unopposed action of the sympathetic supply), and abduction with slight depression of the globe (caused by the unopposed actions of the lateral rectus and superior oblique muscles). Three outcomes are possible: (1) no recovery; (2) recovery caused by regeneration; and (3) aberrant regeneration. Recovery may take up to 12 months. When aberrant regeneration occurs, paradoxical eye movements are observed. Typically, eyelid elevation occurs on attempted adduction or downward gaze. Pupil constriction and accommodation may accompany downward gaze, with pupil dilation on abduction. Cocontraction of the vertical rectus muscles may result in restriction of upward gaze.

Cranial Nerve IV Palsy

Cranial nerve IV palsy may be unilateral or bilateral. The superior oblique muscle is responsible for depression of the globe in adduction and also produces intorsion. Vertical diplopia, which may be accompanied by torsional double vision (in which the two images appear to be rotated with respect to each other), occurs. Torsion is especially problematic with bilateral cases. The presumed site of injury is the origin of the trochlear nerve from the posterior brainstem, perhaps following contrecoup injury on the tentorium cerebelli. Spontaneous recovery may occur up to 12 months after injury. Surgery for the squint is deferred to allow recovery to take place.

Cranial Nerve VI Palsy

Cranial nerve VI palsy results in failure of abduction. The nerve may be damaged at its exit from the brainstem or within the cavernous sinus. Abducens palsy may also result from a traumatic caroticocavernous sinus fistula. Surgery for the squint is again deferred to allow spontaneous recovery to take place.

Referral to an ophthalmologist is indicated for all cases of this type of cranial nerve injury. Sequential assessment is carried out to determine whether recovery is taking place before considering whether extraocular muscle surgery is indicated.

OCULAR MOTILITY DISORDERS CAUSED BY ORBITAL INJURY

Diplopia is a common sequela to orbital injury. Causes include entrapment of an extraocular muscle or its adjacent fascia, intramuscular hemorrhage and edema, orbital hematoma, disinsertion of an extraocular muscle, and injury to the trochlea, through which the superior oblique tendon passes.

Anatomy of the Orbit

The anatomy of the orbit is described in Chapter 13. The orbital walls are thin and are protected anteriorly by the thick orbital rim. The weak areas constitute the floor anterior to the inferior orbital fissure, which continues medially as the lamina papyracea of the ethmoid. The medial half of the orbital floor is weakened further by the infraorbital canal or groove. Fine connective tissue septa surround the globe and invest the muscles and other intraorbital contents. They connect with the periorbita of the orbit. In the case of an orbital fracture, entrapment of the septa may result in mobility disturbance. The extensive interconnections of these septa probably contribute to the wide range of motility disorders that may follow an orbital wall fracture, with little radiologic evidence of entrapment of soft tissue.

BLOWOUT FRACTURE

In 1943, Pfeiffer[39] was the first to review a series of cases with traumatic enophthalmos and proposed a possible mechanism of orbital floor fracture. In 1957, Smith and Regan[40] reproduced blowout fractures experimentally in cadavers. They demonstrated that posterior impaction of the globe may cause a blowout of the thin orbital floor by transmission of sudden pressure.

Similar fractures can, however, also be produced by blunt injury to the inferior orbital rim of a skull without orbital contents.[41] When a small missile (e.g., squash ball) strikes the eye, a blowout fracture is unlikely to occur, whereas a large compressive object is more likely to cause such an injury. Evidence suggests that a compressive bone injury contributes significantly to the development of a pure blowout fracture. Thus, both hydraulic forces and buckling of the inferior orbital floor probably contribute to the orbital floor blowout fracture. Converse and Smith[42] introduced the concepts of the pure blowout fracture, in which only the orbital floor is involved, and the impure lesion, in which the compression produced in an orbital rim fracture causes a similar pathologic condition.

Clinical Signs

The clinical signs of blowout fracture are as follows:

1. Enophthalmos may initially be masked by tissue swelling (Fig. 18-23).



FIGURE 18-23 Enophthalmos. The patient also has significant limitation of elevation of the left eye.



FIGURE 18-24 Limitation of elevation of the right eye.

It may result from the combined effects of prolapse of orbital fat, enlargement of the size of the orbital cavity, fat necrosis from trauma or infection, and fibrotic shortening of extraocular muscles.

2. Impairment of eye movement, which may cause diplopia, may be caused by muscle entrapment, fascial entrapment, injury to extraocular muscles, intraorbital or intramuscular hemorrhage, nerve damage, or the breakdown of a previously latent squint that has become manifest.

Double vision may not occur for a number of reasons. For example, one eye may have poor vision because of amblyopia. We have seen a number of patients who had a previous eye injury or an intrinsic eye pathologic condition (e.g., retinal detachment) that might have contributed to the failure of the patient to take evasive action. Some patients who have received successful patching for amblyopia when they were children are unaware of double vision, despite normal acuity in each eye. This situation results from alternating fixation, in which the patient chooses to use one eye or the other, but never both simultaneously. In such cases, the motility disorder does not in itself provide an indication for surgery.

Typically, there is restriction of up and down gaze (Fig. 18-24). Elevation of intraocular pressure on attempted upward gaze is suggestive of entrapment. Occasionally, retraction of the globe on upward gaze may be seen, caused by entrapment of the inferior rectus muscle. This is a subtle clinical sign in which the eye retracts back from the lower eyelid by approximately 0.5 to 1.0 mm. The eye movements are examined as described earlier. It is important to differentiate between entrapment and muscle bruising. Retraction of the globe, elevation of intraocular pressure on upward gaze, and a positive forced duction test can be helpful in this regard.

3. Pseudoptosis and deepening of the supratarsal fold accompany the enophthalmos.

   If the eyelid covers the pupil, the patient will attempt to elevate both eyelids, and lid retraction will be seen on the opposite side.

4. Orbital emphysema may be seen shortly after injury, but it absorbs spontaneously.
5. Infraorbital nerve anesthesia may occur.
6. Serious injury to the eye can occur in a significant proportion of patients, as described earlier.

Visual function is therefore assessed in all cases, and a detailed slit lamp and ophthalmoscopic examination of the globe should be carried out in every case to check for treatable abnormalities that might otherwise be missed.

Diagnosis of Blowout Fracture

CT is the investigative modality of choice and is described in more detail in Chapter 17.[43]

Medial Wall Blowout Fracture

Medial wall fractures rarely occur in isolation and are usually associated with orbital floor fractures.[44-46] It is important to recognize this condition because of the potential sequelae of enophthalmos and, more rarely, entrapment, resulting in restriction of lateral gaze, for which the results of late surgery are poor. The clinical signs of restriction of abduction and retraction of the globe on horizontal gaze should be sought in all cases. Subcutaneous emphysema and epistaxis should increase the index of suspicion for this diagnosis. Plain radiography and CT may reveal an opaque ethmoid sinus. Early surgical intervention for such fractures (within 2 weeks) has been advocated to avoid more complex repair, which may result from post-traumatic wound healing.[47]

Treatment Goals

The goals of treatment are to preserve normal binocular vision by restoring normal ocular motility and to prevent cosmetically unacceptable enophthalmos. The field of binocular single vision (BSV) is the area in which the patient has single vision. The aim with regard to ocular motility is to obtain as large a field of BSV as possible, centered on the primary positions of gaze and downward gaze. It may not be possible to abolish double vision entirely.

Whether surgical repair of the orbital floor should be carried out—and, if so, when—is a subject of controversy.

Surgery within 2 weeks is generally recommended for most patients with significant diplopia or at risk of enophthalmos, and observation for those with a good range of extraocular movements and at low risk of enophthalmos.[48,49] However, it has also been suggested recently that surgery can be delayed for up to 4 weeks, with satisfactory outcomes.[50] These guidelines do not apply to pediatric white-eyed blow-out fractures, which usually require early surgical intervention.[51]

Notable enophthalmos with an extensive orbital floor defect at the time of the initial examination will not resolve spontaneously, and surgical repair of the orbital floor can be carried out soon after injury. A motility disorder is common following surgery. Gradual recovery of eye movement subsequently takes place in most cases. Patients who remain symptomatic may require glass prisms or surgery for the squint.

Anecdotal evidence has suggested that orthoptic exercises may expand the field of BSV. There have been no clinical trials of this treatment, but it is logical to teach the patient to develop as wide a range of eye movement as possible. In theory, this therapy may have the combined benefits of diminishing the scarring (fibroblasts cannot grow across moving tissue planes), increasing the range over which the patient can fuse images, and sustaining the power of the extraocular muscles after injury.

FRACTURES OF THE ORBITAL ROOF

Up to 9% of all facial fractures involve the orbital roof.[52] In addition, penetrating injuries may perforate the orbital roof and enter brain tissue. Careful neurologic assessment and comprehensive radiologic evaluation are required if such an injury is suspected. Ophthalmic assessment is also essential; one study has reported the incidence of ocular injuries following orbital roof fracture to be 38%.[53] A range of clinical sequelae has been described, including the presence of bone fragments, which can interfere with superior rectus muscle function and cause impairment of elevation and also ptosis of the upper eyelid.

TRAUMA TO THE TROCHLEA

The superior oblique muscle passes through the trochlea close to the medial superior orbital margin. If the muscle is injured, subsequent fibrosis can result in tethering, which in turn leads to restriction of elevation of the eye in adduction. Entrapment of the superior oblique tendon in an orbital roof fracture has also been described.[54] Many patients recover spontaneously, but injection of steroids or ophthalmic surgical intervention may be required.

DISPLACEMENT OF THE GLOBE

The eye may be displaced in any dimension following orbital injuries—namely, anteroposteriorly, horizontally, or vertically.

PROPTOSIS

Proptosis (exophthalmos) is common initially because of hematoma and swelling of orbital tissue. In most cases, this condition resolves spontaneously. Subperiosteal hematoma, notably of the orbital roof,[55] may give rise to persistent proptosis associated with downward displacement of the globe. Persistent proptosis may also result from inward displacement of orbital bone fragments.



FIGURE 18-25 Hypoglobus.

ENOPHTHALMOS

Enophthalmos is a common late sequela that may initially be masked by intraorbital tissue swelling and hematoma. Expansion of the orbit, prolapse of soft tissue through a blowout fracture, necrosis of soft tissue, and fibrosis all contribute to the clinical picture. A sunken upper eyelid may be a factor in the poor cosmetic appearance. Impaired ocular motility may or may not be present.

VERTICAL DISPLACEMENT

Vertical displacement commonly accompanies an orbital fracture. In the acute phase, the eye may be displaced upward by hematoma. In the late phase, downward displacement of the globe (hypoglobus) is more common (Fig. 18-25).

HORIZONTAL DISPLACEMENT

Horizontal displacement occurs laterally when the medial ligament has been severed or the orbital bones have been laterally displaced. A displaced globe may simulate a squint. However, the corneal light reflexes are symmetrical, and neither vertical nor horizontal displacement causes double vision.

TRAUMATIC HERNIATION OF THE GLOBE INTO THE MAXILLARY SINUS

Patients with this disorder have been reported[56,57] to have good recovery of visual function following surgical restoration of the normal anatomy. Clinical evaluation is described in the ophthalmic assessment section of this chapter. Surgical treatment is indicated for patients with an unsatisfactory cosmetic appearance. Deep recession of the upper eyelid may accompany enophthalmos. In these cases, packing of the orbital floor beneath the periosteum may give good cosmetic results. In a patient in whom enophthalmos is accompanied by a blind eye, a good cosmetic result may be obtained with the use of a positive (magnifying) lens in the patient's glasses, or a



FIGURE 18-26 Laceration of upper eyelid following orbital trauma.

vertical prism can be used to improve cosmesis when a blind eye is vertically displaced.

INJURIES TO THE EYELIDS

EYELID SWELLING AND HEMATOMA

Eyelid swelling and hematoma are common following orbital injury. These injuries are usually innocuous but may indicate a major underlying ocular injury, particularly when the bruising is clearly demarcated, resembling the eye of a panda. Spontaneous resolution takes place during the ensuing weeks.

EYELID LACERATIONS

Eyelid lacerations (Fig. 18-26) should be repaired within 72 hours. It is better to wait for good operating room facilities and to carry out formal surgery than to perform an unsatisfactory repair in the middle of the night. The eye and orbit are examined for evidence of injury and a high index of clinical suspicion is maintained for retained foreign bodies. Radiologic examination, including CT, is performed if retained organic material is suspected. Appropriate antitetanus treatment is provided and the wound is immediately débrided. Corneal exposure is an indication for immediate treatment.

Surgical Repair

Surgical repair requires accurate repositioning in layers, not just superficial skin closure. When the eyelid margin has been severed, the integrity of the margin is restored with a nonabsorbable suture passing through the gray line. This step is carried out first and allows subsequent accurate suture placement. The tarsal plate is repaired with an absorbable suture, with care being taken that this suture passes through only a partial thickness of the plate to prevent corneal damage. Tissue should not be discarded because even unattached tissue may heal. Local skin grafts and flaps may be required if there has been gross loss of tissue.

Repair of the upper eyelid requires great care to prevent ptosis. The wound is explored to ensure the integrity of the levator and superior rectus muscles and of the globe. The levator complex is carefully isolated, and any lacerations are sutured. The integrity of the orbital septum is restored. The eyelid is then repaired. Failure to recognize damage to the levator may result in unsightly ptosis, which is difficult to repair by secondary surgery.[58]

EYELID AVULSION

Avulsion of the eyelids requires immediate treatment to protect the cornea. An antibiotic eye ointment or 1.5% methylcellulose is instilled regularly to prevent corneal ulceration. Prompt surgical management is required for these challenging cases and, ideally, this should be performed by an oculoplastic surgeon using lamellar repair principles.[59]

PROGRESSIVE SHORTENING OF THE LOWER EYELID

This condition, caused by fibrosis, may occasionally complicate any approach to the inferior orbital floor.

NASOLACRIMAL INJURIES

Damage to the lacrimal drainage system following trauma often results in epiphora. Although spontaneous recovery can occur, surgical management such as dacryocystorhinostomy may be indicated after several months

CANALICULAR LACERATIONS

Lacerations of the canaliculi are repaired at the time of primary surgery, ideally with the use of an operating microscope. Detailed descriptions of this surgery are available in standard ophthalmic surgical texts. However, the results of surgery are often disappointing, and secondary intervention may be necessary.

INDIRECT OPHTHALMIC CONSEQUENCES OF INJURY

TRAUMATIC RETINAL ANGIOPATHY (PURTSCHER'S RETINOPATHY)

Multiple discrete, superficial infarcts of the retina manifesting as loss of central vision, accompanied by the development of multiple cotton wool spots adjacent to the optic nerve head, may appear as a result of severe skull fracture, chest compression, and long bone fractures.[60] The patient complains of loss of vision in one or both eyes 24 to 48 hours after injury. No specific treatment exists, but the prognosis is good, with spontaneous gradual recovery of vision taking place during the ensuing 2 to 3 months in most cases.

CAROTICOCAVERNOUS SINUS FISTULA AND ARTERIOVENOUS ANASTOMOSIS

Fistula formation between the arterial and venous systems may occur following craniomaxillofacial trauma.[61,62] The condition is usually painful. Clinical signs include gradually increasing ophthalmoplegia, chemosis, proptosis, and dilated blood vessels (of both the conjunctiva and the eyelids), which may be accompanied by pulsating exophthalmos (Fig. 18-27). In some cases, an intracranial bruit may be heard. The intraocular pressure is usually

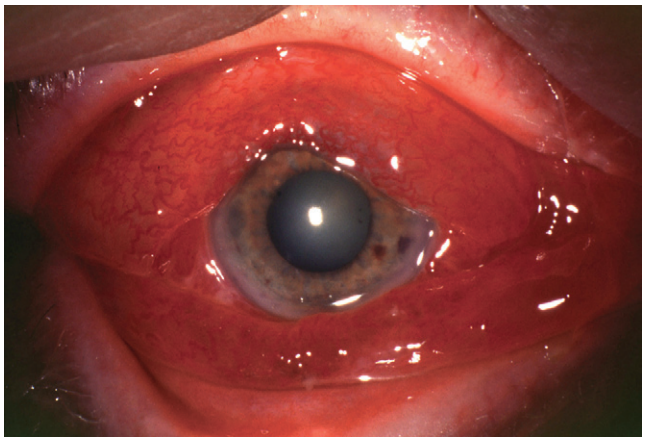


FIGURE 18-27 Caroticocavernous sinus fistula showing marked chemosis and dilated blood vessels.

increased and loss of vision may ensue if the condition becomes more severe. Neurosurgical referral is indicated.

FACIAL PALSY

All patients who have facial palsy as a result of injury should be assessed for the presence of Bell's palsy. The patient is asked to close the eyes and the degree of cover of the cornea is determined. The cornea is examined in detail for the development of ulceration. Tarsorrhaphy is indicated for patients who have corneal ulceration; ointment is instilled at night into the eyes of patients in which corneal coverage is incomplete. Alternatively, botulinum toxin can be injected into the levator palpebrae superioris.[63] This results in complete ptosis and protects the cornea for 4 to 6 weeks. Such treatment is indicated when recovery of facial nerve function is anticipated.

PAPILLEDEMA

Examination of the optic nerve heads for the development of papilledema is essential for all patients suspected of having raised intracranial pressure resulting, for example, from a chronic subdural hematoma.

RELATIONSHIP BETWEEN MAXILLOFACIAL AND EYE INJURIES

Trauma resulting in midfacial fractures is often associated with ocular injuries of varying severity. All patients who have sustained such trauma should have their visual acuities checked at the time of examination. If an eye injury is suspected, the patient should be managed appropriately and referred promptly for a thorough ophthalmic assessment.

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CHAPTER

19

Evaluation and Management of Frontal Sinus Injuries

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OUTLINE

Development, Anatomy and Function
Pathophysiology
History of Treatment
Overview of Clinical Decision Making
Goals of Treatment
Epidemiology
Fracture Classification
Diagnosis of Frontal Sinus Injuries
History and Physical Examination
Imaging Studies
Management of Frontal Sinus Injuries
Antibiotic Therapy
Operative Treatment
Postoperative Management
Complications in Frontal Sinus Injuries
Perioperative Complications
Early Complications
Late Complications

The frontal sinus is an important component of the complex skeletal junction between the cranium and face. Injury to this area can occur in isolation or, more commonly, may be associated with other injuries to the brain, skull, orbits, globes, midface, and overlying soft tissues. As such, treatment of traumatic injuries to this region may require transcranial, subcranial or conventional maxillofacial surgical techniques and commonly requires multidisciplinary evaluation and treatment. The goal of contemporary management is the restoration of form and function, with minimization of morbidity and mortality.

The last several decades have been witness to considerable changes in the diagnosis and management of facial trauma in general, and frontal sinus injuries in particular. The greatest impact has been made by the ready availability of computed tomography (CT) imaging, which now allows for accurate visualization and diagnosis of injuries. The development and popularization of craniofacial techniques by Paul Tessier and others have combined with the refinement of surgical equipment and microplate fixation to allow for the predictable execution of complex surgical reconstruction. More recent advances in endoscopic instrumentation and skull base techniques are continuing to transform management of frontal sinus injury by increasing emphasis on the restoration of a functional sinus in addition to a safe sinus.

The purpose of this chapter is to provide an overview of the contemporary diagnosis and management of injuries of the skull involving the frontal sinus.

DEVELOPMENT, ANATOMY, AND FUNCTION

The embryology and development of the frontonasal region provide an important context for a thorough understanding of the anatomy and subsequent patterns of injury in the frontal region. This must be combined with an appreciation of normal physiologic function to help direct treatment.

The craniofacial skeleton shows evidence of intramembranous ossification of the nasal and frontal bones at 50 to 60 days' gestation.[1,2] By 4 months in utero, the earliest signs of frontal sinus development are present as the middle meatus begins to expand superiorly, creating an early frontal recess.[3] At this intranasal location, it is common for numerous furrows to form that will eventually evaginate into the frontal and ethmoid bones.[4] One or more of these frontal recess furrows most often pneumatize the frontal bone to become the developing frontal sinus. Alternatively, ethmoid infundibular cells may provide the source of pneumatization that leads to sinus formation; more rarely, the frontal recess may propagate directly into the frontal bone.[5] Additional accessory furrows of the frontal recess are important for the development of agger nasi cells and anterior ethmoid air cells. Some of these remain modest in size and contained within the ethmoid while others expand considerably without this confinement.

The developmental complexity and heterogeneity of the frontal sinus and anterior ethmoid air cells has the secondary effect of creating a highly variable nasofrontal drainage system. In as many as 80% to 85% of the population, no discernible duct is present and an ostium-type outflow tract serves to drain the sinus.[6,7] Alternatively, relative compression of the proximal part of the frontal sinus by developing ethmoid cells may create a true ductal drainage system. When a duct is present, it may vary from 1 to 20 mm in length and 1 to 6 mm in width.[8] Drainage into the nose will typically occur below the middle turbinate near the middle meatus, bearing a



FIGURE 19-1 A midsagittal view characterizes the normal skeletal anatomy of the intranasal region. The blue marker demonstrates the frontonasal outflow tract draining medial to the uncinate process below the middle turbinate (removed).

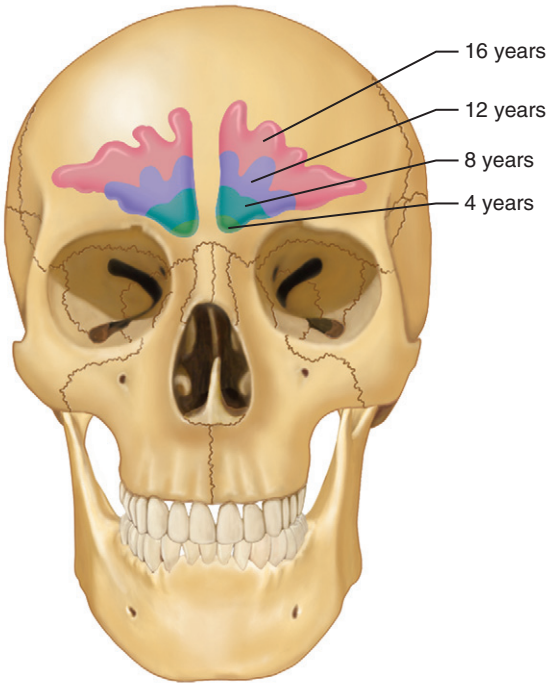


FIGURE 19-2 Frontal sinus development correlated with age—green, 4 years; blue, 8 years; purple, 12 years; pink, 16 years.

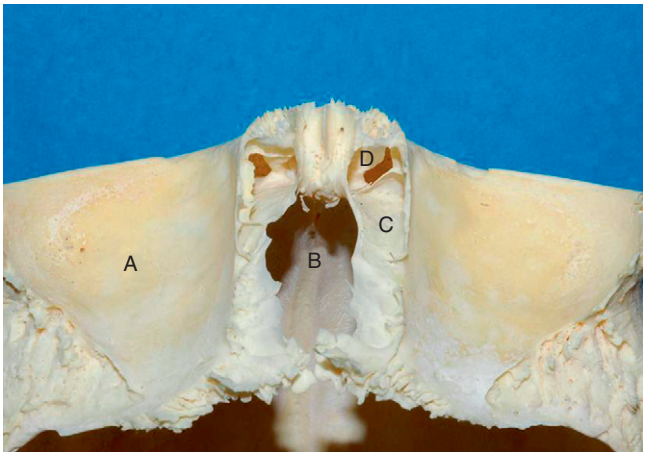


FIGURE 19-3 Inferior view of the frontal bone demonstrates **A,** Orbital roof. **B,** Cribriform plate (removed). **C,** Fovea ethmoidalis. **D,** Frontonasal ostium.

variable relationship to the ethmoid infundibulum based on the sinus origin (Fig. 19-1). Usually, drainage is directly into the frontal recess and medial to the uncinate process, but less commonly this may occur above or into the ethmoid infundibulum lateral to the uncinate, with rare occurrences above the ethmoid bulla.[9,10]

Over time, the frontal sinus pneumatizes to a highly variable degree. Underdevelopment of the frontal sinus is relatively common, with an estimated 4% of the population showing no development and another 5% demonstrating minimal pneumatization.[11] Approximately 10% of the population has only unilateral development of the frontal sinus.[3] When present, the sinus is not typically identifiable radiographically before the age of 6 years and does not show appreciable development until puberty.[12] Size consistent with that of the adult is normally attained by the mid to late teen years (Fig. 19-2).

The fully developed frontal sinus is expected to be highly variable in size and shape. When present, asymmetry between right and left is the norm and bony septations may be present. The sinuses may be present in duplicate or triplicate bilaterally. In general, the sinus is rather diminutive, with a capacity of between 5 and 16 mL.[4,12] The average height of a developed frontal sinus is 32 mm and the average width is 26 mm.[13,14]

The proximity of the frontal sinus to important anatomic structures accentuates the importance of thoughtful management of these injuries. The anterior portion of the sinus borders the dense frontal bone superiorly and supraorbital rim inferiorly; these are the thickest and strongest boundaries. In between these buttresses, the anterior wall becomes more attenuated and somewhat weaker across the face of the sinus. The thinner cortical posterior wall of the sinus is adjacent to the intracranial cavity and skull base. In the midline, the posterior wall of the frontal bone is adjacent to the crista galli, which rises superiorly from the cribriform plate; laterally, the cribriform plate transitions to the fovea ethmoidalis of the anterior skull base (Fig. 19-3). On the deep surface, the dura is dense and tightly adherent and thins along the midline of the anterior skull base due to multiple neurovascular foramina.[15] Notably, after complete pneumatization of the sinuses, the final position of the cribriform plate is variable and often becomes situated in a position well inferior to the neighboring ethmoid air cells.

The posteromedial portion of the sinus floor contains an ostium that is the beginning of the nasofrontal outflow tract (Fig. 19-4). The lateral aspect of the floor is

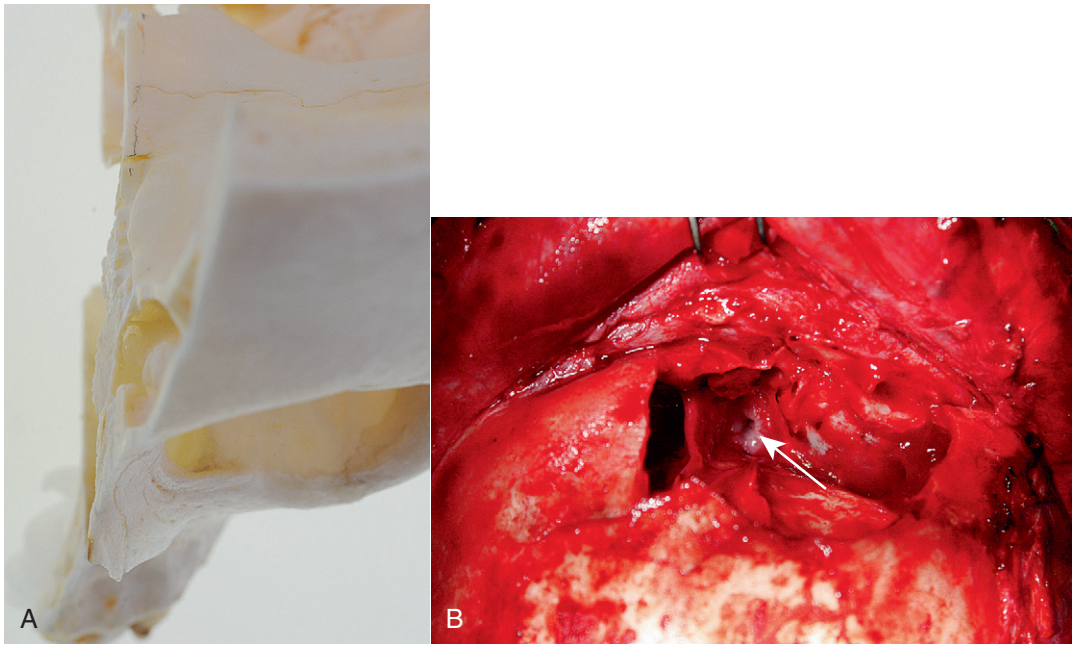


FIGURE 19-4 The nasofrontal outflow tract drains in the posteromedial floor of the frontal sinus. **A,** This is demonstrated on a skull model sectioned in the midsagittal plane and in a clinical example viewed from above **(B). B,** Prerepair.

confluent with the medial portion of the orbital roof. Anteriorly and inferiorly is the nasoethmoidal complex, which has a significant likelihood of concurrent injury. This may manifest with telecanthus, loss of nasal bridge support, and medial orbital wall fractures. The relevance of these injuries in the context of frontal sinus trauma is their relationship to nasofrontal outflow tract obstruction as well as the position of the cribriform plate and potential for intracranial violation.

The arterial supply to the frontal sinus region is from the anterior ethmoid artery and the branches of the sphenopalatine artery via the middle meatus.[2] The supraorbital, anterior superficial temporal, anterior cerebral, and middle meningeal arteries all supply the frontal bone.[16] Venous drainage is transosseous into the subcutaneous, orbital, and intracranial veins. The diploic veins of Breschet are associated with foramina in the frontal bone significant for deeply invaginating sinus mucosa, which can be a source of mucocele formation if incompletely removed during obliteration or cranialization procedures. Also, they allow for direct vascular connections between the mucosal and dural venous systems.[3,12,17,18] This pattern of venous drainage has clinical implications for the development and management of intracranial abscess associated with frontal sinusitis and infection. The frontal sinus is innervated by nerves that follow arterioles including the lateral posterior superior nasal branches of V2, as well as the anterior ethmoid nerve branching from V1.[19]

Normal sinus function is maintained by pseudostratified, columnar, ciliated respiratory epithelium covered by a layer of mucin. The cilia beat at the rate of about 10 to 15/second, with mucociliary clearance from the frontal sinus into the middle meatus of the nose.[10,20] Interestingly, the frontal sinus is the only sinus in which there is some retrograde flow of mucus, with movement superiorly along the medial wall, laterally along the superior aspect, and then back to the ostium along the inferior aspects of the sinus. The rate is slowest at the roof of the sinus and the fastest around the nasofrontal duct.[13,21]

PATHOPHYSIOLOGY

The location of the frontal sinus allows it to serve a protective role to the brain, in addition to providing normal sinus function. Although the presence of a frontal sinus has been confirmed to increase the likelihood of frontal bone fracture, it acts as a shock-absorbing barrier to the intracranial contents.[22] The frontal bone at the anterior sinus wall is able to withstand direct trauma up to 990 kg of force.[23,24] Conceptually, this force can be attained by an unrestrained passenger suffering a motor vehicle collision at 30 mph.[25] As the frontal bone's protective capacity is exceeded, concomitant intra- and extracranial injuries should be anticipated. The surrounding bones of the anterior skull base, orbits, and nasoethmoid complex are significantly weaker, leading to their potential for associated fracture. The posterior wall and anterior cranial base are particularly concerning because of the potential for dural tears, leading to communication between the intracranial compartment and sinus environment, with the possibility of meningitis or brain abscess.

Normal frontal sinus function relies on adequate drainage, which may become impaired with nasofrontal outflow tract damage or obstruction. In simplistic terms, obstruction can lead to mucus buildup and development

of an expanding mucocele within the frontal sinus. This can be compounded by bacterial infection and, together, lead to erosion of surrounding bone and development of osteomyelitis or sequestration. Drainage will follow the path of least resistance and may present externally as a draining sinus tract. A more subtle presentation may be seen with intracranial or orbital extension. As such, assessment of the patency and subsequent management of the nasofrontal outflow tract are critical decision making elements in the treatment of frontal sinus injuries. Chronic inflammation in the adjacent thin-walled spaces of the ethmoid and frontal recess air cells can lead to edematous changes in the nasofrontal outflow tract, contributing to frontal sinus drainage problems. Patients with a history of recurrent anterior ethmoid sinusitis are at a higher risk of developing recurrent frontal sinus infections after trauma to the frontal sinus.

HISTORY OF TREATMENT

Complications from frontal sinus trauma and sinusitis in the age before antibiotics could be devastatingly morbid and frequently lethal. The history of surgical interventions was largely based on the treatment of chronic suppuration and sinusitis. The earliest reported operation on the frontal sinus was by Viega in 1586 for treatment of a frontal osteoma.[26] As early as 1870, an attempt at surgically treating a frontal pyocele by external and intranasal approaches was published by Wells.[5,27] Soon after, reports of treatment for infection of the frontal sinus by puncturing the frontal recess to improve drainage or pack the sinus followed, but were not embraced secondary to the risk of inadvertent entry into the intracranial space.

Jacobs credits Ogston in 1884 with the first substantial description of an external approach to the frontal sinus to establish drainage, outflow tract dilation, and intranasal drain placement.[5,28] Reidel, in 1898, reported on a radical exenteration of the sinus walls and supraorbital bar, followed by removal of all sinus mucosa and leaving the overlying soft tissues to retract into the defect against the posterior wall.[26] The hope was to reduce the potential for mucocele, mucopyocele, meningitis, or brain abscess, but at the cost of profound disfigurement.

In 1903, Killian presented a more conservative variation of Reidel's procedure that maintained the supraorbital bar limiting the ablation to the anterior table and frontal sinus mucosa while using postoperative stents in the outflow tract.[5,29] Despite the limited improvement in cosmesis, morbidity and mortality remained high secondary to persistent disease, most likely from postoperative closure of the outflow tract.[30] This too was quickly discarded as a viable treatment option.[31]

By 1921, Lynch had introduced a promising procedure whereby a medial periorbital incision was used for access to the floor of the frontal sinus and anterior ethmoid air cells, with the aim of extirpating the mucosal lining and opening the nasofrontal drainage system, again using stents to maintain the sinus drainage.[32] Despite the appeal of a more limited operation, results with this technique were also disappointing because complete removal of the mucosal lining was difficult and restenosis of the nasofrontal outflow tract was common.[33]

Fractures of the frontal sinus in the first half of the twentieth century were generally approached nonoperatively and most healed uneventfully.[34] Even so, compound fractures were expected to have high mortality, approaching 50% if untreated when associated with pneumocephalus.[35] Treatment was still required in select circumstances and Jacobs has stated thatt because of "continued reports of at least a 30% failure rate with the Lynch frontoethmoidectomy, as well as the associated difficulty in visualizing the distal portions of the frontal sinus, the osteoplastic school began to gain acceptance."[5]

Beginning in 1934, Bergara and Itoiz developed the osteoplastic flap approach, in which the anterior frontal sinus wall was removed but maintained on an inferior pedicle of pericranium to allow replacement of the bone flap.[36,37] This procedure allowed improved access to the damaged sinus so that removal of the mucosal lining could be accomplished more thoroughly, all while preserving the anterior table for a more natural-appearing reconstruction. The use of adipose tissue for obliteration of the frontal sinus can be traced back to Marx in 1910,[26] but Bergara and Itoiz[36,37] and later Montgomery scientifically examined and popularized the technique, publishing a series of papers emphasizing the importance of nasofrontal drainage function and frontal sinus obliteration with autologous fat to prevent inflammatory complications.[38,39]

Once considered the gold standard for chronic frontal sinusitis, the osteoplastic flap has since fallen out of favor because of its associated complications—cerebrospinal fluid (CSF) leak, frontal bossing, supraorbital neuralgia, chronic sepsis, mucocele formation, and osteitis.[40-42] With the development of modern craniofacial techniques, it became clear that wide subperiosteal undermining, most often through a coronal scalp incision along with primary bone graft reconstruction, were viable strategies in the craniomaxillofacial region. Subsequently, this led to further improvements in surgical access and more aggressive attempts at primary reconstruction using these principles. Impressive results, with low morbidity after mucosal exenteration and fat graft obliteration of sinuses with injured nasofrontal ducts according to these methods, have since been reported.[6,43]

Removal of the posterior wall of the frontal sinus in cases of severe comminution or pneumocephalus had been described earlier,[34,35] but cranialization of the frontal sinus in its contemporary form can be attributed to Donald and Bernstein.[44] Encouraged by the work of Nadell and Kline performing primary reconstruction of depressed skull fractures in penetrating cranial injury with low rates of infection,[45] they described a procedure in which the posterior frontal sinus wall was removed, all sinus mucosa was eliminated, and the intracranial contents were isolated from the nose by obstructing the nasofrontal outflow tract. Importantly, they advanced the idea that one could reconstruct the anterior table, even in the setting of contaminated injury after disinfection. This provided improved cosmetic and functional results in the group of patients with complex frontobasilar injury. Since that time, treatment has largely focused on variations of the techniques of cranialization, obliteration,

anterior table reconstruction, and management of the nasofrontal outflow tract.

OVERVIEW OF CLINICAL DECISION MAKING

GOALS OF TREATMENT

The goals of management of frontal sinus injuries are structural protection of the intracranial contents, isolation of the intracranial compartment from the aerodigestive tract, provision of a functional sinus, cosmetic restoration of natural frontal contour with the use of minimally visible incisions, minimization of perioperative morbidity, and prevention of postoperative infectious and inflammatory complications.

A review of the literature provides little definite direction regarding the management of patients with frontal sinus injuries. Most studies are retrospective reports and series, with biased samples and limited follow-up. Treatment outcomes are poorly documented, as are the effects of operative versus nonoperative management. Importantly, Chuang and Dodson have suggested that we do not know how often bad outcomes occur in the untreated injury and we have no information to support that treatment decreases the risk for an adverse outcome.[46] Treatment recommendations are therefore based on theoretical outcome predictions according to knowledge of frontal sinus pathophysiology and local intracranial vulnerability, which likely encourages overtreatment with the hope of minimizing complications. Further work will hopefully bring additional clarity to the proper use of current techniques.

EPIDEMIOLOGY

Since frontal sinus development is not appreciable in early childhood and gradually enlarges throughout the teen years, fractures are rare in children and uncommon in adolescents.[47,48] For those treating adults, frontal sinus trauma remains infrequent being reported in 5% to 15% of all facial fractures from major trauma centers and presumably represents a much smaller proportion at community centers.[13,34,49-57] In the contemporary civilian literature, frontal sinus injuries are frequently associated with blunt trauma secondary to motor vehicle accidents (MVAs), causing demographics to skew toward major trauma statistics.[53,55,58] Although young and old are reported, the average age is consistently in the fourth decade and the overwhelming majority of injured are male (Fig. 19-5).[50,51,59,60] Associated maxillofacial fractures may be present in over 70% of patients.[58,61] Regarding the sinus itself, most fractures involve the anterior table alone (43% to 61%) or the anterior and posterior tables (19% to 51%). Frontal recess or frontonasal floor involvement with possible disruption of the drainage system occurs less commonly (2.5% to 21%). Isolated injuries of the posterior table are rare (0.6% to 6%).[57,62,63]

FRACTURE CLASSIFICATION

There is no universally accepted classification of frontal sinus injuries and specific numbering systems are largely academic. From a clinical perspective, distinction is commonly made between lateral and central injuries.[52,64] Injuries of the lateral fronto-orbital skeleton frequently involve the supraorbital margin and lateral orbital wall, as well as occasionally involving the temporal or parietal skull. Care is focused on assuring dural and cranial vault integrity and the reconstruction of orbital volume, with limited regard for sinus involvement.

Central injuries are directly related to the frontal sinus and further distinction should be made based on the integrity of the naso-orbital-ethmoid (NOE) complex[12] and skull base.[65] Classification here is divided into specific functional and anatomic units, including the anterior table, posterior table and skull base, dural competency (cerebrospinal fluid leakage), NOE complex and nasofrontal outflow tract integrity. With the ready availability of high-resolution CT imaging, diagnosis and distinction of these injuries are realistic.

Numerous treatment algorithms can be found throughout the literature.[15,58,61,66] Similar to classic facial trauma treatment principles, an inside-out approach is advocated that emphasizes the importance of frontobasilar integrity and seeks functional sinus preservation, when possible.

DIAGNOSIS OF FRONTAL SINUS INJURIES

HISTORY AND PHYSICAL EXAMINATION

Frontal sinus fractures are commonly caused by high-velocity events and are frequently associated with other systemic or craniomaxillofacial injuries.[51] Over 50% of patients diagnosed with frontal sinus fractures will suffer neurologic injury, with as many as 25% demonstrating associated ophthalmologic injury.[12] Subdural or epidural hematomas requiring acute neurosurgical intervention may be found in as many as 8% to 10%.[67] Rodriguez et al have found cervical spine injuries in 7% to 14% of his series, depending on the severity of the injury.[58] Prior to arrival at the hospital, patients are regularly intubated, sedated, and medically paralyzed. Initial evaluation often occurs in the emergency department or intensive care unit and the most of the history is often taken from first responder records and other medical staff, depending on the condition of the patient and availability of family members or historians. Even in the setting of an apparently isolated frontal sinus injury, one needs to maintain a high degree of suspicion for other craniomaxillofacial and anatomically remote injuries. A comprehensive and systematic trauma examination is always indicated and reviewed elsewhere in this text.

The comprehensive craniomaxillofacial examination should begin with a focused neurologic evaluation, with particular attention to the Glasgow Coma Scale (GCS) score and cranial nerve function. This is closely tied to a thorough ophthalmologic examination, looking specifically for visual acuity, pupillary reactivity and symmetry, signs of foreign bodies and globe injury, and/or increased intraocular pressure. Careful inspection and palpation of all the hard and soft tissues with specific detailed attention to several key areas also follows. All the bony buttresses must be firmly palpated through the often edematous overlying soft tissues to identify any

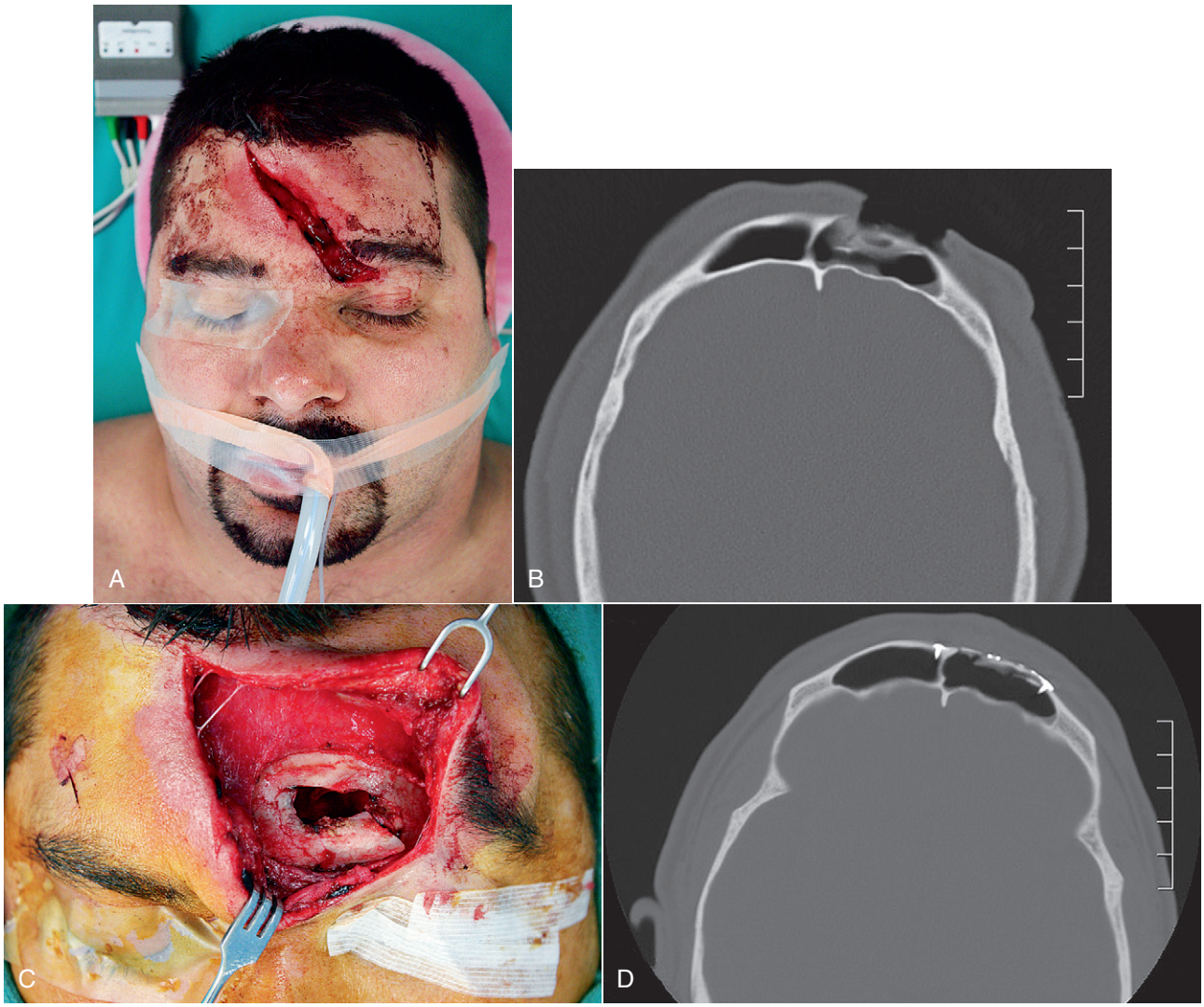


FIGURE 19-5 **A,** Frontal sinus trauma is most commonly seen in men. **B,** Anterior table involvement demonstrated on CT scan. **C,** Use of existing laceration for surgical access. **D,** CT scan demonstrating postoperative result.

irregularities, mobility, and crepitus. If aware, patients may complain of pain or paresthesia in the forehead and scalp. Identification of medial canthal displacement is critical and is indicative of concomitant NOE injury. If present, dressings should be temporarily removed to allow for visualization of the soft tissues. Significant periorbital edema and ecchymosis are common, making the examination challenging. When identified, lacerations should be carefully explored to identify any underlying open bony injuries. Specific attention should be given to periorbital lacerations and the status of the lacrimal system. Further review of specific soft tissue injuries and their management is also presented elsewhere in this text.

The nasal cavity should be gently cleaned and inspected for septal or mucosal injuries. The presence of rhinorrhea or otorrhea is not uncommon in the setting of complex midfacial trauma and may be related to CSF leakage. Identification of a CSF leak is important, although at times difficult, because this finding specifically alters patient and injury management. In a series by Bell and Chen, rhinorrhea was present in 26% of frontobasilar fractures, but CSF leakage in only 4.6%.[51] If cooperative, patients may be asked to lean forward and perform the Valsalva maneuver to increase CSF flow or may be asked about posterior nasal drainage. Use of the ring or halo test can be suggestive of the presence of CSF. Suspected fluid is placed on gauze or filter paper and an inner ring of blood may be seen, surrounded by an outer ring of clear CSF. Assessing the chemical composition of the fluid may also provide additional diagnostic support because CSF has a higher glucose concentration and lower chloride concentration compared with serum. Importantly, the presence of the beta-2 transferrin isoenzyme is most diagnostic.[68] However, collected fluid requires electrophoresis for separation of the proteins and the Western blot technique to detect the beta-2 transferrin isoenzyme, which may take up to 4 days to process in the laboratory, resulting in a delay in diagnosis.[69]

IMAGING STUDIES

Although the clinical examination must never be devalued, few areas of craniofacial trauma are aided by modern CT technology as much as frontobasilar injury. Multislice helical CT scanners allow for rapid, high-resolution data acquisition; 1- to 1.5-mm axial slices provide detailed images, which are extremely useful for assessing injuries to the frontal sinus and midface.[13,70-72] Axial, coronal, and sagittal images are requisite for comprehensive data collection (Fig. 19-6). Axial images clearly reveal the location, severity, and degree of comminution of anterior and posterior table fractures, as well as demonstrate the presence or degree of nasoethmoid impaction and status of the lacrimal system. Coronal images best demonstrate fractures of the frontal sinus floor, orbital roof, and walls. Sagittal views are important for assessing the nasofrontal outflow tract. Three-dimensional image reconstructions provide excellent pictorial representations of fracture patterns and the position of major segments. However, they are generated by volume-rendering algorithms that average and eliminate surface detail and should not be used exclusively for fracture diagnosis.

MANAGEMENT OF FRONTAL SINUS INJURIES

ANTIBIOTIC THERAPY

Antibiotic use in the setting of facial trauma continues to be controversial because the literature is devoid of well-planned and executed randomized prospective trials. A practical approach to antibiotic therapy is to treat fractures prophylactically with external contamination or gross disruption until the bones have been reconstructed and stabilized and the soft tissues cleansed and repaired.[73] For open frontal sinus injuries, extended-spectrum penicillins, cephalosporins, or clindamycin are appropriate. Otherwise, perioperative antibiotic therapy should follow the surgical recommendations and guidelines for clean contaminated surgery in the head and neck, including incisions through oral and pharyngeal mucosa, as indicated.[74]

OPERATIVE TREATMENT

Access to the Fronto-Orbital Region

Surgical access that minimizes facial scarring is a basic tenet of craniomaxillofacial surgery. When considering elective approaches to the frontal sinus region, the preferred approach to meet this objective is the coronal flap. If performed correctly, it rapidly and bloodlessly provides complete access to the frontal bone and upper face and also provides the most desirable aesthetic results, except in bald or balding patients (Fig. 19-7). An added benefit of coronal access is the ready availability of autogenous bone and soft tissue that may be needed during repair and reconstruction of frontal injuries, with minimal additional dissection.

The coronal approach has been meticulously described by Ruiz et al[75]; it consists of an incision behind the top of the vertex of the head with postauricular extensions bilaterally. The use of a postauricular coronal incision eliminates visible scars and decreases risk to the frontal branch of the facial nerve in reoperated patients. If significant exposure of the root of the zygoma is anticipated, the inferior extension is moved to the preauricular position. The inferior extent of the incision dictates the degree of inferior and caudal exposure of the facial skeleton, not the anteroposterior position. Use of a sinusoidal incision avoids a straight line scar and is thought to be useful in camouflaging the scar after healing, especially when the hair becomes wet. Occasionally, secondary operations are performed in cases where a previous coronal scalp flap has been used. Although it may be tempting to place the incision in a different location, it is preferable to incise through the original scar due to the potential effect of the previous scar on flap perfusion and wound healing.

There are a number of other approaches to the frontal and nasoethmoid areas (Fig. 19-8). Not infrequently, existing lacerations can be used and extended if necessary to provide limited access to the anterior table of the frontal sinus (Fig. 19-9) The gull wing or eyeglass incision can result in rather unattractive scars, which tend to be highly visible because of their presence in the prominence on the brow and the resultant reflection of the light. The open sky approach is equally problematic, leaving an H-shaped scar over the brows and nasion region. The use of these direct approaches should be limited to patients who are balding and have heavy rhytids or scars (Fig. 19-10).

Management of Posterior Table Fractures

Management of posterior table fractures is determined by the degree of frontobasilar involvement, condition of the nasofrontal outflow tracts, and amount of concomitant brain injury. There is little debate that grossly disrupted anterior and posterior table injuries with violation of the dura and frontal lobe contamination require operative treatment. Displaced basilar skull fractures with CSF leakage also belong in the operative category. More nuanced views lead to controversy as posterior table injuries become more moderate. Using an animal model to quantify risk stratification, Donald has recommended surgical treatment for patients with posterior table defects of greater than 25%.[18] Other clinicians have advocated that with a patent nasofrontal outflow tract and no concurrent dural tears or brain injury, any amount of posterior table displacement is inconsequential and may be managed with observation.[58] Determination of what constitutes a complex posterior table fracture necessitating surgical intervention for treatment and protection of the intracranial contents requires clinical judgment and collaboration with a neurosurgical colleague in equivocal situations.

Cranialization is the preferred technique to ablate the sinus while maintaining forehead contour in the setting of complex frontobasilar injury.[15,44,76,77] Neurosurgical involvement is often necessary for craniotomy access, management of intracranial injury, and dural repair (Fig. 19-11).

Access to the entire frontal sinus is generally limited through the anterior table fracture. Complete exposure

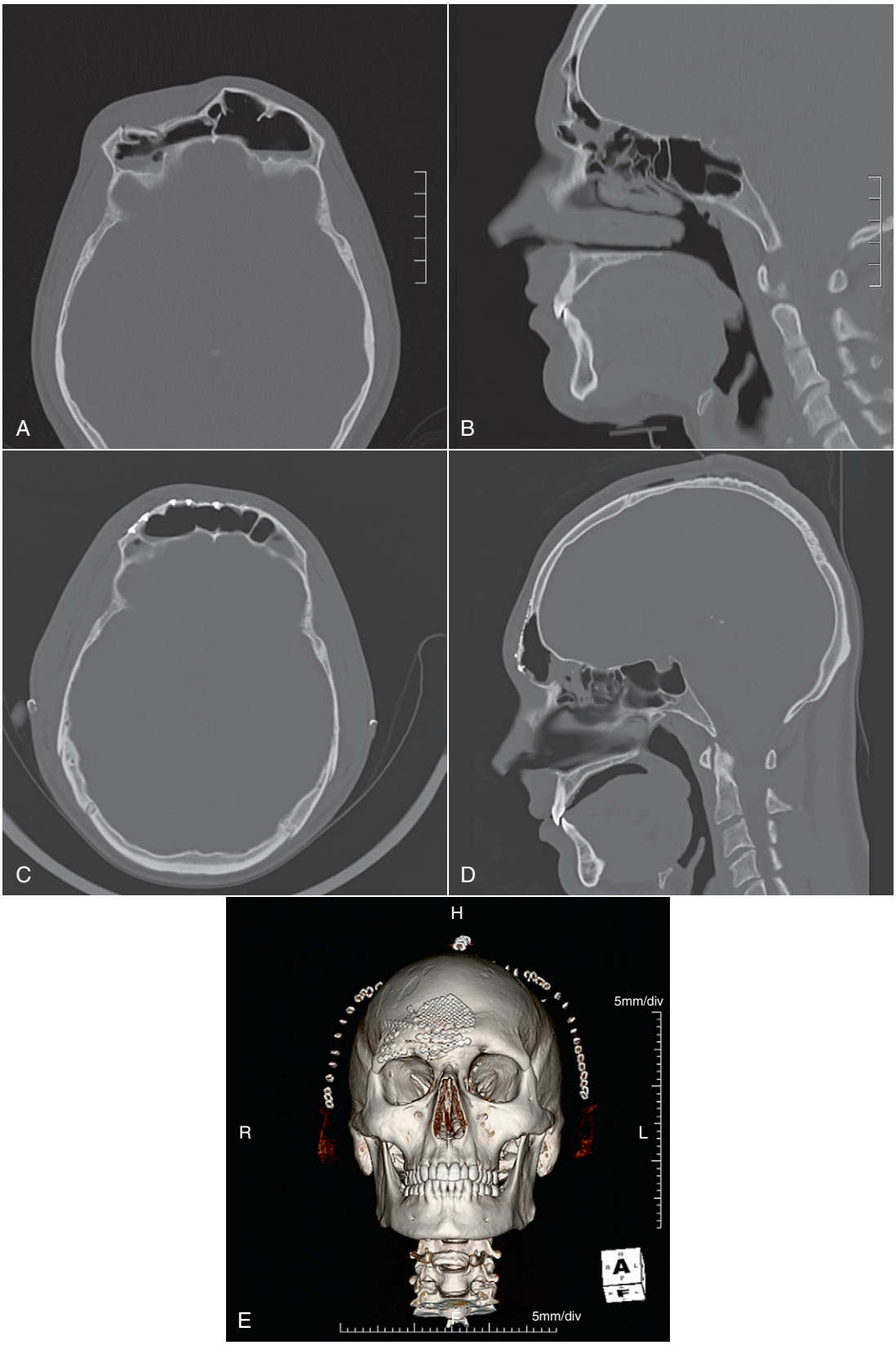


FIGURE 19-6 Multiple plane, fine cut CT scans are required for diagnosis and management of frontal sinus injuries. **A, B,** Preoperative axial and sagittal CT scans demonstrating anterior table fracture with a patient NFOT. **C-E,** Postoperative axial, sagittal and three-dimensional reconstructions of repair.



FIGURE 19-7 **A,** Immediate postoperative result after coronal incision for frontal sinus fracture repair. Note the posterior position of the incision. **B,** Result after regrowth of hair. Note the well-hidden scar.

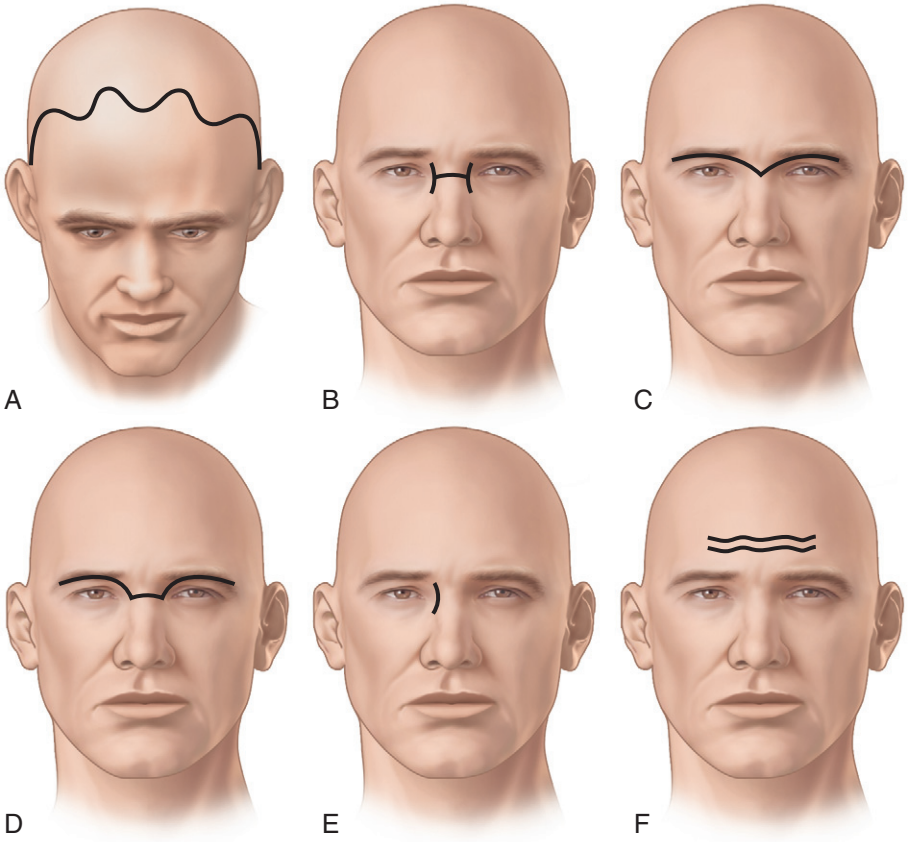


FIGURE 19-8 Surgical access and incisions to the frontal sinus region. **A,** Coronal, coronally from the temporal region to the temporal region. **B,** Open sky, two incisions in the medial orbital region connecting over the nasal bridge. **C,** Two gull wing curved incisions at or inferior to the brow, ending at the nasion. **D,** Butterfly, a combination of gull wing and open sky incisions. **E,** Sewall, a single-side medial orbital incision. **F,** Incisions through the existing lacerations.



FIGURE 19-9 **A-D,** Lacerations associated with a frontal sinus injury that has allowed for direct reconstruction.

of the posterior table is achieved by means of a bifrontal craniotomy. The entire posterior table is then removed, including loose or necrotic fragments. Meticulous elimination of the frontal sinus mucosa is carried out and often requires the use of a rotary drill and hand curettes. Necrotic brain tissue is excised and dual repair is completed, commonly with pericranial patches or allogenic dural replacement materials and tissue glue. Any bony irregularities of the inner frontal bone are smoothed with hand or rotary instruments (Fig. 19-12).

Any remaining sinus mucosa can then be everted into the nose and the nasofrontal outflow tract is sealed. Various materials can be used for obstruction, including abdominal fat, autogenous bone, temporalis muscle, and fascia. A layered approach from the nose to the frontal sinus is recommended; studies have reported that local soft tissue flaps decrease the risk of infection.[15,77-79] The pericranial flap is readily available for additional reinforcement and demonstrates continued vascularity following harvest.[80] It can be harvested centrally or laterally when concurrent forehead lacerations are present (Fig. 19-13). Finally, the anterior table fragments are cleaned and, if necessary, disinfected before being replaced and stabilized with biodegradable or titanium microplate fixation.

Management of Cerebrospinal Fluid Leaks

Traumatic CSF rhinorrhea can be classified as accidental or surgical. Management has consisted of conservative therapeutic measures, lumbar diversion, intracranial or

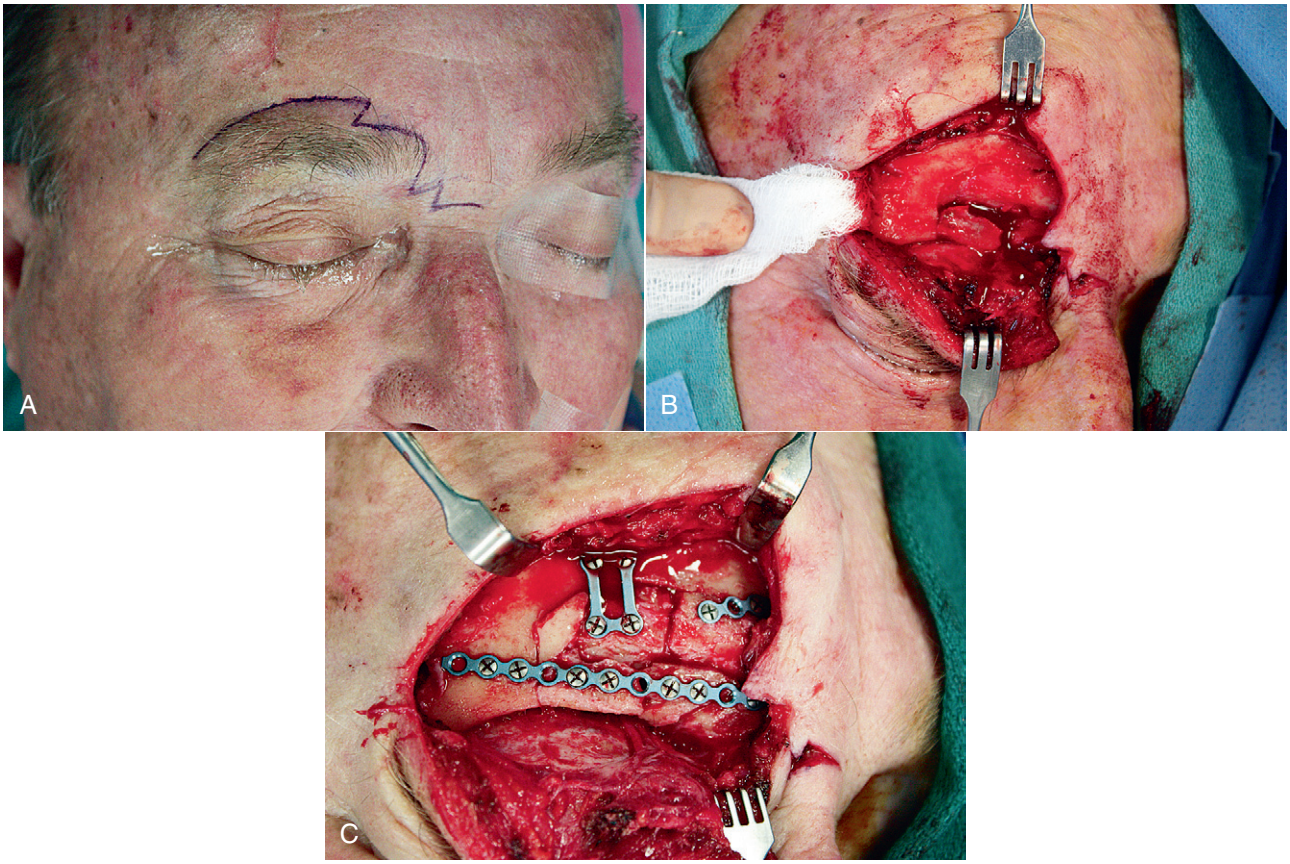


FIGURE 19-10 Preexisting scars and skin creases may be used for access to treat frontal sinus injuries in carefully selected patients.

extracranial operative repair, and transnasal endoscopic repairs. Again, we find little guidance from the literature about the appropriateness of prophylactic antimicrobial therapy, the usefulness of lumbar drainage, or the risks and benefits of early or late surgical intervention.

The most common surgical causes of ACSF leak are from rhinologic and neurosurgical procedures,[81] and any CSF leak identified intraoperatively should be repaired. Accidental CSF rhinorrhea can present immediately or delayed, with 95% manifesting within 3 months of injury.[82]

With a clinical suspicion of CSF rhinorrhea, the most important next diagnostic steps are confirmation of a leak and localization of the defect. Beta-2 transferrin is the gold standard for leak confirmation.[68] Fine-cut coronal and axial CT scans should be the primary imaging modality; magnetic resonance imaging (MRI) may be a useful complement. Adjunctive localizing tests include radionuclide cisternography with the use of intrathecal tracers and intranasal pledgets, CT cisternography with IV contrast, and MRI cisternography.[81]

Intrathecal fluorescein is the most commonly used CSF tracer, usually in the preoperative period. A retrospective review of 420 applications of intrathecal fluorescein has described two grand mal seizures, which were attributable to simultaneous intrathecal application of contrast.[83]

A meta-analysis of CSF rhinorrhea treated surgically has reported greater than 90% and 97% success rates with the first and second attempts, respectively.[84] Factors to consider in the approach to closure include the cause of the leak, associated elevated intracranial pressure, encephalocele formation, and site and type of defect.[82]

Endoscopic repair of CSF rhinorrhea has become the gold standard, particularly for the ethmoid roof and sphenoid sinuses.[85,86] Schlosser and Bolger have published extensively on CSF rhinorrhea and described surgical considerations in detail.[82] Proper exposure is first obtained and several millimeters of mucosa around the bony defect are removed. Any encephalocele should not be pushed intracranially, but should instead be reduced with bipolar cautery followed by meticulous hemostasis.

Frontal sinus skull base defects may also be treated with an external approach. This includes a frontal osseous flap and frontal sinus obliteration. The mucosa in the frontal sinus is removed and the bone is drilled with a diamond burr to prevent mucocele formation in the postoperative period, as described earlier.[82]

Leaks can be repaired with an overlay, underlay, or combined approach (Fig. 19-14). Defects larger than 5 mm may be better managed with a combined approach. Materials and sealants used include temporalis fascia, fascia lata, muscle, turbinate mucosa, fat, cartilage, bone, human acellular dermis, xenogenic collagen dural

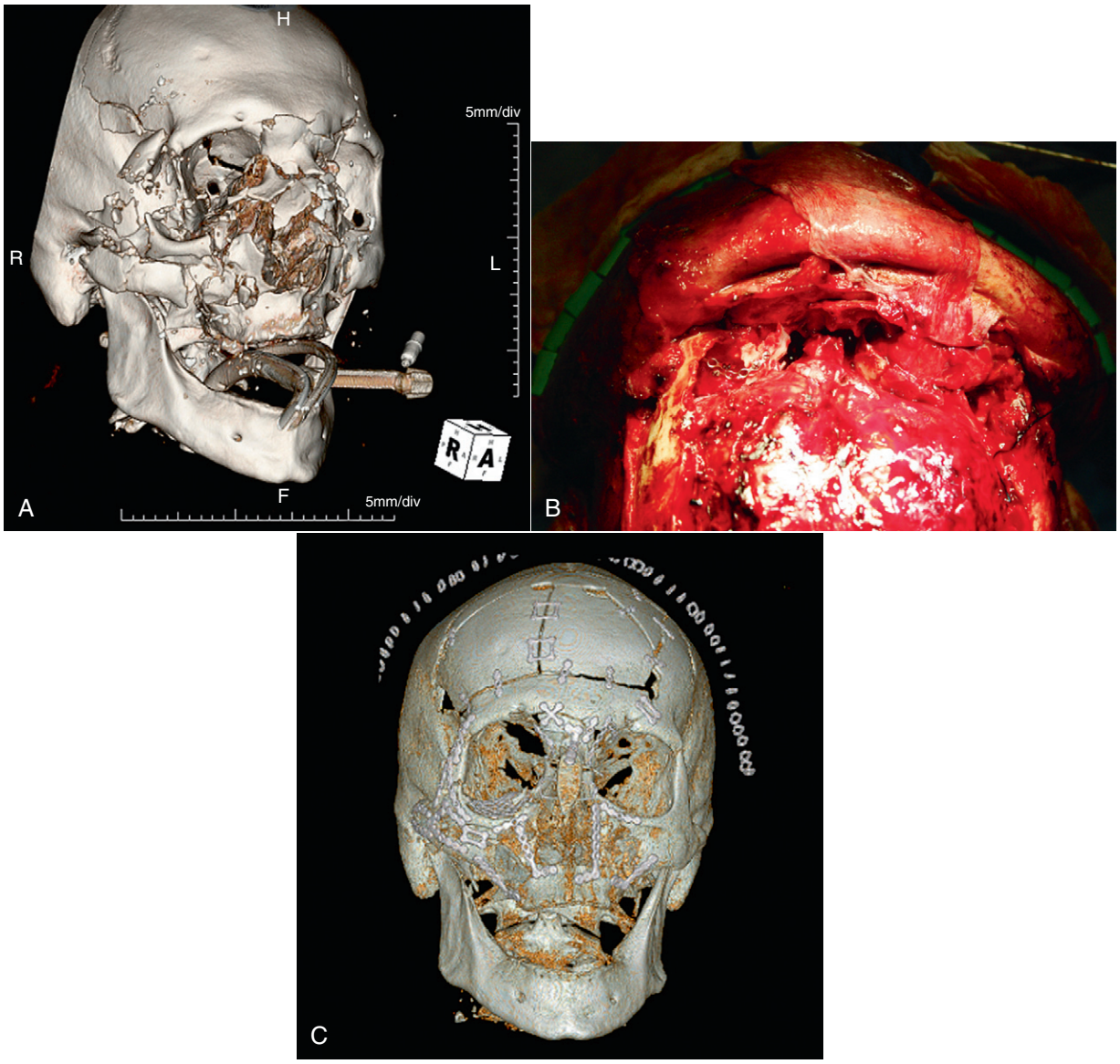


FIGURE 19-11 Complex frontobasilar injury requires combined neurosurgical and craniofacial treatment, often with bifrontal craniotomy for increased exposure. **A,** Preoperative CT scan showing complex injury. **B,** Intraoperative access with bifrontal craniotomy. **C,** Postoperative CT scan demonstrating reconstruction.

substitutes, and fibrin glue.[81] There is no apparent difference with graft choice and much of it depends on surgeon preference.[84] Fibrin glue has been shown to increase graft adherence and strength of repair in animal models.[87] Degradable packing such as Gelfoam and Surgicel are commonly used. Foley catheters can act as a bolster to help promote graft adherence. Postoperatively, patients are restricted to bed rest and the lumbar drain is managed, if present. Antibiotics are not routinely administered, but acetazolamide may have a role to decrease CSF production. Continuous positive airway pressure is usually restricted.[82]

There is support for the idea that post-traumatic CSF leakage in minimally displaced fractures is largely self-limiting and can be initially treated with conservative measures such as bed rest, head elevation, stool softeners, and avoidance of coughing, sneezing, or straining for 1 to 2 weeks. The use of prophylactic antibiotics is controversial, because these have not been shown to decrease the incidence of meningitis. A lumbar drain may be inserted, depending on the extent of the leak, likelihood of spontaneous closure, and coexistence of elevated intracranial pressure.[81] Over two thirds of these leaks have been shown to close with conservative management.[88,89]

Management of Frontal Recess Fractures Involving the Nasofrontal Outflow Tract

Fractures of the nasoethmoid complex, supraorbital rim, and frontal sinus floor may obstruct the nasofrontal outflow tract. This is used as a surrogate for frontal sinus function because it is hypothesized that damage to the

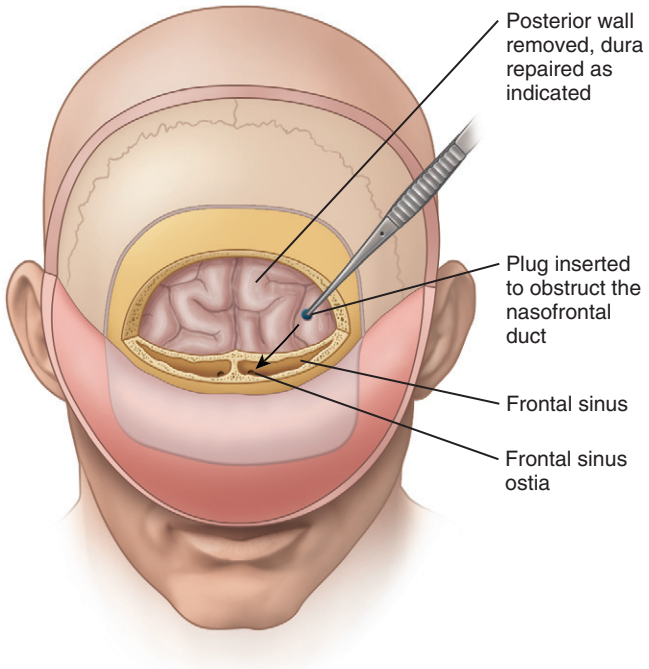


FIGURE 19-12 After careful débridement of the frontal sinus region, with meticulous removal of all remaining sinus mucosa, the nasofrontal ducts are obliterated.



FIGURE 19-13 Anteriorly based pericranial flap that can be used for repair and obliteration.

nasofrontal drainage system and associated obstruction of frontal sinus drainage increases the risk of developing mucoceles and postoperative inflammatory complications. As such, assessing the status of the nasofrontal outflow tract is a key issue in managing frontal sinus injuries.

The status of the frontal sinus floor and nasofrontal outflow tract is first assessed by CT. Specific indicators of injury include gross outflow tract comminution, frontal sinus floor fracture, and anterior table medial wall fracture. However, evaluating the patency of the drainage system by imaging can be challenging because the

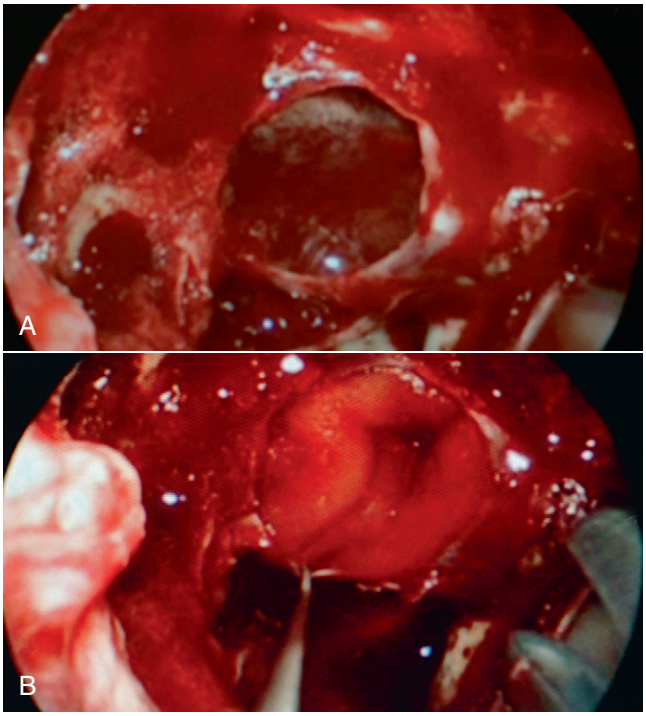


FIGURE 19-14 **A,** Intraoperative endoscopic view of an iatrogenic CSF leak during a skull base resection of a pituitary adenoma. This was repaired with a combined approach. **B,** A dural substitute (Durasis) was used for the underlay. This was then covered with a pedicled nasoseptal flap.

functional significance of each radiographic finding is difficult to determine; intraoperative exploration and evaluation are at times necessary.

From a superior approach, saline, various dyes, or contrast may be used intraoperatively to assess patency of the drainage system. One approach is to place a wide-bore angiocatheter into the opening of the nasofrontal outflow tract and introduce propofol into the duct. The fluid should be visualized from beneath the middle turbinate or as a collection in the posterior pharynx to confirm nasofrontal outflow tract patency. A nasal speculum or endoscope can assist in the identification and diagnosis.

If the nasofrontal outflow tract is not patent, conventional management recommendations support open surgical treatment with obliteration.[58] The goals of obliteration are the elimination of all sinus mucosa, obstruction of the drainage system, and obliteration of the remaining dead space. Access to the frontal sinus should be performed with the aim of complete exposure and access while facilitating natural-appearing postoperative reconstruction (Fig. 19-15).

Just as with cranialization techniques, complete removal of the sinus mucosa is carried out by curettage and the use of a surgical burr with irrigation. Unilateral injury of a nasofrontal drainage tract is not predictably treated by removing the intersinus septum, which relies on the contralateral drainage system; therefore, treatment of the sinus as a single entity is recommended.[15] Any remaining nasofrontal duct mucosa can then be inverted into the nose; obstruction of the nasofrontal

outflow tract should be carried out in a layered approach with autogenous tissue.

Sinus obliteration has been performed with a host of materials reported in the literature, including hydroxyapatite, bone cements, bone, cartilage, muscle, absorbable gelatin sponges, spontaneous osteoneogenesis, temporalis fascia, acrylic or methyl methacrylate, and fat.[15,26,90-99] Fat is the most common choice, can be easily harvested from the abdomen, and has been shown to persist in a cat model,[100] although this method is not without its detractors. Rodriguez et al have demonstrated a higher rate of complications when obliterating with fat (22%) compared with obliteration without fat (5%).[58] Others have suggested that no one technique has been demonstrated to be superior to another.[101]

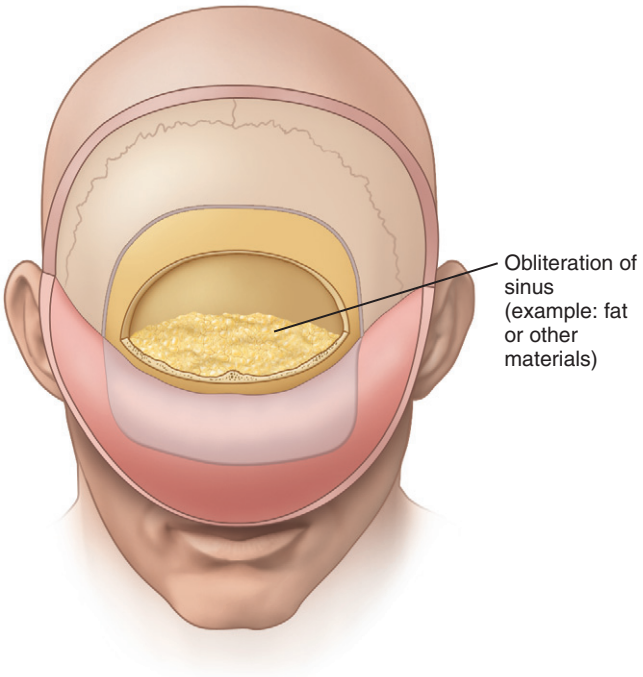


FIGURE 19-15 The appropriate harvested or grafted material is packed into the prepared sinus cavity.

The anterior table is then reconstructed with the previously removed frontal bone, titanium mesh, or free cranial bone grafts, all secured with biodegradable or titanium microplate fixation (Fig. 19-16).

Endoscopic Approach

More contemporary views allow for the growing suitability of endoscopic approaches for injuries in which there is evidence of nasofrontal outflow tract obstruction and/or anterior table fractures.[102,103] This allows the surgeon to avoid obliteration of the sinus and is established as an appropriate option in the nontraumatic setting.[104]Although gaining interest, it is yet to be commonly used in frontal sinus trauma. A review of 158 frontal sinus fractures has described just one patient who was treated endoscopically.[57] However, with the increase in skills, techniques, and technology, endoscopic management of frontal sinus disease, including traumatic, is gaining relevance.[103]

The endoscope can be inserted through a small trephination to diagnose frontonasal duct damage and can aid in placing a stent. Stenting of the frontal recess in the acute setting, however, has been associated with a high incidence of restenosis.[6,17,55]

The modified Lothrop technique, or Draf III procedure, involves removal of the floor of the frontal sinuses, including the anterosuperior septum and intersinus septum.[105] This approach also avoids injury to the supratrochlear and supraorbital nerves. Endoscopic clinical follow-up may be easier than interpreting MRI images in patients who have undergone frontal sinus obliteration. This approach has been successful in the primary management of selected frontal sinus fractures.[103]

Postoperatively, patients who have had Draf III procedures are followed closely for frequent débridement. Saline irrigation is usually recommended. With these measures, up to 90% of patients will have a patent frontal recess, with normal mucociliary drainage[104] (Fig. 19-17).

Management of Anterior Table Fractures

Simple greenstick or minimally displaced anterior table fractures do not require surgical management. Historically, displacement of less than the thickness of the anterior table has been considered inconsequential.

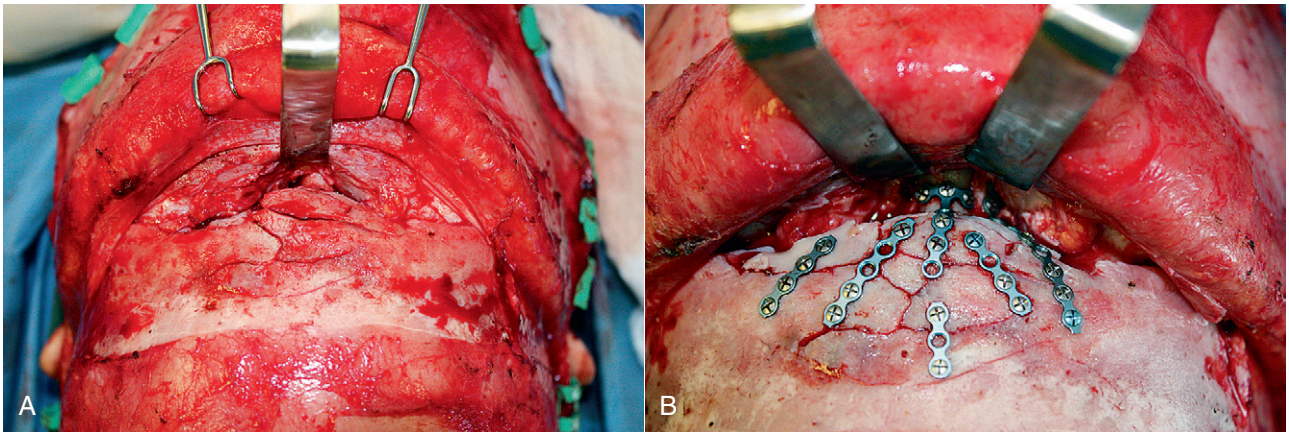


FIGURE 19-16 **A,** Comminuted frontal sinus and NOE fracture. **B,** Bone reconstruction completed with titanium fixation in place.

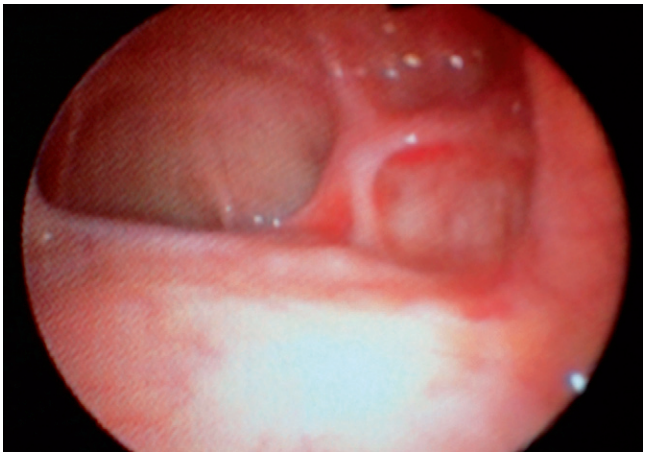


FIGURE 19-17 Endoscopic view taken in clinic of a right frontal sinus 3 months after a Draf procedure.

Frequently, clinical reevaluation after 7 to 10 days may be required to determine the amount of visible deformity after soft tissue swelling has resolved. Displaced and multifragmented fractures may require operative intervention to address contour deformities and perhaps decrease the risk of mucocele formation. Case reports have indicated that minimally displaced and noncomminuted fractures may be addressed via an endoscopic or insufflation approach.[101,103,105,106] These injuries are largely cosmetic in nature and may be amenable to delayed recontouring or camouflage with autogenous grafting or alloplastic implants. Antibiotics are not indicated unless the fractures are open or contaminated. Sinus decongestants are a useful adjunct during the observational period.

POSTOPERATIVE MANAGEMENT

Initial postoperative management efforts include serial clinical examinations with an emphasis on wounds and neurologic and ophthalmologic findings. An initial postoperative CT scan is indicated for evaluation of the postoperative bony reconstruction and then as a baseline for future examinations. Other postoperative medical management considerations include addressing pain, inflammatory concerns, and sinus patency issues. Postoperative pain management should address the patient's chief complaints. The use of perioperative prophylactic antibiotics in the head and neck for 24 hours or less is currently recommended. In case of foreign body or gross contamination, antibiotic treatment may be continued for a period of 7 to 14 days.[107]

Decongestants should also be considered following frontal sinus surgery. Common decongestants include the systemic α-adrenergic agonists (e.g., pseudoephedrine) as well as topical (e.g., oxymetazoline spray); these medications can effectively decrease the volume of nasal mucosa by acting on receptors in venous vessels.

Complications can occur years after injury, making follow-up a critical component of the successful management of frontal sinus injuries; however, this may be difficult to achieve in practice. One current recommended strategy is weekly follow-up for the first month, then every 3 months for the first year, then annually for the first 5 years, and then once every subsequent 5 years.[13] For all frontal sinus injuries, immediate postoperative radiographic evaluation and serial postoperative CT may be indicated for an extended period of time because mucoceles have been diagnosed decades after surgery by various investigators.[7]

COMPLICATIONS IN FRONTAL SINUS INJURIES

Advancement of the quality of care requires a review and discussion of complications. Furthermore, identification of injuries and procedures that are at a higher risk of complications should direct surgical techniques as well as follow-up. However, the frequency of complications associated with the observation or surgical management of frontal sinus injuries is challenging to ascertain. The literature on this topic is limited for several reasons and is therefore devoid of clear-cut answers. Sample sizes are small due to the rarity of frontal sinus injuries. Care is further diluted across many hospitals and treatment institutions, and therefore limits research initiatives. Diagnostic methods continue to improve and treatment methods continue to evolve across multiple specialties that overlap to treat craniomaxillofacial injuries. Local, consistent long-term follow-up of patients treated for facial injuries is unlikely ever to be a reality.

Chuang and Dodson have meticulously reviewed the literature with the use of a Medline search from 1980 to 2003, looking for significant inflammatory complications, including persistent frontal sinus pain or headache, meningitis, brain abscess, mucopyocele, osteomyelitis, and persistent CSF leak or fistulae after treatment. In their study, 25 articles were included but were largely noted to be of poor quality (level 4 case series).[46] They concluded that despite significant limitations in the articles reviewed, in the setting of operative management of frontal sinus injuries, the mean incidence of serious inflammatory complications is approximately 9% (range, 0% to 50%; 95% confidence interval [CI], 0% to 21%). An additional review of elective craniofacial procedures that induced iatrogenic injury to the frontal sinus was also included to estimate complication rates associated with observation of injuries for comparison.

Two significant retrospective reviews have been published more recently, both of which have included treatment algorithms. In the first, Bell and Chen reviewed their contemporary treatment of 144 patients over the past 10 years.[51] In their study, 28 patients were excluded due to insufficient records. The focus of the treatment algorithm was the maintenance of a functional sinus when possible. Of the 116 patients that were included, 66 were observed due to minimally displaced fractures, while 50 underwent surgical treatment. There were no complications recorded in the observational group, while 16% of the surgical group sustained a complication within the 90-week follow-up period.

In the second report, Rodriguez et al included a more extended 26-year review of the treatment of 857 patients with frontal sinus fractures.[58] Using the status of

the nasofrontal outflow tract as the main diagnostic parameter, 353 patients were observed (no clear nasofrontal outflow tract obstruction), with a 3.1% complication rate, and 504 patients underwent surgical treatment (suggested nasofrontal outflow tract obstruction), with a 10.4% complication rate. By their protocol, patients who demonstrated nasofrontal outflow tract obstruction required cranialization or obliteration with autogenous tissue other than fat. Both procedures carried an approximate 10% complication rate.

A prospective trial to define treatment approaches and complication rates further is neither feasible nor ethical. With this understanding in mind, the above-noted studies can be used to provide a framework for the discussion of complications associated with the treatment of frontal sinus injuries.

Additional delineation of specific types of complications and their chronologic occurrence is also clinically relevant. Characterization may be made based on the severity and subsequent requirement for reoperation or hospitalization, as well as the timing of appearance.

PERIOPERATIVE COMPLICATIONS

Perioperative complications may be related to the repair of the maxillofacial injury or other systemic traumatic injuries, if present. Specific injuries may include intracranial bleeding, seizures, neurologic damage, cerebrospinal fluid leakage, ophthalmologic injury, and hematoma. In general, with meticulous surgical technique, these are rare. Postoperative infection, meningitis, and brain abscess may occur more frequently, depending on the level of comminution, extent of exploration and surgical repair, and presence of wound or sinus contamination.

EARLY COMPLICATIONS

Early complications are defined as those occurring within the first 6 months after injury.[15] They are categorized as inflammatory, infectious, or aesthetic.

Sinonasal and Intracranial Communication

Pneumocephalus can result from craniofacial trauma or as a postoperative complication. With frontal sinus trauma, this tends to resolve and is usually of little consequence.[108]

Tension pneumocephalus results from a combination of air pressure from the aerodigestive tract and a ball valve phenomenon (Fig. 19-18). CT imaging is the best initial investigation. If mild in severity and without symptoms, it may initially be managed conservatively. Surgical treatment to close the communication usually involves the same approach as the initial surgery. This can include endoscopic, external, or combined approaches.[108,109]

Sinusitis

Frontal sinusitis may occur early in the postoperative period. Patients may present with increased edema, erythema, and generalized tenderness in the forehead and periorbital regions. As noted, decongestants are prophylactically recommended for all patients undergoing sinus surgery to prevent nose blowing. They also help minimize mucosal inflammation and facilitate normal drainage and function, with the goal of preventing postoperative sinusitis. Radiographic imaging is a necessity and may demonstrate mucosal thickening. Initial medical management of the delayed presentation of sinusitis consists of decongestants, with consideration given to the use of antibiotics, depending on the severity of associated clinical and radiographic findings such as fevers and sinus opacification.

Meningitis

Invasive infection may progress from bacterial sinusitis. Intracranial abscess and meningitis may be the most serious early infectious complication associated with frontal sinus trauma. The reported incidence of meningitis appears to be between 0.9% and 6%.[17,51,61,67] The cause may be associated with incomplete obstruction of the nasofrontal outflow tract, allowing aerodigestive communication with the intracranial space or obliterated frontal sinus cavity. Also, inadequate frontonasal sinus drainage, remnants of sinus mucosa, dural tears, and cranial defects may all create conditions favorable to the development of an infectious complication. Direct extension of contaminated materials or tissues through



FIGURE 19-18 This patient previously had a craniotomy and frontal sinus cranialization. Postoperatively, he developed tension pneumocephalus with sneezing. **A,** Before bag-mask ventilation. **B,** Subcutaneous emphysema with bag-mask ventilation.

small fractures of the diploe can also result in meningitis, empyema, cavernous sinus thrombosis, and intracranial abscess. Patients may present with sinusitis symptoms as well as severe headaches, seizures, focal neurologic findings, change in vision, and fevers. Again, follow-up imaging is a necessity and management may require medical therapy (e.g., antibiotics, decongestants), with or without surgical exploration and treatment (e.g., abscess drainage, repair of dural or cranial defects, separation of the aerodigestive tract and central nervous system, obliteration of dead space, cranialization).

Cosmetic Irregularities

Contour irregularities and cosmetic deformity may be noticeable as the edema from the initial injury subsides. This can be seen in observed and reconstructed patients. In addition, loss of frontal bone through infection or resorption may compromise the original repair. This can usually be managed after a period of soft tissue maturation by the addition of free bone grafts, alloplastic implants (e.g., porous high-density polyethylene [Medpor], polyetheretherketone [PEEK]), or onlay bone cement. Palpable or visible hardware can be removed, if necessary, at times through small stab incisions. The availability of low-profile rigid fixation and the use of primary bone grafts have decreased the likelihood of frontal bone contour deformities in the contemporary management of these injuries.

LATE COMPLICATIONS

Late complications of frontal sinus fractures occur 6 months or more after the initial injury[15] and have a reputation for being indolent and insidious in nature. These are generally infectious or inflammatory and include mucocele or mucopyocele formation, late frontal sinusitis, and brain abscess secondary to a frontal sinus infection. Some of these complications appear many years after the original injury. The delay between injury and appearance of a complication may draw into question the true relationship between the injury and subsequent complications.

Mucocele and Mucopyocele Formation

Respiratory mucosa trapped between fracture segments or remaining after sinus obliteration may continue to grow, resulting in mucoceles or mucopyocele, sometimes decades after the injury. A mucocele is an expansive cyst filled with mucus secreted by goblet cells in the ciliated mucosa.[110] In the post-traumatic setting, this is usually a late manifestation of frontal recess obstruction. Mucoceles can range from small to large, with bony expansion and erosion (Fig. 19-19). CT and MRI imaging can help determine the extent and whether there is intracranial communication.[111] The expansile nature of this benign lesion accounts for its potential morbidity, which can include brain mass effect and blindness.[112]

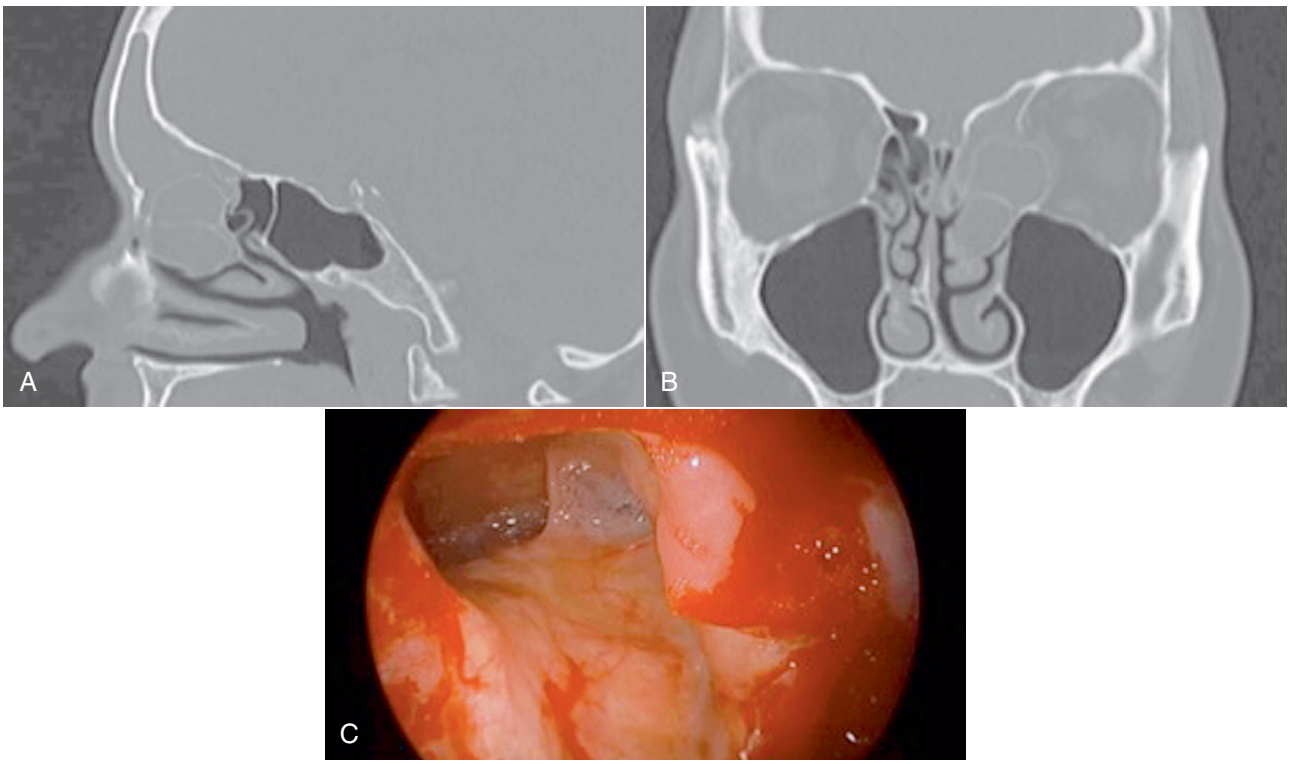


FIGURE 19-19 Saggital **(A)** and coronal **(B)** CT images of two left ethmoidal mucoceles, stacked on each other, with orbit expansion and frontal recess outflow obstruction. This patient was treated endoscopically, with removal of the mucoceles. **C,** Endoscopic view of left frontal sinus.

Surgical treatment of a mucocele includes endoscopic, external, and combined approaches. A simple marsupialization with restoration of the frontal outflow tract is an effective option, with low morbidity. This may be the best option in a mucocele that erodes the posterior wall of the sphenoid or frontal sinus. Removal of the posterior aspect of the mucocele would potentially result in a CSF leak. Leaving it maintains sinonasal and intracranial separation. Frontal sinus obliteration and cranialization are other options.[110]

Mucopyoceles are infected mucoceles and are more likely to be acutely symptomatic (Fig. 19-20). Management is more urgent and intracranial and/or intraorbital extensions are more common.[113] IV antibiotics are included in their treatment. Surgical management includes the same options as listed above.[114]

SUMMARY

The management of frontal sinus injuries continues to challenge craniomaxillofacial trauma surgeons because of the low incidence of injury, regional anatomic complexity, and absence of good data supporting clinical decision making. The goals of management are structural protection for the intracranial structures, isolation of the intracranial and extracranial compartments, restoration of natural frontal contour, and prevention of infectious and inflammatory complications. Frontoorbital fractures involving the central face are most influenced by the frontal sinus and require complex decision making regarding treatment. Operative treatment should proceed based on the complexity of frontobasilar involvement, presence of persistent CSF leakage, degree of nasofrontal drainage disruption, and degree of anterior table displacement. Complications may occur many years after treatment, requiring long-term radiographic and clinical follow-up.

ACKNOWLEDGMENTS

We wish to thank Drs. Sung-Kiang Chuang and Thomas B. Dodson for their previous contributions to this chapter.

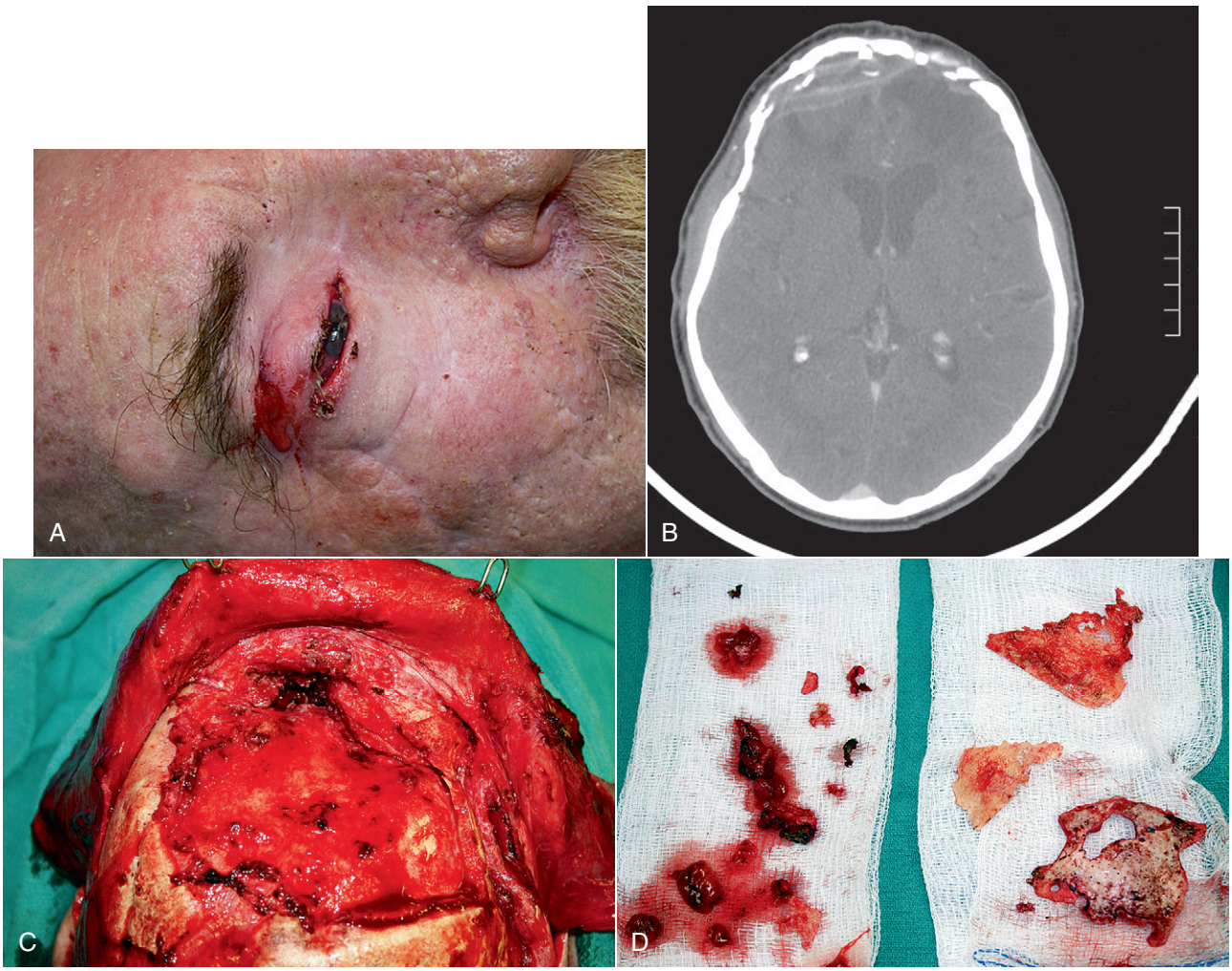


FIGURE 19-20 Mucopyocele complicating frontal sinus repair. **A,** Cutaneous fistula and purulent drainage. **B,** CT demonstrates frontal epidural abscess, mucocele, and osteomyelitis. **C,** Aerodigestive communication with the intracranial cavity. **D,** Mucocele and osteomyelitis.

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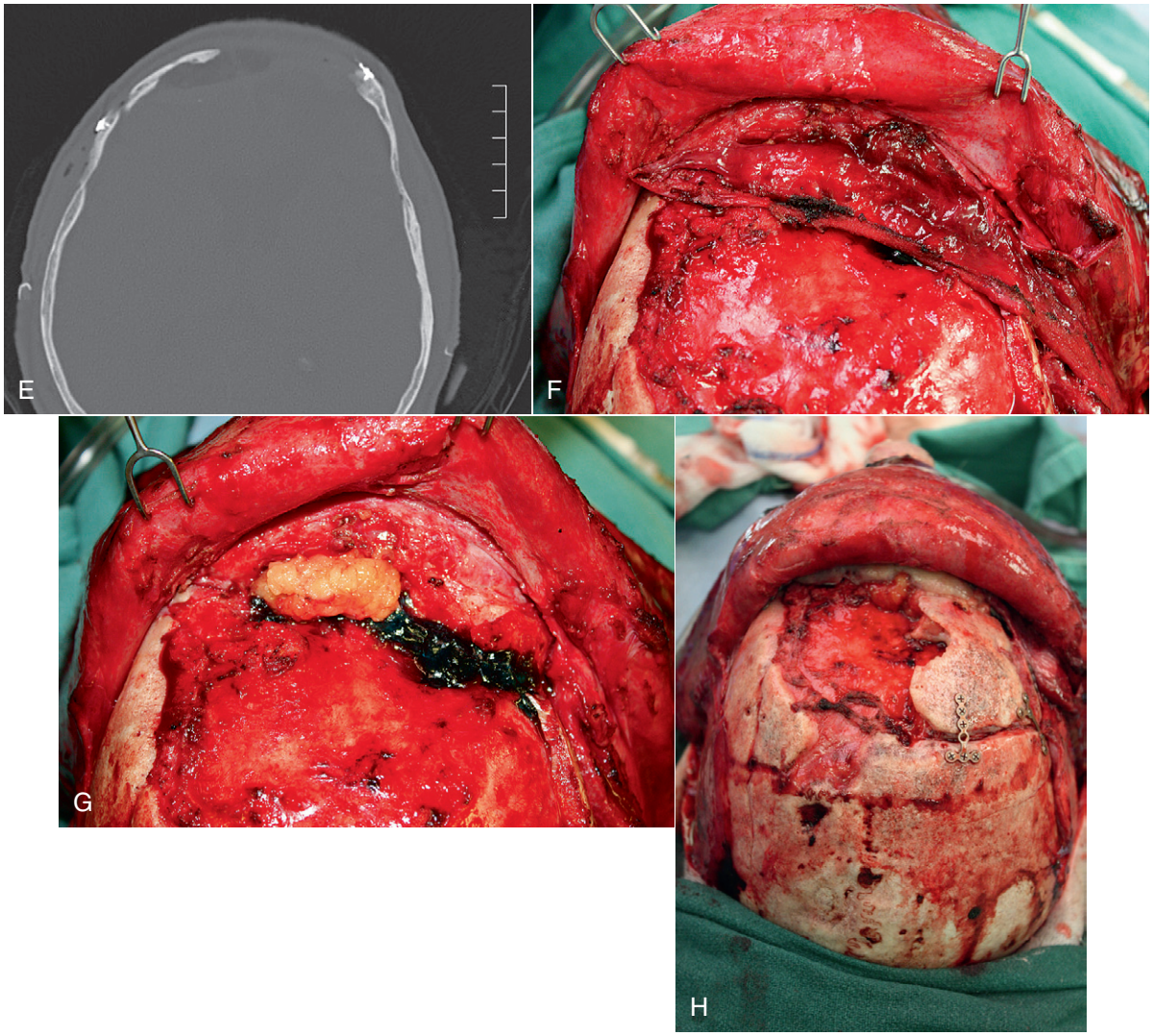


FIGURE 19-20, cont'd **E,** CT scan showing débridement. **F,** Pedicled soft tissue coverage of the NFOT. **G,** Autogenous fat–layered closure. **H,** Reconstructed bone coverage using titanium miniplates.

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CHAPTER

20

Nasal Fractures: Evaluation and Management

Sharon Aronovich | Bernard J. Costello

OUTLINE

Function of the Nose
Epidemiology
Anatomy
Evaluation of Injury to the Nose
History and Physical Examination
Nasal Patency and Airflow Dynamics
Radiographic Examination
Treatment of Nasal Injuries
Closed Reduction of Nasal Fractures
Open Treatment of Severe Nasal Injury
Post-Traumatic Deformity
Special Considerations for Pediatric Patients
Complications
Prevention of Nasal Injuries

Nasal trauma is a common occurrence in pediatric and adult patients because of the exposed and central position of the nose on the face.[1-4] There is a broad spectrum of primary and secondary nasal trauma deformities that the craniomaxillofacial surgeon must be able to address. This chapter reviews the evaluation and treatment of nasal trauma and secondary nasal deformities.

FUNCTION OF THE NOSE

The nose and perinasal tissues have functional and aesthetic roles. The aesthetic value of the nose is a key aspect of facial appearance and a very recognizable feature of the face. It is also the most exposed and prominent area of the face. The nasal form is directly related to its function. When treating nasal trauma, clinicians should aim at optimizing function as a primary objective in conjunction with the aesthetic goals. Nasal function is important for normal respiration, humidification, speech production, and sensations associated with smell and as an aesthetic facial feature.

EPIDEMIOLOGY

Nasal fractures are the most common facial fractures seen and occur at least twice as often in males as in females.[1-6] The high frequency of nasal fractures can be attributed to its prominent location in the facial skeleton and the comparatively smaller amount of force needed to produce a fracture when compared with other facial bones. Athletic injuries, interpersonal altercations, falls, and motor vehicle accidents (MVAs) account for the greatest proportion of causes. In the children and adolescents, sports and falls are the major causes of isolated nasal bone fractures.[2-4,7,8] For example, a study of sports-related fractures in U.S. high schools has found a 10.1% rate of fractures with the highest rate in football, basketball, and soccer.[4] The investigators found that when facial fractures occurred, nasal fractures were the most common injury and resulted in more than 3 weeks of time lost or medical disqualification from participation in sports, in addition to incurring substantial costs to the patient's family and health care system.

Regional differences in the presentation of nasal fractures can be observed. In a retrospective study of Brazilian children aged 5 to 17 years, Cavalcanti and Melo have found that facial injuries were 3-fold more frequent in males aged 13 to 17 years; the most common causes of these injuries were falls and MVAs.[3] Among facial injuries in that study, nasal fractures were also most common, 51.3%, followed by the zygomatic-orbital complex, 25.4%. In another retrospective study, Hwang et al have reviewed and analyzed the medical records of 236 patients with facial bone fractures caused by athletic activity who were treated at one institution between 1996 and 2007.[9] The investigators noted that the age group with the highest frequency of these injuries was 11 to 20 years (40.3%), with a significant male predominance across all age groups (13.75:1). There were 128 isolated nasal fractures, with soccer accounting for 39%, followed by other sports, including martial arts.

Nasal injuries have been shown to be common in adults and children and may be associated with significant morbidity. Associated injuries of adjacent structures should be suspected based on common injury patterns. The age and the environment play a key role in determining the injury incidence and pattern.

ANATOMY

The nasal bones are paired and joined at the midline; they articulate with the frontal bone superiorly and the frontal process of the maxillary bones laterally. Caudally,

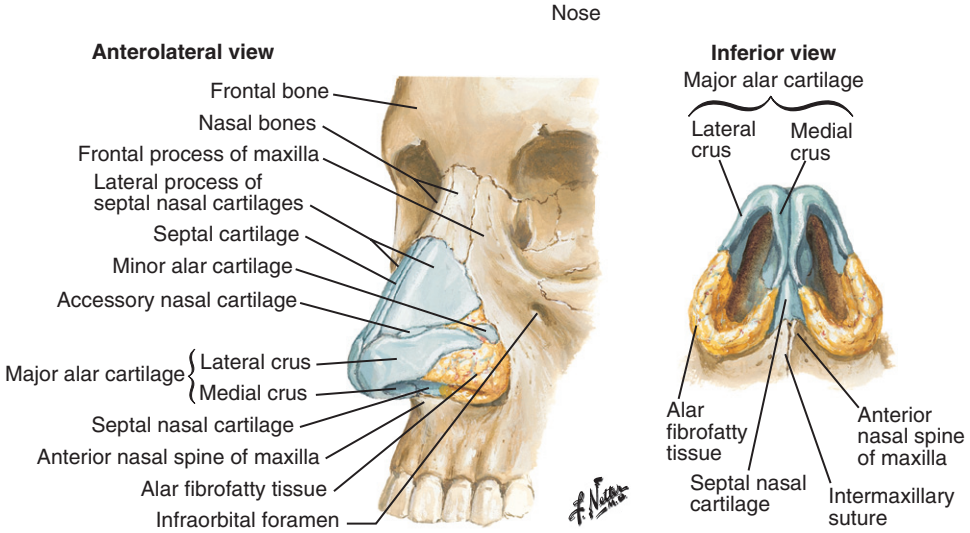


FIGURE 20-1 Bone and cartilage structures of the nose. (Netter illustration from www.netterimages.com, © Elsevier Inc., All rights reserved.)

they interact with the upper quadrilateral cartilages. Basally, they articulate with the nasal septum, which is formed by the perpendicular process of the ethmoid and the vomer inferiorly (Fig. 20-1).

Nasal air passage may be affected by a variety of conditions. Anatomically the internal and external nasal valves must be patent, with the former having an angle of at least 10 to 15 degrees. A deviated nasal septum, nasal spurs, concha bullosa, nasal polyps, and inflammatory sinonasal disease represent examples of altered anatomy that potentially become sources of nasal obstruction and may contribute to nasal airflow obstruction (Fig. 20-2).

The neurosensory innervations of the nose are rich and complex (Fig. 20-3). The overlying nasal skin receives its innervation from the dorsal nasal and external nasal nerves, branches of the anterior ethmoidal and ophthalmic (to infratrocheal) nerves, respectively. Intranasally, the septum and lateral walls are innervated by branches of the sphenopalatine and anterior and posterior ethmoidal nerves. The nasal floor receives some fibers from the nasopalatine nerve and from the greater palatine nerve. The parasympathetic supply originates from the superior salivatory nucleus in the medulla, travels with the nervus intermedius and cranial nerve VII to the geniculate ganglion, and continues along the greater petrosal nerve and through the vidian canal to reach the pterygopalatine ganglion. The postganglionic fibers travel to the sinonasal mucosal, including the septum and turbinates.

Although concerns exist among clinicians about nasal tip edema or vascular insufficiency resulting from operative management of nasal injuries and the use of vasoconstrictors, this has not been substantiated by research studies. Lymphoscintigraphy, along with cadaver dissections and histologic studies, have revealed that the primary blood supply for the nasal tip arises from the lateral nasal branches of the facial artery (Fig. 20-4). Other arteries supplying the nasal tip include the columellar branch of the superior labial artery and the external nasal branch of the anterior ethmoid artery. Moreover, it was found that the major arterial, venous, and lymphatic vessels course at or superficial to the musculoaponeurotic layer of the nose. Therefore, open approaches must limit dissection deep to this musculoaponeurotic layer to maintain nasal tip blood supply and drainage conduits.[10]

EVALUATION OF INJURY TO THE NOSE

HISTORY AND PHYSICAL EXAMINATION

A detailed history exploring the mechanism and timing of injury, and loss of consciousness, will help distinguish isolated nasal and septal fractures from those likely to be associated with other injuries. Any history of prior nasal and/or septal trauma or surgery should also be documented. Recent pretraumatic photographs may be helpful to appreciate the extent of nasal and septal deformities. The possibility of nonaccidental trauma must be considered. Concussion and brain injury symptoms such as headache, nausea or emesis, dizziness, disorientation, or lethargy must be elicited and managed appropriately by the trauma and neurology teams.

Observing the nasal dorsum from the frontal, worm's eye, and bird's eye views will help the clinician appreciate any external nasal deformities. Overlying edema is frequently present with decreased definition of the melonasal angle and may mask the severity of the underlying skeletal deformity. The clinician should examine for the presence of nasal and septal deviation, step deformities, and crepitus on palpation. The intranasal examination with a nasal speculum, appropriate lighting, suction, and vasoconstrictor is important to assess the status of the cartilaginous and bony nasal septum, rule out septal hematoma, and determine the origin and extent of epistaxis and/or cerebrospinal fluid (CSF) rhinorrhea. In some cases, it may be helpful to use nasoendoscopy with a fiberoptic scope to evaluate the posterior nasal complex and nasopharynx further. Those skilled in the use of this type of instrumentation may also find it helpful to evaluate the posterior airway and the presence of bleeding.

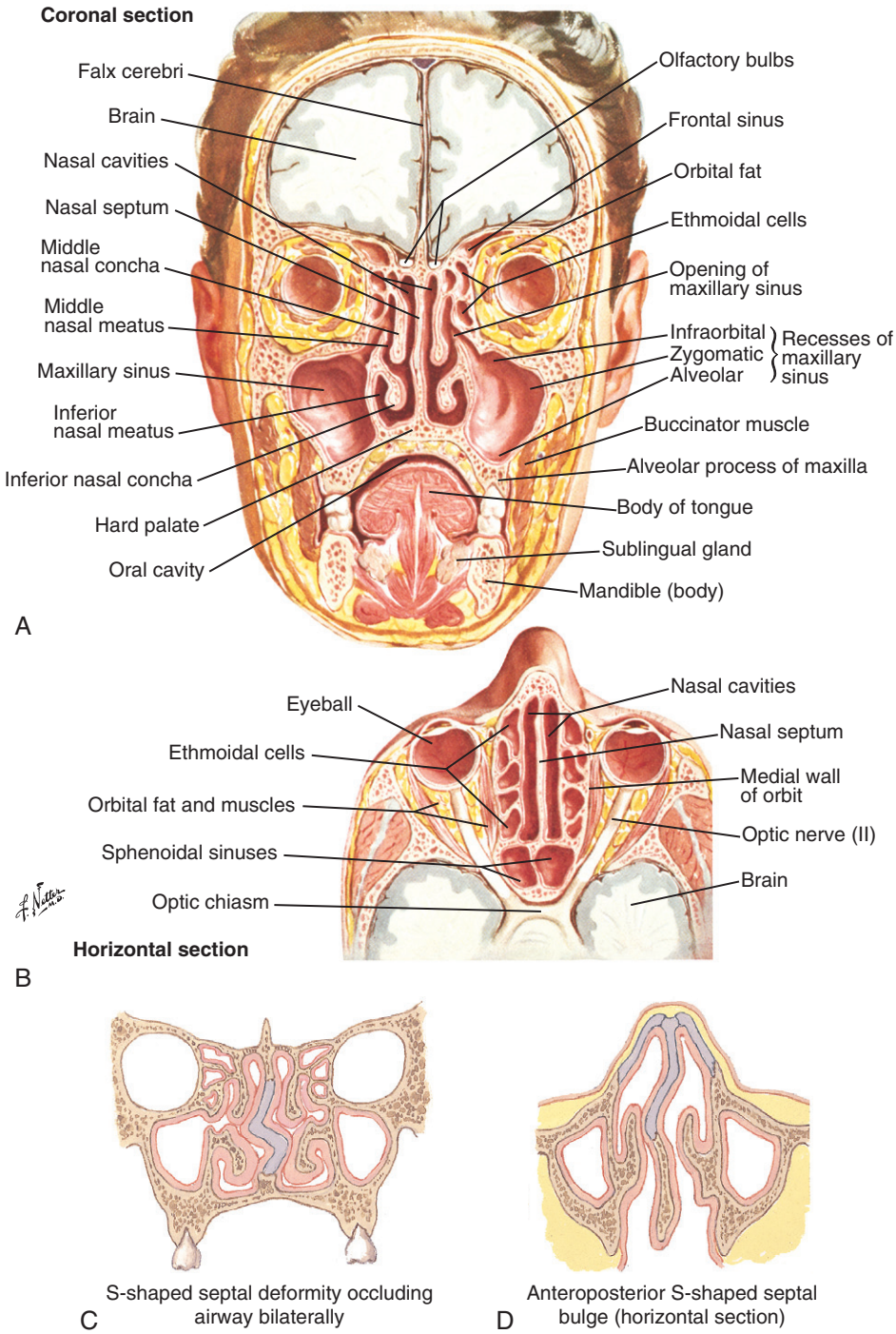


FIGURE 20-2 Internal structures of the nose. **A, B,** coronal and axial sections of the nasal region. **C, D,** Coronal and axial sections of the deviated septum that is S-shaped and obstructing both nasal cavities. (Netter illustrations from www.netterimages.com, © Elsevier Inc., All rights reserved.)

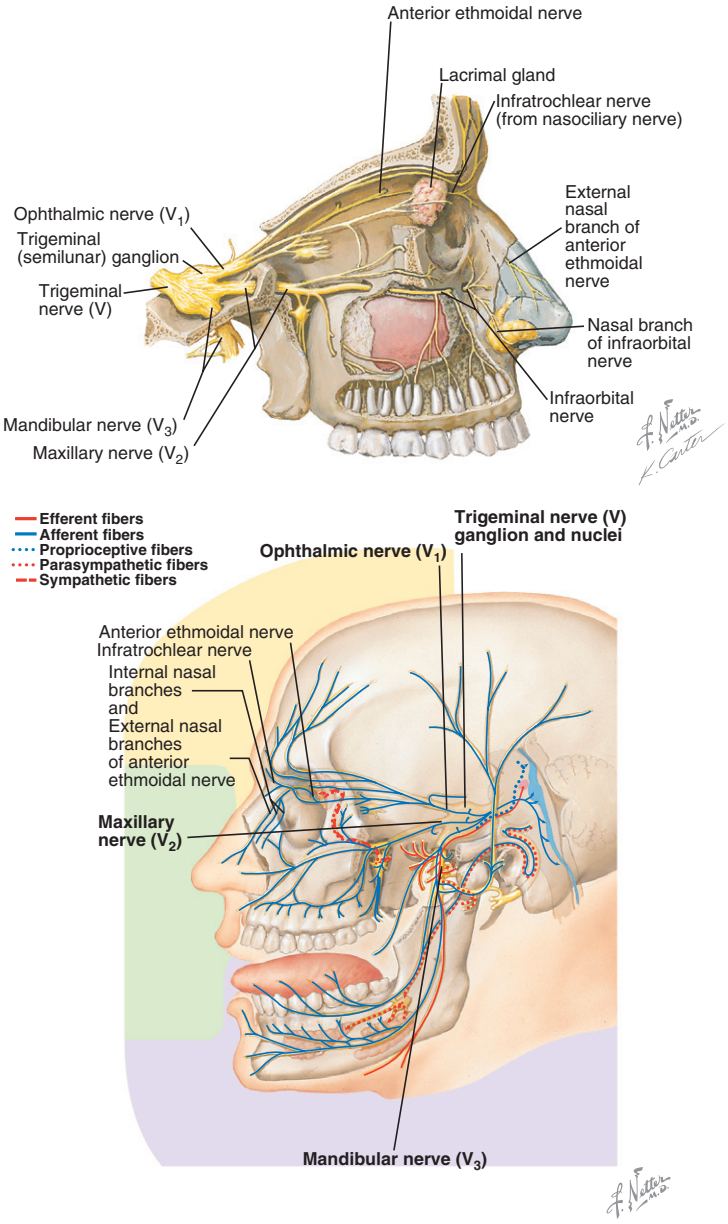


FIGURE 20-3 Neuroanatomy of the nasal complex. (Netter illustrations from www.netterimages.com, © Elsevier Inc., All rights reserved.)

A detailed facial examination is also important and should include visual acuity, extraocular muscle movement, pupil size and reactivity, intercanthal distance, medial canthal tendon position, mandibular range of motion, and occlusion. In many cases, forces great enough to fracture the nasal and septal bones are substantial enough to injure adjacent structures, such as the globe and orbit floor.

During the examination, the astute clinician will recognize that some patients may exhibit signs and symptoms of a concussion. As such, a symptom-driven neurologic examination is important in a subset of patients with facial injuries. Sports-related concussions associated with facial fractures are often undertreated and those with other causes may be missed entirely. Patients who have complaints of headaches, irritability, confusion, visual changes related to traumatic eye injury, poor sleep patterns, or other related symptoms should have an evaluation by a properly trained specialist who sees and treats concussions on a regular basis. This is particularly important for patients who plan on returning to physical activity or sports that might involve blows to the head.

NASAL PATENCY AND AIRFLOW DYNAMICS

The internal nasal valve primarily determines nasal resistance to airflow because this is typically the narrowest zone.[11-14] This valve is triangular in shape and is formed by the junction of the caudal upper lateral cartilages and nasal septum.[15-23] Inferiorly, it is bound by the nasal floor and posteriorly by the inferior turbinates. It has a cross-sectional area of approximately 55 mm^2, with an angle of 10 to 15 degrees in whites. Fixed and dynamic causes of valve obstruction may be responsible. For instance, dynamic obstruction on inspiration may be seen with post-traumatic weakness of the upper or lower lateral cartilage. The Cottle test, in which the cheek is pulled laterally, may improve nasal airflow by pulling the upper

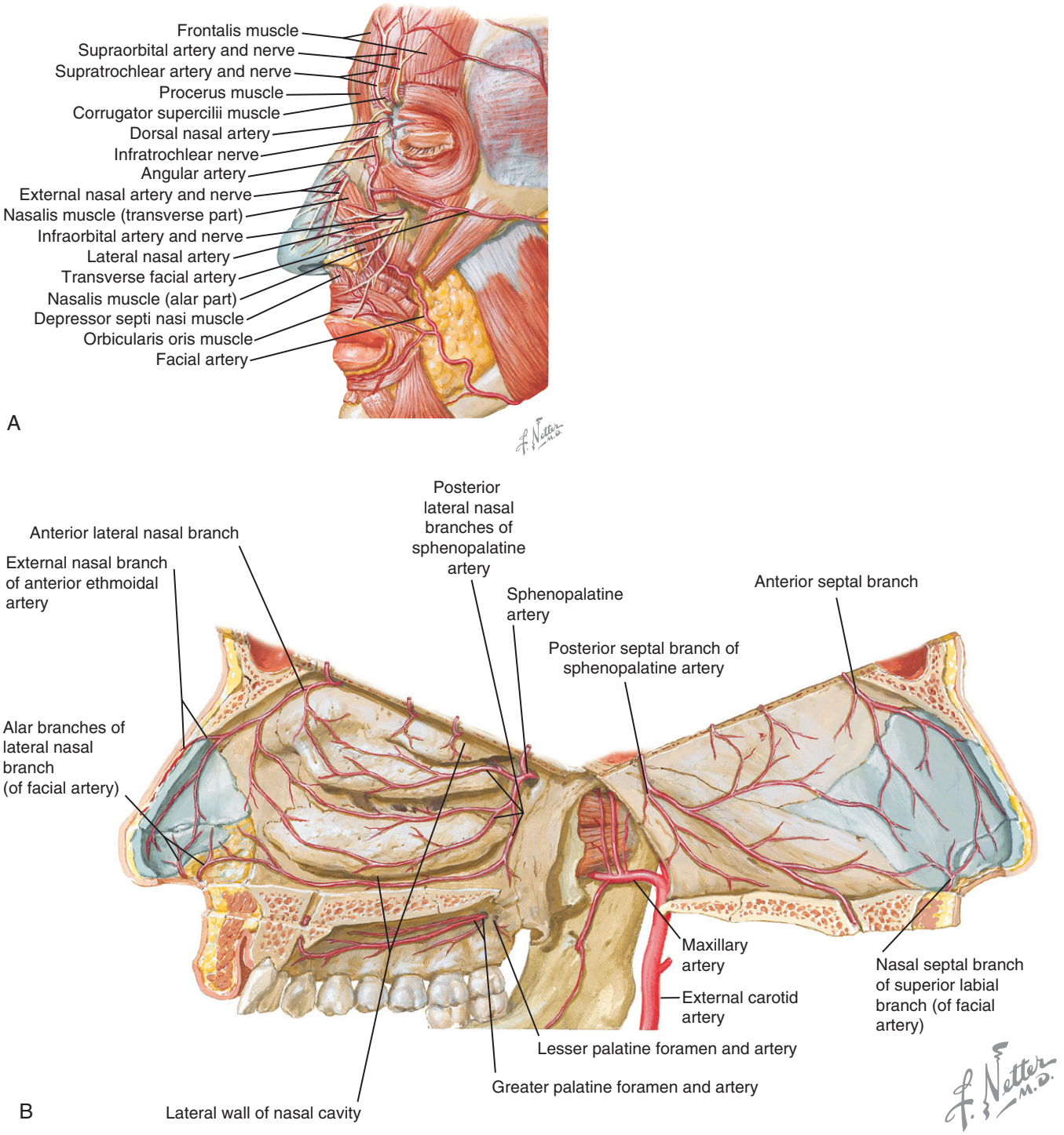


FIGURE 20-4 **A, B,** Vascular supply of the nasal complex. (Netter illustrations from www.netterimages.com, © Elsevier Inc., All rights reserved.)

lateral cartilage laterally and opening the internal valve area.[24] Imaging modalities such as computer tomography (CT) may demonstrate the presence of contributing factors such as sinonasal disease, concha bullosa, and a posterior septal spur or septal deviation.

RADIOGRAPHIC EXAMINATION

A variety of options are available for imaging and categorizing nasal fractures and the surrounding region.[25-30] Many isolated nasal injuries with no loss of consciousness and a clear history of the mechanism can be diagnosed and treated without the need for exposing the patient to radiation. Plain radiographic films may be adequate to assess the extent and displacement of nasal bone fractures when no other injuries are suspected, but they are used less often with the advent of CT.

Additionally, because of extensive overlap of other anatomic structures, some nasal fractures cannot be completely visualized with plain films alone. CT, although not required in every patient, provides excellent detail. In

addition, concomitant fractures such as orbital, naso-orbital ethmoid, zygomatic, maxillary, and frontal sinus or cribriform plate fractures may be identified more easily and treated more precisely. On occasion, identification of these additional fractures may alert the clinician to possible complications, such as obstruction of the nasolacrimal duct with its associated epiphora or suspicion of a dural tear with CSF rhinorrhea. On occasion, pneumocephalus may be identified if the injuries extend into the anterior cranial base. Recently, cone beam CT has been used in dental offices and other settings to visualize bony lesions, dysmorphology, and fractures. If one suspects injury extensive enough to cause multiple fractures at multiple sites, cone beam CT may not be recommended because imaging of the brain tissues may be needed to evaluate the possibility of epidural or subdural bleeding, parenchymal brain injury, or bleeding in the cisterns.

TREATMENT OF NASAL INJURIES

For most routine nasal fractures, operative management of nasal fractures requires consideration for the timing, anesthetic, setting in which treatment is rendered, and details of the operative approach.[31-33] The recommended time frame to repair nasal fractures is within the first week after injury. It is acceptable to perform immediate treatment, but at times it may be helpful to allow swelling to decrease prior to definitive treatment. Some recommend delaying surgery because it is may sometimes be difficult to judge the adequacy of reduction in the presence of significant edema. However, surgeons must remember that the elastic cartilaginous framework may be difficult to reduce as fibrin organization and fibrosis ensues; thus, delayed repairs beyond 1 week may become increasingly more difficult to treat with closed reduction alone. Children have a tendency to heal the bony tissue more quickly, which may make it more difficult to achieve a detailed repositioning. Treatment within the first several days facilitates the healing process, limits patient disability, and decreases the patient's time spent away from school, work, or other activities.[34]

Local anesthesia or the use of sedation can be used, but it may put the patient at increased risk for bleeding in and around the airway. This can lead to laryngospasm in patients who are obtunded, particularly children. When the nasal complex is bleeding, it may complicate airway management and reduce the surgeon's ability to reduce the fracture adequately under good conditions in a safe manner. We prefer to treat nasal and septal fractures with a brief general anesthetic in the operating room using an endotracheal tube or laryngeal mask ventilation device. Either airway option can be used for adults or children.

While the authors prefer general anesthesia for children during operative manipulation of nasal fractures. However, there are advocates for the choice of either local or general anesthesia, and some data to support that decision. Contributing factors may include patient cooperation, financial constraints, operator experience or comfort, and others.

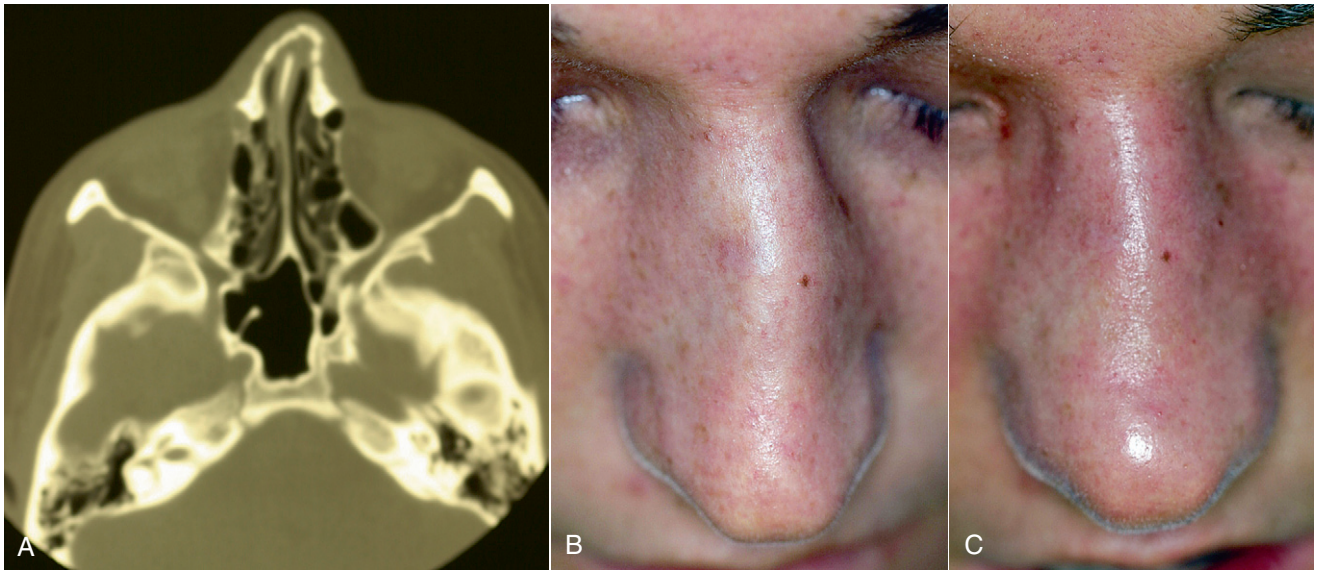
Cook et al compared the manipulation and reduction of nasal fractures under local anesthesia (LA) and general anesthesia (GA). In this study, LA was administered with 0.5% bupivacaine blocking the infraorbital, infratrochlear, and external nasal nerves.[35] Patients in the LA group rated how painful the combined anesthesia administration and fracture manipulation had been on a scale from 1 to 5 at the 4-hour and 8-week time point after reduction. They also rated nasal airway patency and the surgeon rated the cosmetic result. After manipulation, the patency of nasal airways was comparable between the LA and GA groups. The local anesthesia group rated their median pain score as 3 out of 5. Pain was attributable to the infiltration of LA solution. When asked about their choice of anesthesia in case they would require nasal manipulation in the future, 24 of 25 patient in the LA group and 16 of 25 in the GA group indicated that they would opt for LA. There were no reported cases of perioperative airway compromise documented in these patients.

The same investigators also randomized two LA techniques among 50 consecutive adult patients with clinically displaced nasal fractures.[36] One group received blocks of the infraorbital, infratrochlear, and external nasal nerves by intranasal infiltration and the other group was given generalized infiltration of the nasal dorsum by an external route. All patients received intranasal cocaine. Postoperatively, patients recorded their overall discomfort level and subjective nasal airway patency. The surgeon also recorded the cosmetic result. They found the internal route to be significantly more painful ($p < .001$) and with no advantage to the patient with respect to postoperative airway patency or cosmesis. Their study suggested that the external method is preferable. Epistaxis was never severe enough in any of the patients to warrant nasal packing or hospital admission. In addition, the group undergoing intranasal infiltration expressed a greater willingness to have GA in case they were to have the procedure again as compared with the external infiltration group, but the difference did not reach statistical significance. The authors noted that local anesthesia is an effective, economical, and well-tolerated treatment option for closed reduction of nasal fractures in adults. However, they cautioned against using LA in pediatric patients because of behavioral and compliance issues, anxiety, and airway concerns.

Although the operative approach for isolated nasal bone fractures typically consists of closed reduction, nasal fractures associated with significant obstruction, septal deviation that is not reducible with closed maneuvers, or loss of nasal septal support may require open repair and reconstruction. The open techniques are sometimes accomplished with osseous and/or cartilaginous grafts, primarily or in a secondary staged procedure.

CLOSED REDUCTION OF NASAL FRACTURES

Most nasal fractures are treated with closed reduction and various forms of external stabilization, with or without packing materials for the internal nose (Fig. 20-5). This approach efficiently repositions the



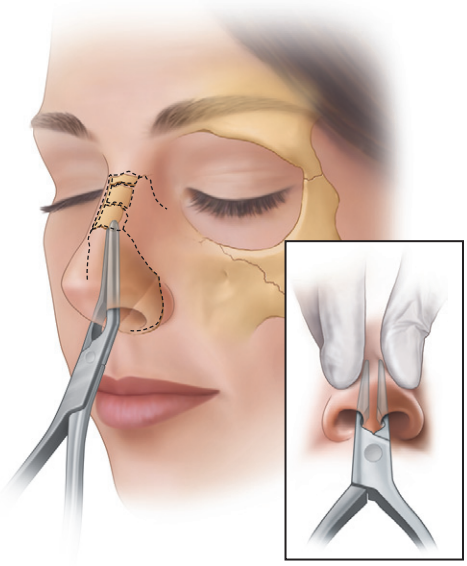








FIGURE 20-5 **A,** Axial CT scan shows displacement of a nasal fracture in a teenager after an assault. **B,** Displacement is evident clinically. **C,** After reduction of the nasal and septal bones, the nasal complex is more symmetric. **D-G,** Diagrammatic representation of the procedure typically performed for reduction of the nasal and septal bones, with external and internal splinting of the reductions.

comminuted segments and is successful for achieving appropriate form and function for most injuries.

After induction of general anesthesia, the airway is secured with an oral endotracheal tube (ETT) or laryngeal mask airway (LMA). A throat pack may be placed, if desired. Although corneal protection is encouraged, adequate clearance must be present for nasal manipulation and application of a nasal splint. The extent of the nasal deformity and deviation is noted from the frontal, worm's eye, and bird's eye views. An appropriately sized nasal speculum and good suction help provide good visualization of the nasal cavity. It is important to reexamine the nose under these more ideal conditions, including the nasal septal form, integrity of the nasal floor, nasal turbinates size, patency of external and internal valves, presence of bleeding sites, and integrity of the cartilaginous structures.

A local anesthetic is injected for postoperative patient comfort, blocking the infraorbital nerves bilaterally. Local anesthetic may also be used intranasally to block the anterior ethmoidal, sphenopalatine, nasopalatine, and superior labial nerves; however, care must be taken not to distort the overlying dorsal nasal soft tissues. A vasoconstrictor such as oxymetolazone nasal spray is applied bilaterally. In addition, cotton pledgets or neurosponge patties soaked in oxymetolazone are inserted for optimal mucosal vasoconstriction prior to manipulation. Care should be taken to avoid drops of oxymetazoline from contacting the cornea or sclera, because this leads to decreased tear volume and tear flow. In addition, an associated anesthetic sensation may promote a postoperative corneal abrasion; patients will often scratch their eyes after extubation prior to arriving at the postoperative care unit and create a significant corneal abrasion.

Nasal bones can be repositioned with digital manipulation using the maxillary and frontal bones for anchorage and guidance for reduction. Adequacy of the reduction is determined by palpation and visual inspection from multiple views, as noted. The Goldman elevator may be used to restore nasal septal projection, lifting the overlying nasal bones superiorly and anteriorly while palpating with an overlying finger. The instrument is also run along the superior-lateral portion of the pyriform rim to appreciate any step deformities. A common error made by inexperienced surgeons is to overproject the fractured nasal bones more laterally and anteriorly than their original positions, thereby giving the patient a much broader appearance to the lower portion of the nasal bones as they integrate with the maxillary bones laterally and inferiorly.

The Asch forceps may be used to assess and treat nasal septal deviations. Care must be taken because these maneuvers may damage or lacerate the nasal mucosa. Some prefer using the Walsham forceps and Boies elevator. Throughout the operative manipulation, the nasal cavity should be suctioned aggressively to minimize blood pooling in the nasopharynx and hypopharynx.

Significant septum manipulation or mucosal disruption may warrant placing a septal splint secured with mattress sutures bilaterally. Gauze packing, ¼ inch, or commercially produced packs coated with antibacterial ointment may be used. Nasal packs and the splint help stabilize comminuted nasal bones and prevent inward collapse of bony fragments. Packing may allow control of bleeding and prevent septal hematoma formation. Over the long term, they can prevent synechiae in patients with extensive mucosal laceration and abrasion because the swollen and displaced mucosa can become inappropriately healed to an adjacent site, causing significant obstruction.

On occasion, formal posterior nasal packing may be needed to control bleeding temporarily. A posterior nasal pack may consist of three cottonoids tied together and introduced orally to the nasopharynx via a red rubber catheter that is passed nasally and retrieved in the oropharynx. Alternatively, a posterior nasal pack may be quickly established with the following: a Foley catheter introduced nasally and inflated for proper seal after passing the velopharyngeal valve; or one of several commercially available devices that have balloons available to inflate for various areas of the nose. Bleeding can then be addressed in the operating room with electrocautery or other local measures. Rarely, extended surgical maneuvers for control of bleeding or interventional radiology techniques may be necessary for recalcitrant bleeding. Appropriate evaluation for the potential for blood dyscrasias should be carried out in those who have unusual bleeding.

Finally, an external nasal splint may be applied. Splints are custom-made from plaster, cut and shaped to fit and made out of metallic and cloth material, or customized from a thermoplastic material that is softened in hot water. The overlying skin is cleaned and dried. A mild skin adhesive is applied onto the nasal skin, and ¼-inch strips are placed from the radix to the tip to protect the skin from thermal injury and provide a sticky surface for the splint. One strip may be applied as a sling, suspending the nasal tip to the nasal dorsum superiorly and providing upward support to the tip during healing. Another thin coat of adhesive is then applied over the wound strips, being careful not to allow the adhesive to drip in the eye, because this can cause significant damage to the cornea. The thermoplastic splint material is trimmed and dipped into the hot water bath to make it pliable and moldable. Care is taken to avoid thermal injury to the patient's skin. The splint should be molded with active digital pressure along the entire length as it cools and sets. The splint helps maintain the symmetrical position of the nasal bones and protects the injured area from displacement by external forces. It also helps reduce the overlying soft tissue edema. It is usually triangular in shape and should not extend too closely to the medial canthi, nasal tip, or lateral ala region.

Nasal packs are usually removed 1 to 3 days after surgery. The septal splint may be maintained for 1 week or longer, as needed. Patients are instructed to avoid strenuous physical activity for a period of 4 to 6 weeks while the disrupted nasal bones, cartilage, and mucosa heal. Exertion may bring about epistaxis in some patients; it is important to warn patients who may attempt return to sports or other activities about this risk.

Patients are followed postoperatively and are evaluated for a patent nasal passage and unhindered nasal airflow, as well as nasal septal symmetry and aesthetics.

Massage of the nasal bones is encouraged, starting between 2 and 4 weeks postoperatively, to mold comminuted fragments and encourage remodeling until a more smooth nasal contour is achieved.

OPEN TREATMENT OF SEVERE NASAL INJURY

Most open approaches to complex nasal fractures are accomplished from a bicoronal incision in coordination with other related fractures such as the frontal sinus and naso-orbital-ethmoid (NOE) complex (Fig. 20-6). NOE fractures are discussed elsewhere in this text. Fixation from above, with small, low-profile fixation devices, is helpful when significant comminution is present. It is important to establish appropriate projection of the nasal bones in conjunction with these other fractured structures. Rarely, primary bone grafting may be helpful at the initial reconstruction. These grafts are usually taken from the readily accessible parietal bone as a partial-thickness graft cantilevered from the nasofrontal junction. Primary bone grafting techniques are helpful for patients who have had an unusual degree of comminution or those who have had the bones avulsed from a unique traumatic injury, such as a dog bite or ballistic injury.[37-40] However, in most cases, the components required for the reduction of even severe fractures are present and can be repositioned without resorting to primary grafting techniques.

Rarely, direct open approaches via more local rather than a coronal incision may be used for isolated severe nasal fracture repairs, such as those from dog bites or ballistic injury (Fig. 20-7). These are generally used because the existing laceration allows for access. Flaps are raised to access the lower or upper lateral cartilages and septum for manipulation, reduction, and fixation. Trans-septal sutures, external splinting, and repairs of the cartilaginous structures may be helpful. Fixation placed through local incisions must have adequate soft tissue coverage to prevent wound healing problems and the devices must be placed accurately with a very low profile.[41]

POST-TRAUMATIC DEFORMITY

A significant number of patients who endure nasal fractures may benefit from secondary rhinoplasty and/or septoplasty to address residual deformities after closed reduction techniques are performed.[42-47] Typically, 6 months or more of healing and remodeling is recommended prior to performing a secondary septorhinoplasty. A discussion of the specific techniques is beyond the scope of this chapter, but the reader is encouraged to review the concepts of rhinoplasty, which include treatment of the deviated nose, asymmetric nasal tip, poorly projected nasal tip, lack of a supra–tip break, nostril deformities, and deviated septum. Nasal bone osteotomies, dorsal reduction, trimming and repositioning of the upper and lower lateral cartilages, alteration of the nasal valve, and use of the columellar strut graft are all helpful techniques commonly used in a secondary rhinoplasty. Alterations of the nasal tip projection, contour, and position can be made using a variety of techniques, including a columellar strut, peck, umbrella, and/or shield grafts.

For most of these injuries, patients will require an open approach with some form of a columellar-splitting incision to gain proper access to each anatomic component (Fig. 20-8). Post-traumatic asymmetrical deformities can be particularly challenging. Surgeons must be comfortable with a wide variety of rhinoplasty techniques to address the post-traumatic nasal deformity successfully and achieve a successful reconstruction.

Septoplasty is commonly required when revision nasal surgery is being considered. A deviated nasal septum can be repositioned using an intranasal approach with unilateral access to the nasal septum via a Killian hemitransfixation incision and subperichondrial plane of dissection. The obstructing midline maxillary crest of bone and inferior septum may need to be removed to allow the remaining septum to hinge back into position. The septum position can be altered by cross-hatching or making radial cuts in the cartilage, allowing the deviated portion to bow in a more appropriate direction. Resection of a deviated component may be a good alternative as long as appropriate structure remains for nasal support. A portion of the resection may be helpful for columellar strut grafting. Alignment of the septum is maintained with trans-septal mattress sutures; stability is provided with an internal nasal splint for approximately 1 week. In addition to septal deviation, separation of the upper lateral cartilage from the nasal bones may collapse the internal nasal valve. Not all intranasal obstruction after trauma should be attributed to a deviated septum. A detailed assessment of the turbinates, nasal valve, nares, and sinus health should be performed. A positive Cottle test may necessitate treatment by placement of spreader grafts.[48] These cartilaginous grafts are placed between the septum and upper lateral cartilages to increase the internal nasal valve angle and improve efficiency of breathing. They may also help the aesthetics of the dorsum in some cases.

A saddle nose deformity often requires an osseous or cartilage dorsal strut graft, which may be harvested from the calvarium (parietal bone), iliac crest, or rib. Inferior subperiosteal and subcutaneous planes of dissection to the nasal tip via a coronal flap or intranasal incision create a pocket of the thin and custom-shaped graft. The graft is shaped, contoured, and cantilevered off the frontal bone with a miniplate or, if placed from below, placed within a self-retaining pocket of dissection. Temporary fixation sutures and an external nasal splint may be used to stabilize a cartilage graft if placed from an intranasal incision (Fig. 20-9).

SPECIAL CONSIDERATIONS FOR PEDIATRIC PATIENTS

Nasal fractures are uncommon in infants and very young children because of the lack of projection and relative elasticity of the underdeveloped nasal bones and associated cartilage. They also lead relatively protected lives during young childhood. Nonetheless, nasal fractures still represent one of the most common types of facial

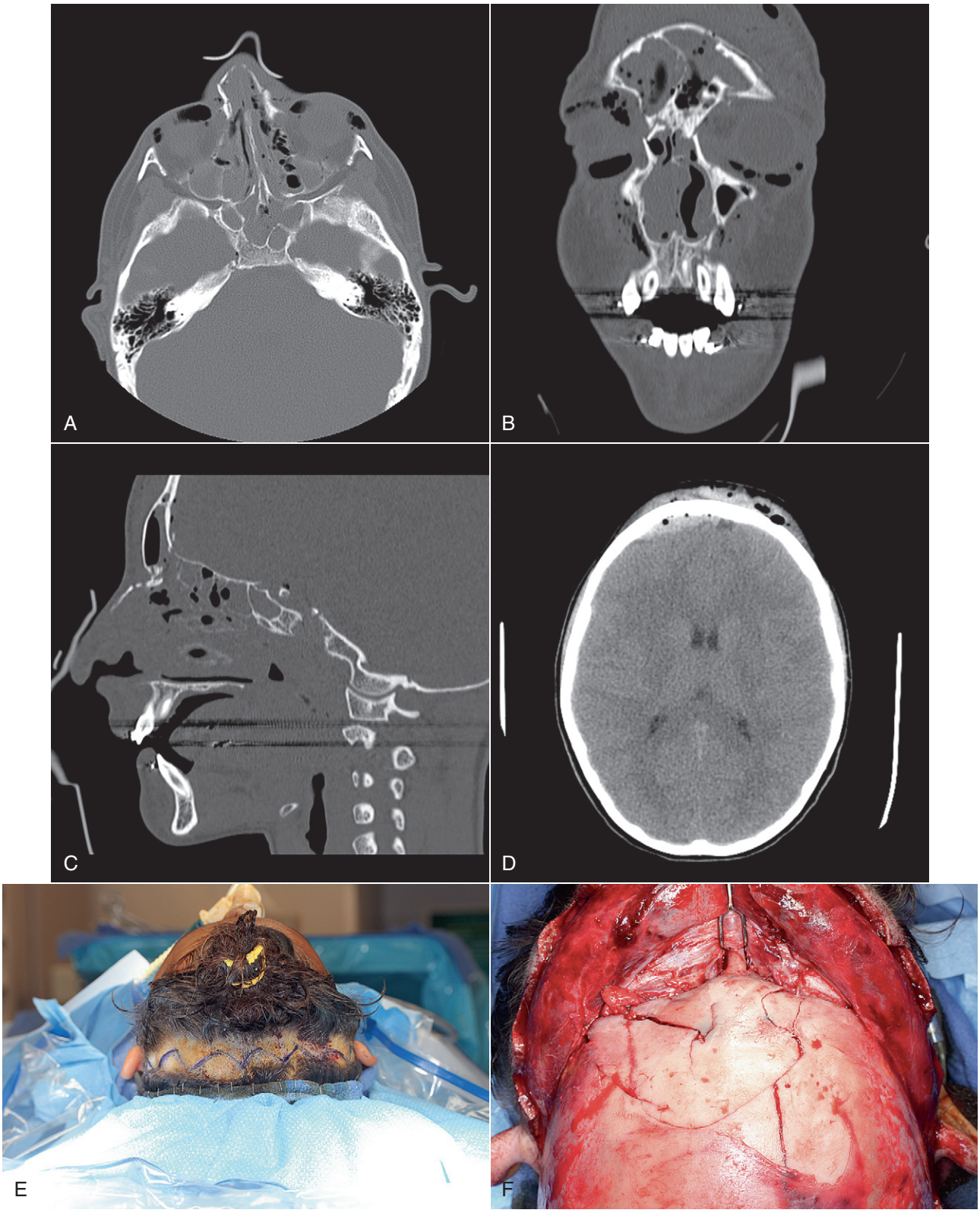


FIGURE 20-6 This teenager was involved in an MVA and sustained complex and cranial injuries, which included a complex nasal fracture. Detailed imaging is required to plan the reconstruction in this type of injury. **A-C,** Axial, coronal, and saggital views of the craniofacial skeleton detail the fractures and their degree of comminution, displacement, and involvement with the cranial base**. D,** The axial view of the skull and brain shows a subdural in the frontal region, with some brain injury. **E,** A zigzag coronal incision is shown prior to dissection. This approach provides full access to the nasal fractures. **F,** The comminuted cranial vault and nasal bones are shown prior to reduction and plating. The open approach provides the appropriate visualization for this level of injury.

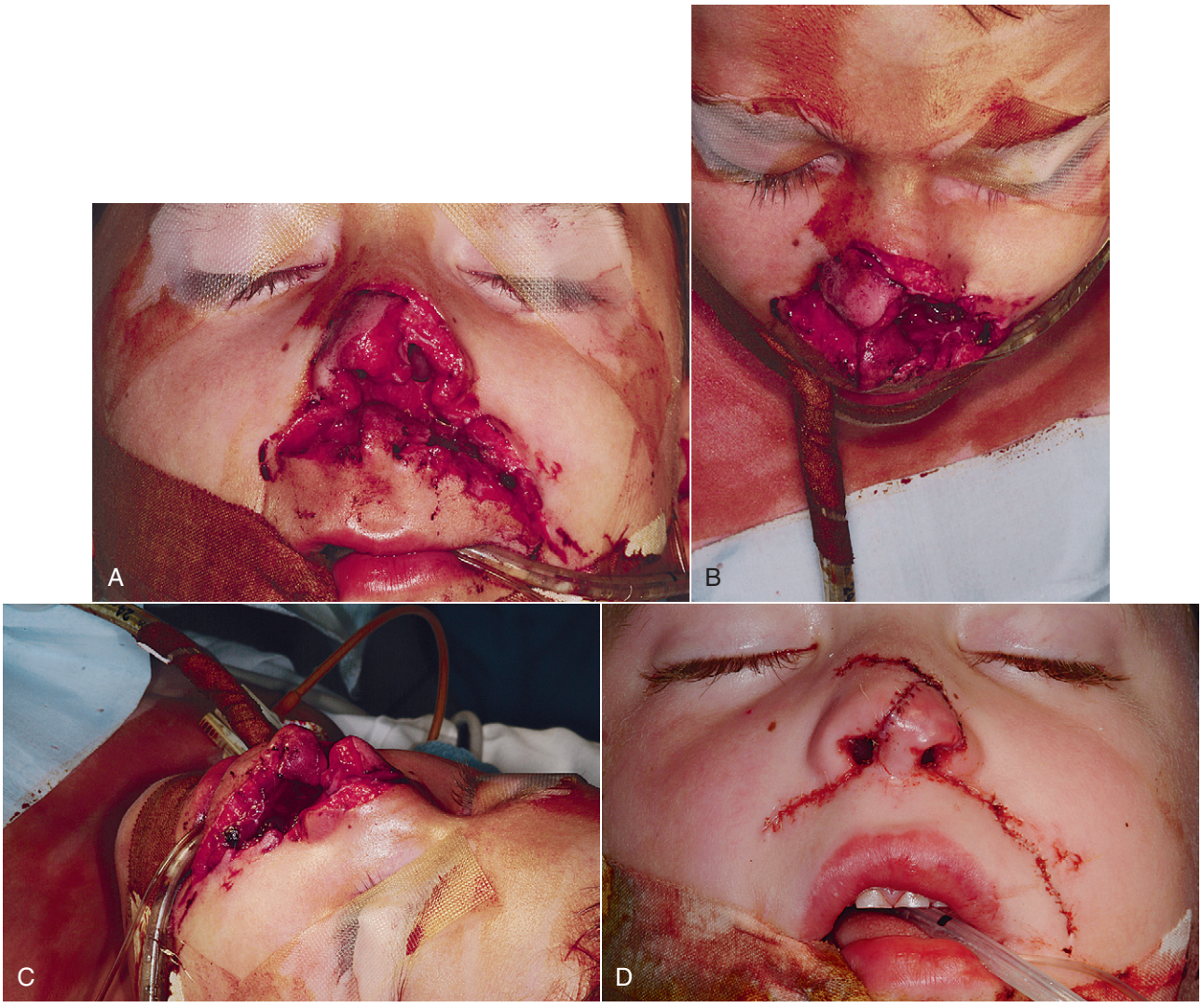


FIGURE 20-7 **A-C,** An open approach is required for this young boy who suffered a dog bite to the nasal complex, with loss of some lower lateral cartilage and overlying soft tissue. **D, E,** Primary repair is achieved with a detailed closure. **F-H,** Initial healing is good, but a revision will likely be helpful once additional growth is complete.



FIGURE 20-7, cont'd



FIGURE 20-8 An open rhinoplasty approach to reposition traumatically disarticulated lower lateral cartilages and a loss of dorsal support after severe nasal trauma sustained from a snowmobile accident.



FIGURE 20-9 A lack of dorsal support is seen 1 year after severe craniofacial, nasal, and septal fractures from a fall from two stories. A dorsal graft is helpful to mask the deformity.

fractures in this group. As a child nears adolescence, the pattern and prevalence of nasal fractures follow more closely that of adults. As with any injury, the clinician should be aware of possible nonaccidental trauma and, when it is suspected, take appropriate steps in the setting of a comprehensive trauma team that has appropriate resources to investigate and deal with any related issues appropriately. In the United States, clinicians are required by law to use the appropriate mechanisms for reporting these injuries if any suspicion exists that the cause is nonaccidental trauma.

There is considerable postnatal growth of the nasomaxillary complex downward and forward because of continued expansion of the brain and cranial base. The nasofrontal suture plays a mediating role. The nasal septum also grows vertically because of its intrinsic and extrinsic growth potential. In addition, as with other bones of the membranous viscerocranium, areas of surface apposition and resorption affect its final shape and size. Injury early in this process may impede growth.[49-52]

Skeletal nasal width has been measured anthropometrically. The nose finishes most of its growth by age 5, but still has significant growth throughout the adolescent years. At 8 years of age, the widest distance between the lateral nasal walls reach roughly 90% of their final size. The adolescent years are marked by significant vertical nasal growth and are primarily limited to cartilage and soft tissue, because the nasal bones usually have completed growth earlier. By the age of 14, most patients will have anatomic components that are more completely grown and there is little difference in fracture patterns or treatment protocols when compared with adults.[52]

In children, nasal fractures are treated in a similar fashion.[53,54] Growth will not typically correct nasal fractures. Closed reduction techniques may be performed at all ages, when necessary. In the young child, the use of internal nasal splints and packing may require additional anesthetics to manage their removal. Parents should be cautioned that secondary rhinoplasty may be helpful to address post-traumatic deformities that may be more obvious or significant later in life. Most of these can be addressed in the teenage years, but more significant deformities can be addressed after the age of 5 years if a substantial psychosocial or functional benefit can be gained with operative intervention. Thus, in those for whom nasal airflow and psychosocial issues are not priorities, treatment of secondary traumatic nasal deformities should be delayed beyond the majority of growth in the area of concern. This approach favors more stable and predictable long-term results. Long-term follow-up is important in children because nasal obstruction, growth disturbances, or cosmetic outcome throughout the different phases of maturation may change.

A fractured and displaced nasal septum may cause airway obstruction and growth disturbances that require timely management. Although standard techniques of closed reduction are appropriate in most cases, open techniques may be necessary in unusual or severe cases. Many believe that acute open repairs are best avoided until maturation of the nasal complex, whenever possible. Some surgeons also emphasize the importance of avoiding the nasal spine of the maxilla during open reduction septoplasty because this is thought to be an important area of growth. There is little evidence to support this concern, but a conservative approach is used by most, given the potential growth consequences.

COMPLICATIONS

A septal hematoma is a rare complication of nasal trauma and may present with a complaint of nasal obstruction, pain, or fever when infected.[55-60] The intranasal examination typically reveals a distinct bulge of a cherry to purple hue arising from the septum, with palpable fluctuance. It may be unilateral or bilateral, with its epicenter typically over Kiesselbach's plexus. This collection of blood separates the septal cartilage from its perichondrium and should be treated promptly to prevent necrosis of the septal cartilage. Treatment consists of incision and drainage, nasal packing to prevent reaccumulation of the hematoma and promote intimate contact between the perichondrium and nasal septum, and antibiotic coverage.[61-65] Occasionally, a drain may be indicated and intranasal splints sutured to the septum to compress the perichondrium onto the septum. Complications of a septal hematoma include local infection, meningitis and/or brain abscess, nasal airway obstruction, and septal perforation. Rarely, the latter can lead to a loss of dorsal nasal support and a saddle nose deformity.

Epistaxis is a potential complication associated with nasal fractures and may be anterior or posterior. Significant bleeding may compromise the airway. Anterior epistaxis typically arises from Kiesselbach's plexus, supplied by the sphenopalatine, anterior ethmoidal, greater palatine, and superior labial arteries. Posterior nasal epistaxis may arise from the proximal aspect of the anterior ethmoidal, posterior ethmoidal, or

sphenopalatine arteries. These are located posterior to the middle turbinate or at the posterior superior aspect of the nasal cavity. The surgeon should position the patient upright, with his or her head slightly flexed down to help prevent blood from entering the nasopharynx. Monitoring is helpful because some patients will experience vasovagal syncope. The surgeon can then localize the source of bleeding with proper lighting, a nasal speculum, topical vasoconstriction, and suction. Consideration should be given to topical or local anesthesia with epinephrine. Options for achieving hemostasis include manual pressure, chemical cautery with silver nitrate, local hemostatic agents, and anterior or posterior nasal packing, such as ¼-inch gauze, sponge, or commercially produced packing material. Posterior nasal packing may be accomplished with cotton balls tied together and introduced transorally into the nasopharynx. Balloons and inflatable catheters can also be rapidly applied in case of airway compromise, but are often temporary measures to control bleeding. After passing a Foley catheter transnasally and visualizing it in the oropharynx, the bladder may be inflated with saline and pulled back out to occlude the nasopharynx and the associated choanal arch, which is often the source of posterior nasal bleeding. With a posterior nasal bleed, an additional anterior nasal pack is often necessary for adequate occlusion. Finally, the surgeon must remain cognizant that nasal packing may be uncomfortable and may require procedural sedation or a formal anesthetic.

Many nondisplaced nasal fractures heal without cosmetic or functional deformity. However, of those that require operative treatment, it has been have documented that open and closed reduction techniques for operative fractures produce a high rate of refractory cosmetic deformity.[66] Rohrich and Adams have argued that despite treatment, there is a 14% to 50% incidence of post-traumatic nasal deformity.[45] They contend that contributing factors for these results include the timing of repair, presence of edema obscuring the underlying deformity, preexisting nasal deformity, occult septal deviation, and type of anesthesia. This opinion has also been expressed in other studies.[29,35,36,46] These findings suggest that long-term follow-up, with critical assessment for nasal form and function, is helpful in many cases. Complications may occur more often than appreciated by many surgeons.

PREVENTION OF NASAL INJURIES

Nasal, midface, and other facial fractures in sports can be prevented with the use of helmets or facial shields. A variety of prefabricated and custom options are available. Some of these devices may be used postoperatively in the late stages of healing as players return to their sport or activity. Making coaches and athletic directors aware of the incidence and potential for injury during many sports activities is important to help prevent their occurrence. Various campaigns from a number of associations are helping to convey this message to schools and other groups.

Procacci et al have reported on the fabrication of individual protective facial masks. Impressions of the patients' faces were taken 1 day after surgery with a mixture of impression plaster, with the eyes covered by cotton gauze and the surrounding hair protected with petroleum jelly.[67] Based on this, polymethyl methacrylate and poly(trimethyl hexamethylene terephthalamide) were used to fabricate the customized facial protection shields. Their design allows for the diversion of forces from the nasal region onto the zygomatic and frontal bones while allowing for unimpeded peripheral vision.

SUMMARY

Skilled management of nasal and septal fractures requires a thorough understanding of facial anatomy, causes of injuries, function and aesthetics of the nose, modern operative techniques, timing for reconstruction, setting and anesthesia choices, and possible complications. Some special considerations warrant attention and occasionally a specialist with particular experience in areas such as complicated post-traumatic rhinoplasty or pediatric fractures. Although most operative repairs have good results, secondary reconstructions are surprisingly common. Accordingly, long-term follow-up may be helpful in select patients, but most patients should be informed of the possible long-term aesthetic and functional consequences of their injuries.

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CHAPTER 21

Management of Soft Tissue Injuries

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OUTLINE

Initial Examination
Wound Contamination
Wound Débridement
Anatomy of the Skin
Local Anesthesia
Articaine Hydrochloride (Septocaine)
Topical Agents
Suture Material
Absorbable Sutures
Nonabsorbable Sutures
Surgical Tape
Surgical Needles
Wound Closure
Classification and Management of Soft Tissue Wounds
Contusions
Abrasions
Lacerations
Avulsion Injuries
Delayed Primary Wound Closure
Skin Grafts
Flaps
Apposition of the Skin Margin to Mucosal Membranes
Animal Bites
Injuries to Structures Requiring Special Treatment
Lip
Ear
Nose
Eyebrow
Eyelid
Oral Mucosa and Tongue
Salivary Glands and Ducts
Lacrimal Apparatus
Scalp
Scar Formation
Burns

Soft tissue injuries to the facial structures are commonly encountered in the treatment of the traumatized patient. Soft tissue wounds may be limited to superficial structures, but more serious injuries may extend to involve anatomic structures, such as the facial bones, the sensory and motor nerves of the face, the parotid, submandibular, or nasolacrimal glands or ducts, and dentoalveolar structures. Soft tissue injuries include abrasions, contusions, clean-cut lacerations, contused lacerations, bites, burns of various degrees, and avulsive wounds.

INITIAL EXAMINATION

The initial management of the injured individual must include establishment of the airway, control of hemorrhage, and stabilization of injuries to other major systems before evaluation of facial soft tissue wounds. Fractures of supporting facial bones then must be excluded by careful clinical and radiographic examination. Fractures should be reduced and stabilized before final soft tissue repair. Facial lacerations do not require immediate treatment because of the excellent blood supply to the facial region. During the assessment period or during treatment of other injuries, the wounds should be kept moist with gauze soaked in an antibiotic solution until final management.

Hemorrhage associated with most head and neck wounds may be substantial but can usually be controlled with local measures of pressure and clamping, ligation, or electrocautery of visibly bleeding vessels. Scalp wounds or disruption of major vessels may result in blood loss to the point of hypovolemic shock. According to Lynch,[1] if a patient exhibits shock with facial trauma, one of three conditions is usually present: (1) the trauma is very extensive and complex, with underlying facial fractures, oropharyngeal wounds, or both and possible intracranial injury; (2) treatment has been inordinately delayed and an extended period of controlled hemorrhage or repeated episodes of bleeding have occurred; or (3) the head and neck wounds are associated with other unrecognized injuries, such as long bone fractures or chest or abdominal trauma. The face is well supplied with blood vessels, which are generally small in diameter and generously supplied with elastic fibers. When the blood vessels of the face are completely transected, they tend to contract and collapse; bleeding stops spontaneously because the vessels become occluded with thrombi and compressed by the enveloping hematoma. However, partially transected vessels have a propensity for continued hemorrhage. With an incomplete laceration of an artery, such as the facial or lingual artery, massive bleeding may occur, possibly producing compression of vital structures and potential airway compromise. Along with concomitant injury to the accompanying vein, an arteriovenous fistula may develop.

Persistent bleeding should be evaluated by direct inspection to prevent damage to other vital structures.

The wound may bleed after cleansing and débridement. Copious irrigation with saline or balanced salt solutions assists with removal of blood clots and granulation tissue that may slowly ooze. Direct pressure helps control bleeding from the wound surface and limits hematoma formation. Hematoma formation is a major cause of infection and wound breakdown. If hemostasis cannot be achieved, drains should be considered.[2]

It is recommended before final treatment of the wounds and after cleansing of the skin that photographic records be obtained for insurance and legal purposes. Lawsuits are initiated more and more on the basis of results—not negligence—and the lay public often expects almost perfect results, whether these are realistic or not.[3]

Follow-up photographs will aid in the assessment of healing and scar maturation and the necessity for future scar revision.[3] After the patient's condition has been stabilized, or if there are minimal associated injuries, definitive treatment of the soft tissue wounds should be carried out (Fig. 21-1). Clean wounds may be closed primarily up to 48 hours following injury. Healing of head wounds (nonoral) has been found to be independent of time from the injury to repair.[4] Head wounds may be physiologically distinct from other types of injuries. The relatively greater vascularity of the scalp and face decreases the susceptibility of open wounds to infection. Head wounds are especially amenable to very late primary closure, days compared with the 18 to 19 hours recommended for nonhead wounds.[4] This delay in the primary closure of soft tissue wounds may be indicated if the supporting facial bones have been fractured. Treatment of the fracture should be completed before final soft tissue closure because the wound may provide access to the fracture site and the closure may be damaged during fracture reduction.

Repair of soft tissue wounds may be done with the patient under local or general anesthesia, depending on circumstances. If injuries are extensive, general anesthesia is indicated. If no jaw fractures exist, it is best to intubate the patient orally for injuries above the occlusal plane and nasally for those below. It may be necessary to change the route of intubation during the procedures. With fractures of the maxilla, mandible, and/or dentoalveolar structures, and with soft tissue injuries, nasoendotracheal intubation is necessary to allow for placement of intermaxillary fixation, if only temporarily, when rigid fixation is used. A tracheotomy may be necessary if airway management dictates it for presurgical, surgical, or postsurgical care.

During the final examination, it is extremely important to evaluate whether vital tissue has been damaged. Deep lacerations across the course of the branches of the facial nerve, hypoglossal nerve, and sensory branch of the trigeminal nerve should be evaluated for possible transection. A nerve stimulator may be helpful to stimulate the appropriate muscle groups in the nonparalyzed patient under general anesthesia. If a nerve has been damaged, appropriate microsurgical techniques should be used to attempt to restore function of the nerve. In some avulsive injuries, secondary nerve graft procedures may be indicated. During the examination phase and initial treatment, the severed nerve trunks should be identified and marked with colored tags so that they can be easily located for future reconstruction procedures.

WOUND CONTAMINATION

Wounds can be divided into two groups, clean and contaminated. Prophylactic antibiotics are usually not indicated in clean fresh lacerations of the skin. The probability of contamination increases rapidly and is directly related to the length of time that has elapsed since the initial injury. The contamination of the clean wound is usually via *Streptococcus* and *Staphylococcus* spp. on the skin of the face and multiple types of bacteria if the mucosal layers are violated. Wounds that involve the mucosal linings of the oral cavity and pharynx, especially through and through lacerations from the skin through the mucosal layers, should be considered contaminated. Saliva may carry normal oral flora to deeper structures and wound infections may develop.[3]

The species of bacteria present are of less concern in the development of an infection than the total number of bacteria present within the wound. The infectious inoculum must exceed 10^5 organisms/g of tissue for gram-positive and gram-negative aerobic bacteria.[6-8] The critical number for anaerobes has not yet been determined. Wounds such as simple lacerations and abrasions have low bacterial content. Crushing of tissue, the embedding of foreign bodies or soil, and perforation into the oral cavity with contamination of saliva markedly increase the bacterial count and set the stage for infection. Wounds caused by impact injuries are 100 times more susceptible to infection than wounds caused by shear forces.[9,10]

The location of the injury may be predictive of the number of pathogens in the wound. In general, the composition of the skin microflora allows for subdivision of the body into three major areas.[9]

High numbers of potentially infective organisms exist in the maxillofacial region and in extremely high numbers—almost double the reported infective dose—in the oral cavity.[11,12] This source of heavy contamination accounts for the high infection rates from skin wounds exposed to saliva and human and animal bites.

Tetanus prophylaxis should be instituted with contaminated wounds (Table 21-1). Two thirds of reported tetanus cases in the United States in recent years have followed lacerations, puncture wounds, or crush type of injuries.[10] With a previously immunized patient, if a course of active immunization has not been given within 10 years of the injury, a booster dose of 0.5 mL of tetanus toxoid is recommended. In nonimmunized patients, passive immunization with hyperimmune (human) tetanus globulin, followed by a course of active tetanus immunization, should be instituted.[1,5,13] Any particularly contaminated wound should be considered for administration of tetanus prophylaxis, even though the patient may have had a booster shot in the past 5 years[9,14] (Table 21-2). Antibiotics such as penicillin, cephalosporin, and other drugs active against gram-positive organisms are the drugs of choice for soft tissue injuries.

Factors that impair host resistance to infection may be classified into those that are localized to the wound and those that are systemic in the host (Box 21-1). In heavily



FIGURE 21-1 **A,** The patient initially came to the emergency room with significant intracranial injuries, which had to be stabilized before her facial wounds could be definitively repaired. Before transfer, the emergency room physicians used large silk sutures and surgical staples to close the multiple facial wounds temporarily. **B,** Soft tissue wounds in the facial region are especially amenable to very late primary closure caused by the relatively greater vascularity of the scalp and soft tissue. The wounds may be primarily closed several days following the injury once the life-threatening injuries have been stabilized, fractures of the underlying bones have been identified, and initial soft tissue swelling and edema have subsided. Waiting for the initial soft tissue swelling associated with the accident to subside provides for careful reapproximation of the wound margins and alignment of the facial structures. **C,** 1 month following repair of the facial lacerations. **D-F,** 4 months following the accident.

contaminated wounds, local anesthetics with vasoconstrictor should be avoided.[15]

WOUND DÉBRIDEMENT

Cleansing of the clean wound involves washing the skin and removing foreign bodies from the wound. Soap does not harm the skin surface, because the thick cornified layer of epidermis protects the underlying tissue surface, but soap may enter the wound and cause cellular damage and necrosis. Toxic materials, such as alcohol, hydrogen peroxide, and benzalkonium chloride, and strong soaps, such as those containing hexachlorophene or povidone-iodine, should not have direct contact with the open wound because these materials kill cells on contact.[16] If these are used around the wound, the wound should be thoroughly irrigated with a balanced salt solution (e.g.,



FIGURE 21-1, cont'd

TABLE 21-1 Characteristics of Tetanus-Prone Wounds

Clinical Features	Tetanus-Prone Wounds	Nontetanus-Prone Wounds
Age of wound	>6 hr	≤6 hr
Configuration	Stellate wound	Linear wound
Depth	>1 cm	≤1cm
Mechanism of injury	Missile, crush, burn, or frostbite	Sharp surface (e.g., knife)
Signs of infection	Present	Absent
Devitalized tissue	Present	Absent
Contaminants (dirt, feces, soil, saliva)	Present	Absent
Denervated or ischemic tissue, or both	Present	Absent

lactated Ringer's solution, normal saline). A rule regarding the application of antiseptic is never to put anything in a wound that could not be comfortably tolerated in the conjunctival sac.[17,18]

If the laceration extends into the scalp, mustache, or beard, the area should be shaved to provide good access for débridement and repair.[19] Areas such as the eyebrows or the hairline should never be shaved but rather clipped closely with scissors to provide a landmark for accurate positioning of the soft tissue during the closure.[20] It is important that the alignment of the eyebrow or hairline not be altered because improper orientation is aesthetically displeasing, and the hair can be used as a guide for reconstruction.

The wound must again be inspected for the presence of foreign materials. A pulsatile type of irrigating device is useful for removing debris, necrotic tissue, and loose material. An abrasive wound that contains ground dirt, glass, or other debris should be scrubbed with a scrub brush or toothbrush and detergent soap to remove the foreign material. A no. 15 blade may be used to scrape material that cannot be brushed clean or to remove deeply embedded particles, frequently seen with blast injuries. All material should be removed and time should be taken to clean the wound as completely as possible. If a contaminant is allowed to remain within the tissue, it may become a source of infection or may heal as a permanent type of tattoo that is difficult to treat with future procedures. Polymyxin B sulfate ointment may be used on the wound to remove residual grease or tar that cannot be removed with routine scrubbing techniques. The wound should then be copiously irrigated with a balanced salt solution. Time spent meticulously débriding traumatic wounds during the primary repair period

TABLE 21-2 Recommendations for Tetanus Prophylaxis

	DTP* (0.5 mL IM)	TIG (250 U IM)	Td¶ (0.5 mL IM)	TIG (250 U IM)
History of adsorbed tetanus toxoid (for children < 7 yr)				
Unknown or less than three doses	Yes†	Yes‡	Yes†	Yes
Three or more doses	No¶	No	No¶	No

NOTE: All dog and cat wounds should be considered tetanus-prone wounds.
*Use diphtheria, tetanus (DT) if pertussis vaccine is contraindicated.
†The primary immunization series should be completed.
‡Administer in contralateral extremity.
§Yes, if the routine immunization schedule has lapsed or if more than 5 years since last dose of tetanus toxoid.
¶Use DTP for children <7 years old (DT if pertussis vaccine is contraindicated); Td is preferred to T if patient is ≥7 years old.
¶Yes, if >5 years since last dose. (More frequent boosters are unnecessary and may accentuate side effects.)
DTP, Diphtheria, tetanus, pertussis; IM, intramuscular; TD, tetanus, diphtheria; TIG, tetanus immune globulin.
Adapted from American College of Emergency Physicians: Tetanus immunization recommendations for persons seven years of age or older. *Ann Emerg Med* 15:1111, 1986; and American College of Emergency Physicians: Tetanus immunization recommendation for persons less than seven years of age. *Ann Emerg Med* 16:1181, 1987.

BOX 21-1 Factors That Impair Host Resistance

LOCAL
Foreign bodies
Necrotic tissue
Contused tissue
Vasoconstrictors (local)
Closure under tension
Hematoma
Dead space
Sutures

SYSTEMIC
Hypovolemic shock (hypoperfusion)
Malnutrition
Diabetes
Atherosclerosis
Corticosteroid therapy
Distant infections
Chemotherapy

will prevent unfavorable or aesthetically displeasing results from infection, hypertrophic scars, and foreign body granulomas.[19,21]

Wounds that have been contaminated with foreign materials, such as dirty gravel, metal, tooth fragments, grass, wood, glass, and organic materials, must be thoroughly examined during the initial treatment phase, and

BOX 21-2 Indications for Removal of Foreign Bodies from Soft Tissue*

- Reactivity (thorns, spines, wood, and other vegetative material)
- Heavy contamination (soil, teeth)
- Toxicity (heavy metals, spines with venom)
- Impingement of vital structures (vessels, nerves, and tendons)
- Impairment of mechanical function (restricted joint mobility)
- Intra-articular location
- Intravascular location
- Persistent pain
- Established infection
- Allergic reaction
- Cosmesis
- Psychological distress

*Adequate débridement includes removal of foreign materials from the wound. Accurate localization before removal is essential. Blind searching is time-consuming and may produce further trauma.

the material must be removed to prevent wound infections (Box 21-2). The wounds should be cleansed with detergent soaps and thoroughly irrigated with lactated Ringer's solution or normal saline. Animal bites should be cleansed with detergents and water to remove the animal's saliva and other contaminants from the wound before closure.

A conservative approach should be the rule in débridement of facial wounds. Débridement should be limited to devitalized tissue and tissue that is stained by road tar or contains dirt or other particles that cannot be removed with meticulous scrubbing.

There are at least three mechanisms whereby devitalized soft tissues potentiate infection: (1) as a culture medium promoting bacterial growth; (2) by inhibition of phagocytosis and subsequent bacterial control by leukocytes; and (3) by the anaerobic environment limiting leukocyte function.[22]

Hydrogen peroxide should not be used routinely to cleanse a wound.[23,24] Studies have shown that hydrogen peroxide actually impedes wound healing and has poor bactericidal activity. Hydrogen peroxide 3% diluted 1:1 does not appear to harm wounds protected by mature granulation tissue but is toxic to fibroblasts unless diluted more than 1:100, at which point it has minimal, if any, bactericidal activity.[25] Povidone-iodine (Betadine Surgical Scrub) is also toxic to fibroblasts and bacteria. The stock solution (10% povidone-iodine, 1% available iodine) must be diluted 1:1000 (1 mL/liter) to prevent fibroblast toxicity. At this concentration, the bactericidal effectiveness has been compromised.

These agents should be kept out of the fresh wound and used only to scrub skin surfaces. A gauze sponge can be folded into the wound to prevent the inadvertent entry of detergents during wound healing. Mechanical scrubbing should be avoided unless there is an overwhelming amount of foreign debris in the wound. Although scrubbing can remove particulate matter from the wound, scrubbing of the wound has been shown to

increase wound inflammation.[24] If scrubbing of a wound is necessary, a fine pore sponge and nonionic surfactant, such as Shur-Clens, should be used to minimize the inflammatory response. When the effect of scrubbing wounds with povidone-iodine or hexachlorophene (pHisoHex) surgical scrub solutions on the infection rate was evaluated, researchers found an increased susceptibility to infection because of the greatly increased inflammatory response produced by these solutions coming into contact with the injured tissue.

In otherwise healthy patients, Dire and Welsh[26] found no statistical difference in infection rates when wounds were irrigated with normal saline, 1% povidone-iodine solution, or a nonionic detergent (Shur-Clens). They noted that the mechanical action of high-pressure irrigation, not the solution used, is more important in the prevention of wound infection. Shur-Clens is a nonionic detergent that can be safely used to cleanse periorbital lacerations. Topical application of this agent to experimental animals and humans did not elicit ocular lesions. In contrast, Betadine, Hibiclens (an antimicrobial soap), and pHisoHex surgical scrub solutions caused notable irritation to the eyes and thus should not be used in the periorbital area.

Two groups of antiseptic agents, containing an iodophor or chlorhexidine, have shown promise for preparation of the intact skin around the wound. Both types exhibit activity against a broad spectrum of organisms. They also have a long shelf life, with no significant inactivation. They display a substantive effect on the skin membrane, suppressing the proliferation of bacteria. However, the superiority of one antiseptic over another is difficult to ascertain because most of the comparative studies involve hand washing rather than washing of the operative site. Although these agents can reduce the bacterial concentration of the skin, they appear to damage wound defenses and invite the development of infection. Consequently, inadvertent spillage of these agents into the wound should be avoided.[9,23]

Spillage of an antiseptic solution into a patient's eye can be disastrous. It has been reported in two patients that accidental exposure to Hibiclens resulted in severe and permanent corneal opacification. In experimental studies, rabbit eyes exposed to Hibiclens developed severe irreversible and progressive corneal damage.[9,27]

Irrigation can remove enough wound bacteria to cross the threshold to noninfected wounds, but only if the irrigant is delivered with sufficiently high pressure to disrupt bacterial adherence to the wound surface mechanically. To be clinically effective, irrigants should be delivered with a fluid jet impacting on the wound with psi of 7 lb. This level of pressure can be generated by forcefully expressing saline from a 35-mL syringe through an 18-gauge needle, but cannot be generated by a bulb syringe or by gravity flow irrigation.[28-30]

Rapid and complete invasion of the wound space by fibroblasts is a critical step in normal healing. Dead tissue fragments, hematomas, and foreign bodies act as physical barriers to fibroblast penetration.[17] Débridement of facial wounds should be limited to obviously devitalized and necrotic tissue. Radical excision of soft tissue in the facial region should be avoided. Because of the rich blood supply to the region, excessive débridement is unnecessary and tissue will survive with a very small pedicle. It is better to err on the side of retaining tissue that may not eventually survive than to remove tissue that is necessary for satisfactory repair of the injury. If the wound margin is extremely irregular and reapproximation is difficult, the irregular edges should be excised to produce clean wound margins and minimize scar formation. Occasionally, additional small incisions are helpful in reapproximating tissue and breaking up straight line scars.

During the final examination, it is extremely important to evaluate whether vital tissue has been damaged. Deep lacerations across the course of the branches of the facial nerve and hypoglossal nerve and the sensory branch of the trigeminal nerve should be evaluated for possible transection. A nerve stimulator may be helpful in stimulating the appropriate muscle groups in the nonparalyzed patient under general anesthesia. If a nerve has been damaged, appropriate microsurgical techniques should be used to attempt to restore function of the nerve. In some avulsive injuries, secondary nerve graft procedures may be indicated. During the examination phase and initial treatment, the severed nerve trunks should be identified and marked with colored tags so that they can be easily located for future reconstruction procedures (Fig. 21-2).

ANATOMY OF THE SKIN

The skin covers the body in varying degrees of thickness, elasticity, texture, and mobility and makes transitions into mucosal membranes about the oral cavity, nostrils, and eyelids. The thickness of the skin on the facial region ranges from 0.013 inch over the upper eyelid, 0.030 to 0.040 inch over most of the face, approximately 0.065 inch over the eyebrows, and 0.080 to 0.090 inch over the neck.[31]

The skin is an extensive sensory organ with numerous nerve endings that provide feedback to touch, pressure, temperature, and painful stimuli (Fig. 21-3). It protects against loss of body fluids caused by dehydration, invasion of pathogenic organisms, and excessive exposure to ultraviolet radiation. The skin is also involved in temperature regulation via heat loss through evaporation.[32] Subcutaneous voluntary muscles in the face and neck allow for movement of the skin and for facial expression.

The skin is composed of the surface layer epidermis and the underlying dermal layer. The epidermis is stratified squamous epithelium with five layers (in order from the surface to the dermal layer): the stratum corneum, stratum lucidum, stratum granulosum, stratum spinosum, and stratum germinativum. The epidermis sends projections into the dermis and irregularities of the dermis interlock with the epidermis; these are termed *epidermal pegs* and *dermal papillae*, respectively.[32]

The stratum germinativum, or basal layer, is usually one or two cells thick and has much mitotic activity.[31] The basal layer is responsible for regeneration of the cells in the epidermis in the repair process and for normal turnover of cells in the epidermis. On the face, regeneration results from the germinal layer and epidermal pegs.



FIGURE 21-2 A large wound created by a crush type of injury resulting in a large stellate laceration with surrounding abrasions. Initially, the wound should be thoroughly irrigated with a balanced saline solution and inspected for foreign bodies. Débridement should be limited to obviously devitalized and necrotic tissue. Excessive débridement is limited by the rich vascular supply. Scrubbing the wound should be avoided, unless an overwhelming amount of foreign material is present in the wound, because the scrubbing causes more inflammation and further injury. Next, the wound is cleansed with a nonionic detergent, and gross spillage into open areas is avoided. The wound is then ready for closure.

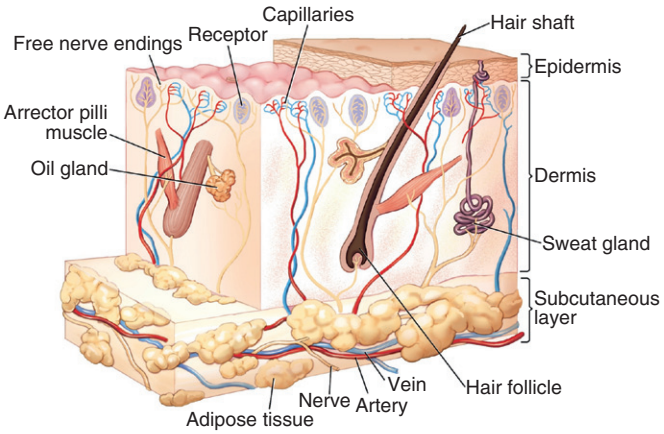


FIGURE 21-3 Cross section of the skin, which contains three layers—the epidermis, dermis, and subcutaneous connective tissue. (From VanMeter K, Hubert R: *Microbiology for the healthcare professional*, St. Louis, 2010, Mosby.)

Because of the large number of epidermal pegs on the face, a notable portion of the epidermal layer can be removed without significant scarring.[33] The stratum spinosum, or prickle cell layer, consists of polyhedral cells with ovoid nuclei. The granular layer is named for its histidine-rich cytoplasmic granules of keratohyalin, thought to be important in keratin formation.[34] Changes in the formation of the granular cell layer are seen in the development of the healing wound. The stratum lucidum is found only on the palms of the hands and soles of the feet. The stratum corneum, the outermost layer of the epidermis, is formed of keratinized flattened cells that are usually without nuclei. The corneum layer is responsible for the variable thicknesses of skin found on the body.

The dermis is divided into two layers, a superficial papillary layer and a deeper reticular layer. The papillary zone is a thin, finely textured zone immediately beneath the epidermal rete ridges. The papillary layer gives rise to the dermal papillae with fine fibrils of collagen and provides a blood supply to the avascular epidermal layer.[32] The papillary dermis and epidermis together form a functional unit that provides an important metabolic area for retaining the normal integrity of the skin.[33,34]

The reticular layer of the dermis is a thick dense mass of collagenous and elastic connective tissue fibers. Reticular fibers, which give the layer its name, are young, finely formed collagen fibers with a narrower diameter than that of mature collagen.[34] Elastic and other collagen fibers in the papillary dermal layer tend to be perpendicularly oriented to the overlying epidermal layer, and the fibers in the reticular layer are mainly oriented tangentially to the epidermal layer.[32] Collagen fibers provide the skin with tensile strength, whereas elastic fibers give the skin its elastic properties.

The orientation of the fibers in the reticular layer and their relationship to the epidermal layer create lines of tension in the skin that are greater in a plane perpendicular to the fibers of the reticular layer than in a plane parallel to these fibers. The predominant orientation of the fiber bundles in relation to the surface differs in different regions of the body. These patterns were described by Langer in 1861.[35] He punched holes in the skin of cadavers and noted the direction of the gape of the wound, indicating the line of tension. Langer's lines run parallel to the principal fiber bundles of the reticular layer and thus produce less tension on the wound margins (Fig. 21-4).

Langer's lines usually indicate the most favorable direction for surgical incisions on the skin, except in some areas of the facial region because of the close relationship between the muscles of facial expression and the skin. The most inconspicuous scars are those that fall within natural creases or wrinkle lines in the skin.[3] When the facial muscles contract, they produce tension on the skin in a direction perpendicular to that of the muscle group. Thus, favorable crease lines for surgical incisions on the face run parallel to the muscles of facial expression; they may not coincide with Langer's lines and, in some areas, such as the upper lip, may run perpendicular to them.[20]

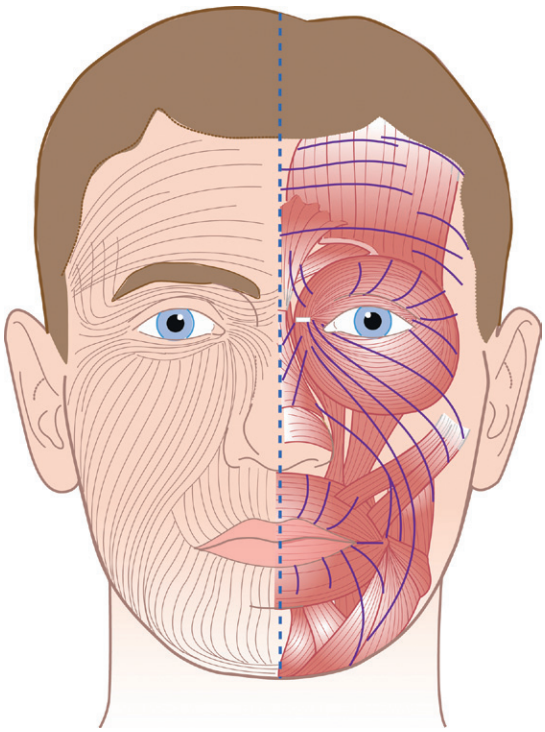


FIGURE 21-4 Langer lines. (From Kaminer M, Arndt K, Dover J, et al: *Atlas of cosmetic surgery*, ed 2, St. Louis, 2009, Saunders.)

The dermis also contains a small amount of fat, numerous blood vessels, lymphatics, nerves and sensory nerve endings, hair follicles, sweat and sebaceous glands, and smooth muscle. The dermis is supported by subcutaneous connective tissue that is thinner in the facial region than in most of the body and is nonexistent in the eyelids. The muscles of facial expression are in the subcutaneous layer and insert into the reticular layer of the dermis.[32]

LOCAL ANESTHESIA

Local anesthesia, with or without premedication, may be used in cases of less severe wounds such as small lacerations, contusions, and abrasions. A 1% lidocaine solution with 1:100,000 epinephrine or 0.5% bupivacaine (Marcaine) solution with 1:200,000 epinephrine may be injected into the wound margin. If possible, nerve blocks are helpful for minimizing the amount of solution necessary for this field block in large wounds and preventing distortion of the tissue by the anesthetic solution. For delivery of a local anesthetic, the following guidelines are recommended:

- Use a needle that is small (25 gauge or smaller).
- Insert the needle into the wound margin, as opposed to piercing the intact skin.
- Pass the needle through subcutaneous tissue.
- Inject slowly.
- Insert the needle no more than two thirds of its length to prevent complications associated with needle breakage.

Adverse reactions to local anesthetics do occur, but fortunately are rare. Allergic reactions can usually be prevented when a thorough patient history has been taken; these reactions are usually caused by the para-aminobenzoic acid (PABA) found in ester local anesthetic solutions or by the preservative methylparaben found in the amide local anesthetic solutions.[36,37] However, the vast majority of patients who claim to be allergic to local anesthetics are not truly allergic to these agents.[38,39] Many patients have been incorrectly labeled as having an allergy to local anesthetics after previously experiencing dizziness, syncope, dyspnea, localized erythema, or pruritus around the time of anesthetic administration.[40,41] Nonetheless, it is difficult to determine those who may truly be sensitive to a particular agent accurately. An alternative local anesthetic for patients who report local allergies is diphenhydramine (Benadryl). At a concentration of 1%, diphenhydramine has a slower onset but a comparable duration and produces anesthesia equivalent to 1% lidocaine, although it is somewhat more painful on injection.[42,43] Diphenhydramine local anesthetic solution is effective for small wounds and can be infiltrated or used for regional blocks. A quantity of 1 mL or 50-mg/mL diphenhydramine solution is diluted in 5 to 10 mL of normal saline to produce a 1.0% to 0.5% solution. When the facial area is involved, a 1% solution is recommended.[44] The risk of complications with the use of diphenhydramine is low, but erythema, regional edema, vesiculation, and sloughing of tissue have been reported. Although in most cases a higher concentration was used (5%), it is recommended to avoid using this agent in areas of poor collateral perfusion such as the digits, pinnae of the ears, and nose. Common adverse reactions to diphenhydramine used as a local anesthetic also include sedation, dizziness, disturbed coordination, epigastric distress, and thickened bronchial secretions. Patients should be warned against driving or other dangerous activities because of the associated sedation.

Toxic reactions to local anesthetics are more common than allergic reactions and are usually caused by accidental intravascular injection or administration of large quantities of the drug. Patients at the extremes of age are particularly at risk. Older adults can have elevated blood levels of the drug because of a decline in liver function and an increased incidence of cardiovascular disease. Overdosage is the common cause of toxicity in pediatric patients.[45] Maximum dosages of all drugs administered by injection should be calculated and should not be exceeded[46] (Tables 21-3 and 21-4).

The initial signs and symptoms of toxicity caused by local anesthetics are mediated primarily by the central nervous system. Initially, the patient may experience slurred speech, shivering, muscular twitching in the face and distal extremities, flushing of the skin, dizziness, tinnitus, and disorientation. Numbness of the tongue and perioral tissue is not mediated by the central nervous system. Rather, the paresthesia is caused by the direct anesthetic effect on the free nerve endings in this tissue. Further elevations of local anesthetic blood levels produce seizure activity and later cardiac and respiratory depression, which can lead to death.[46] The lack of understanding about maximum dosages has led to fatalities in children.[45]

TABLE 21-3 Dosages and Properties of Injectable Local Anesthetics*

Characteristic	Procaine	Lidocaine	Mepivacaine	Bupivacaine
Potency	1	2	2	8
Onset	Slow	Fast	Fast	Moderate
pK_a	8.9	7.9	7.6	8.1
Duration (min)	Short (60-90)	Moderate (90-200)	Moderate (120-240)	Long (180-600)
Protein binding (%)	6	64	78	96
MAXIMUM DOSE (MG/KG)				
Plain	7	4.5	7	2
With epinephrine	9	7	8	3

*Toxic reactions to local anesthetics are more common than allergic reactions; many are caused by accidental intravascular injection or administration of large quantities. The young and the very old are particularly at risk, so it is prudent to avoid excessive dosages and always aspirate prior to delivery.

From Webster RG, McCullough EG, Giandello PR, et al: Skin wound approximation with new absorbable suture material. *Arch Otolaryngol* 111:517, 1985.

TABLE 21-4 Maximum Dosages of Local Anesthetics

	DOSAGE		
Drug	mg/lb	mg/kg	Maximum (mg)
Lidocaine	2	4.4	300
Mepivacaine	2	4.4	300
Bupivacaine*	0.6	1.3	275
Prilocaine	2.7	6	400
Procaine	3	6.6	400

*Bupivacaine is not recommended for patients < 12 yr.

Vasoconstrictors can limit plasma levels of local anesthetics by decreasing the rate of absorption, which reduces the risk of toxic reactions. Additional benefits of vasoconstrictors include increased duration of action of local anesthesia and assisting with hemostasis at the surgical field. However, the use of vasoconstrictive drugs should be avoided or kept to a minimum in patients receiving certain medications such as beta blockers, monoamine oxidase (MAO) inhibitors, and tricyclic antidepressants, or in patients with conditions such as hyperthyroidism, elevated blood pressure (systolic blood pressure greater than 200 mm Hg, diastolic blood pressure greater than 115 mm Hg), and recent cerebrovascular accident or myocardial infarction.[46]

For the patient's comfort, it has been found that lidocaine buffered with sodium bicarbonate can decrease pain on injection. It is recommended that 9 mL of 1% lidocaine be mixed with 1 mL of sodium bicarbonate (44 mEq/50 mL) to provide a buffered solution for injection.[47]

ARTICAINE HYDROCHLORIDE (SEPTOCAINE)

Articaine is an amide local anesthetic introduced in Germany in 1976 and subsequently throughout Europe and Canada (Ultracaine D-S). Articaine (Septocaine) was introduced into the United States in 2000. Articaine is supplied for dental use in 1.8-mL cartridges as a 4% (40 mg/mL) solution combined with 1:100,000 epinephrine. The onset of action is 1 to 6 minutes, provides an average pulpal anesthesia for 45 to 60 minutes, and is active in soft tissue for approximately 2 to 5 hours. The administered dose is excreted in the urine within 24 hours. The maximum dose is 7 mg/kg or 3.2 mg/lb.

One advantage of the use of articaine anesthesia in the oral cavity is that the articaine formulation may possibly spread through hard tissue more effectively than other local anesthetics and provide infiltration anesthesia as effective as nerve block techniques. Some have claimed that buccal infiltration of articaine adjacent to a maxillary or mandibular premolar provides adequate anesthesia to remove the tooth without a palatal injection. Further investigations were carried out to evaluate the use of buccal infiltration of articaine and the elimination of palatal anesthesia injections for the routine forceps removal of teeth.[48]

The pharmacologic and toxic effects associated with articaine are qualitatively similar to those of other amide local anesthetics. Articaine has been associated with methemoglobinemia after IV regional anesthesia, but no reports have been published about methemoglobinemia after injection for dental anesthesia.[49] In a study by Malamed et al,[50] 4% articaine with 1:100,000 epinephrine was compared with 2% lidocaine with 1:100,000 epinephrine. Similar effectiveness and similar rates of adverse events were noted between the two local anesthetics, but a 0.9% incidence of paresthesia in a total of 882 patients was found. Long-term paresthesia, especially of the lingual nerve during inferior alveolar nerve blocks, is a growing concern with the use of this anesthetic for routine dental procedures.[51,52] One may choose to use articaine primarily for infiltration techniques and with caution for nerve blocks.

TOPICAL AGENTS

A combination of tetracaine (0.5%), epinephrine (1:2000), and cocaine (11.8%; TAC), is available for use as a topical anesthetic agent.[53-55] In wounds of the scalp and face, the degree of anesthesia is comparable with that of local infiltration with lidocaine. TAC (0.09 mL/kg) is applied to gauze or cotton balls and held in contact with the wound margin for 5 to 10 minutes or until visible blanching occurs, signaling the onset of adequate anesthesia. TAC is rapidly absorbed through the mucous membranes, eyes, and burned or denuded skin and should not come into contact with these surfaces or the patient may be at risk for severe systemic toxicity or even death. It should also be avoided in areas of end-arterial flow (e.g., the digits, tip of the nose, and pinna) because of its intense vasoconstriction. TAC is used primarily for small wounds, such as simple lacerations, and is most popular in the treatment of pediatric patients.

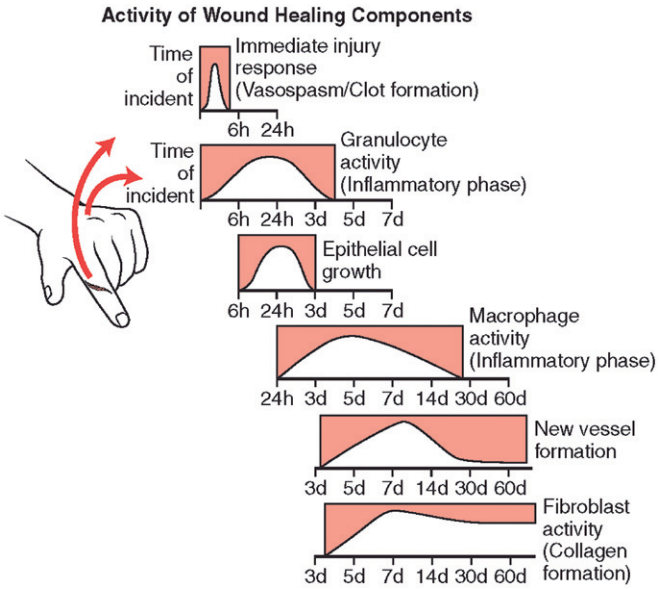


FIGURE 21-5 The chronology of early wound healing. (Adapted from Trott A: Surface injury and wound healing. In *Wounds and lacerations: Emergency care and closure*, ed 2, St. Louis, 1990, Mosby.)

When treatment is provided in the outpatient clinic with the patient under local anesthesia, the same care and diligence should be applied in treating soft tissue wounds as is practiced in the operating room when the patient is under general anesthesia.

SUTURE MATERIAL

The primary purpose for using sutures is to approximate wound margins and enhance tissue healing. To understand the interaction of suture materials with biologic tissue, an appreciation of the wound-healing process is important (Fig. 21-5). Wound healing can be divided into three phases. During the initial lag phase (up to day 5), there is no gain in wound strength and the wound is dependent on sutures and epidermal cellular adhesion to maintain closure. During the fibroblastic phase (days 5 to 15), a rapid increase in wound strength occurs. The maturation phase (day 14 and beyond) is characterized by further connective tissue remodeling. By the end of the second week, when most skin sutures are removed, only 3% to 7% of the final tensile strength has been achieved. By the end of the third week, 20% of the tensile strength is attained and, at the end of the first month, 50% is present. Wounds never regain more than 80% of the strength of intact skin.[56-60] Because the tissue reduces suture strength over time, the relative rates at which the suture material loses strength and the wound gains strength are important. Wounds do not gain strength until 4 to 6 days after injury, so the entire burden of approximating the tissue during this period rests on the sutures.[61] Thus, tensile strength, the amount of tension or pull the suture can withstand before breaking, is an indispensable property of suture material. Tensile strength is proportional to the diameter of the suture. For example, if the diameter of the suture is doubled, the tensile strength quadruples. In addition, a knotted suture has only about one third the tensile strength of an unknotted suture, although this relationship varies with the type of knot and the suture material used.[62]

Suture materials should be at least as strong as the tissue in which they are used. A wide variety of materials—silk, linen, cotton, horsehair, animal tendons and intestines, and wire made of precious metals—have been used for wound closure. The evolution of suture materials has provided a number of options when selecting the correct suture for a particular procedure. The selection of suture material is based on the condition of the wound, tissue to be repaired, strength and knot-holding characteristics of the suture material, and reaction of the surrounding tissue to the suture material.[3,17] Suture material can be classified as absorbable or nonabsorbable, coated or uncoated, natural or synthetic, and multifilament (braided) or monofilament. Synthetic, nonabsorbable sutures include Dacron (Mersilene, Polydek, Tevdek, Ethibond, and Tycron), nylon (Ethilon and Dermalon), and polypropylene (Prolene and Surgilene). Natural, nonabsorbable sutures include silk, cotton, and metals such as stainless steel, tantalum, and titanium. Natural absorbable sutures are made of catgut and plain and chromic collagen. Synthetic absorbable sutures include polyglycolic acid (Dexon), polyglactic acid (Vicryl), and polydioxanone (PDS).[63]

Monofilament sutures are made of a single strand of material. They encounter less resistance when passing through tissue than multifilament sutures and resist harboring microbial organisms. The handling properties of monofilament sutures are good, but care must be taken not to crimp or nick the material because breakage is likely. Multifilament sutures consist of several strands of material braided or wound together, which increases flexibility and tensile strength.[61,64] Multifilament suture materials have been reported to cause more reactions in oral tissue compared with monofilament sutures. This increased the incidence of reactions that may be caused by permeation of the multifilament suture materials by oral bacteria. Silicone and Teflon coating of multifilament sutures does not seem to reduce bacterial invasion of tissue.[65-69]

Measurement of the in vivo degradation of sutures provides a general classification of surgical sutures. Sutures that undergo degradation rapidly and lose their tensile strength within 60 days are termed *absorbable*. Those that maintain their tensile strength for longer than 60 days are termed *nonabsorbable*.[64,70]

Absorbable sutures are used to hold wound edges together temporarily. The suture material is degraded by enzymatic processes, as in gut sutures, or by hydrolysis, as in many of the synthetic materials. Although absorbable sutures have advantages, they respond to the environment into which they are placed. In infected tissue or in a patient who is febrile or protein-deficient, suture breakdown may be accelerated. If the loss of tensile strength outpaces the healing phase of the wound, failure may result. It is important to note that when using absorbable sutures, one must place them well into the dermis to facilitate their hydrolysis and subsequent absorption.

If they are placed in the papillary dermis, they will not hydrolyze as rapidly and may persist for weeks or months longer than anticipated.

ABSORBABLE SUTURES

Surgical Gut Sutures

A surgical gut suture is a twisted suture of proteinaceous sheep or beef intestinal wall that is approximately 98% highly purified collagen strands. The tensile strength of gut material is good, but absorption is by phagocytosis, which is unpredictable and results in gradual loss of strength. The suture will become wiry if allowed to dry out.

Plain gut suture is rapidly absorbed, maintaining tensile strength for only 7 to 10 days, and is completely absorbed within 70 days. Plain gut suture can also be heat treated to form fast-absorbing gut suture, which is used in epidermal suturing when support is necessary for only 5 to 7 days.[70,71] Lister was the first to sterilize sutures and also introduced the treatment of catgut suture with chromic acid to slow its rate of absorption.[72] To minimize tissue reaction, increase tensile strength, and slow the absorption rate of macrophage activity, catgut can be coated with a thin layer of chromium salt solution, which resists enzymatic degradation by the tissue and thus increases tensile strength and prolongs absorption time to longer than 80 days.[73] In noncontaminated wounds, chromic gut sutures minimize tissue reaction, causing less irritation than plain gut in the early stages of wound healing. Tensile strength is retained for 10 to 14 days. It should also be noted that when placed into contaminated tissue, plain gut sutures elicit less infective response than chromic gut sutures.[58] A mild chromic gut suture is also manufactured that is absorbed rapidly (50% in 3 to 5 days) and is used primarily in ophthalmologic surgery.[62] The advantages of chromic catgut materials include absorbability, tensile strength, and knotting qualities. The disadvantages include the wide range of biologic variability in loss of tensile strength over time and a broad range of reactions to these materials in individual patients.[63]

A thin chromic catgut suture has been used for closure of the epidermal layer in facial wounds. The 6-0 catgut material (Davis-Geck 6-0 mild chromic, or Ethicon 6-0 rapidly absorbing gut) is absorbed within 3 to 5 days and does not have to be removed. The material may be used with sterile strips to relieve surface wound tension.[3,74]

Glycolic Acid Homopolymer (Dexon)

This suture, composed of a polymer of glycolic acid, is characterized by a greater knot pull and tensile strength than those of gut. It was introduced in 1970 as the first synthetic absorbable suture.[75] Like Vicryl (polyglactin 910), polyglycolic acid is absorbed primarily by hydrolysis, which results in minimal tissue reactivity. However, because polyglycolic acid has been shown to persist longer in the wound, it generates more tissue reaction than Vicryl, but less than plain gut or chromic gut.[76] Polyglycolic acid suture is braided and often catches on itself, making knot tying and passage through tissue difficult. The suture does not tolerate wound infections well and should not be placed at an infected site. It is also not recommended to use polyglycolic acid suture percutaneously, but this suture is effective in deeper tissue layers.

Glycolic Acid (Maxon)

Maxon is a monofilament strand composed of polyglycolic acid and trimethylene carbonate. Polyglycolic acid suture, along with PDS suture, offers the greatest tensile strength of any type of resorbable suture. The suture retains 70% of its tensile strength at 14 days and 55% at 21 days.[62] This period of tensile strength is much longer than that of the chain polymer form of glycolic acid (Dexon). Complete absorption is accomplished by hydrolysis in 180 days. In vitro studies have suggested that the suspected degradation products of polyglycolic acid and nylon sutures are potent antibacterial agents. These byproducts—glycolic acid, 1,6-hexane diamine, and adipic acid—have shown a marked reduction in bacterial counts when incubated with *Staphylococcus aureus*.[64]

Polyglactin 910 (Vicryl)

This commonly used synthetic suture is composed of a mixture of lactide and glycolide acids and calcium stearate produced in a braided configuration that improves handling properties. The lactide component has hydrophobic qualities. This water-repelling property slows the penetration of water into the suture filaments, thus delaying the loss of tensile strength. Approximately 65% of its tensile strength is retained at 14 days and 40% at 21 days.[77] The suture is degraded by hydrolysis and absorption is complete after 56 to 70 days, with byproducts excreted primarily in the urine. Vicryl is coated with a second type of polyglactin (polyglactin 370) and calcium stearate, which allows for easy passage through tissue and easier knot placement. This suture should be buried in the subcutaneous tissue or in deeper layers. When used in these locations, Vicryl has minimal tissue reactivity and is appropriate if the tissue is infected. However, percutaneous placement is not recommended. When used to close skin wounds, Vicryl is associated with delayed absorption and increased inflammation. Occasionally, the suture is extruded without inflammation, resulting in a small nodule in the suture line. Although this type of suture is available purple and undyed, only the colorless type should be used on the face to prevent showing through thin skin.[78]

Irradiated Polyglactin 910 (Vicryl Rapide)

Vicryl Rapide is irradiated polyglactin 910. It is a braided copolymer that is surface-treated with polyglactin 370 and calcium stearate and has been gamma irradiated. This radiation alters the suture material's molecular structure and enhances its absorption rate. The suture is indicated for short-term wound support for superficial closure, providing stability of the wound for 7 to 10 days. The suture is absorbed over 12 to 14 days and does not require removal. Microscopically, the suture is absorbed primarily by phagocytosis by day 35.[79,80] The degree of inflammation is less than that observed with plain or chromic catgut suture. However, Vicryl Rapide is not recommended for use on facial skin.[81] The suture is slightly brittle but requires little adjustment to normal

suturing techniques. From an economic standpoint, Vicryl Rapide is at most 10% more expensive than similarly packaged gut or chromic gut sutures. Polytetrafluoroethylene (PTFE) sutures are at least four times more expensive.

Vicryl Plus (Polyglactin 910 Coated with Triclosan)

In December 2002, the U.S. Food and Drug Administration (FDA) approved Vicryl Plus antibacterial suture. Designed to reduce bacterial colonization on the suture, this was the first and only suture designed with an antibacterial agent. The agent, triclosan, has been shown to be effective against *S. aureus, Staphylococcus epidermidis,* and methicillin-resistant strains of *Staphylococcus* (MRSA and MRSE), which are the leading surgical site bacteria.[82] There appears to be no adverse effect on wound healing.[83] The addition of triclosan also appears to have no effect on the strength, healing of wounds, handling characteristics, or performance when compared with commonly used polyglactin (Vicryl) sutures.[84]

Polydioxanone (PDS II)

This synthetic monofilament suture is made from the polyester derivative poly-*p*-dioxanone. PDS suture has excellent tensile strength qualities and retains 70% of its original tensile strength at 14 days, 50% at 28 days, and 25% at 42 days.[85] The suture passes through tissue easily, but has significant memory, which compromises the ease of knot tying and knot security. Tissue reaction to the material is minimal, but there is a tendency for the PDS suture to extrude through the wound over time. Because of this tendency, it is recommended that this suture material be used only in tissue layers deeper than the subcuticular layer[3] or be used, in a 6-0 size, for the closure of the epidermal layer in the face.[13] PDS is commonly used in wounds under tension and is appropriate in contaminated tissue.[86] Like Vicryl, PDS II is also degraded by hydrolysis. Absorption is minimum until day 90 and is complete after approximately 6 months, with minimal tissue reaction.

NONABSORBABLE SUTURES

Nonabsorbable sutures are categorized by the *U.S. Pharmacopeia* (USP)[61] as follows:

- Class I: Silk or synthetic fibers of monofilaments with twisted or braided construction
- Class II: Cotton or linen fibers, coated natural or synthetic fibers in which the coating does not contribute to tensile strength
- Class III: Metal wire of monofilament or multifilament construction

Nylon

Braided (Surgilon, Nurilon). This suture is a synthetic nonabsorbable material composed of an inert polyamide polymer. The nylon fibers are braided and then sealed with a silicone coating. Nylon has excellent knot security, tensile strength, and knot pull strength, and little tissue reactivity. The buried suture loses approximately 20% of its tensile strength yearly through hydrolysis.[62] Nylon-braided sutures look, feel, and handle like silk but are stronger and do not have the increased tissue response associated with silk sutures. Multifilament nylon is weaker and less secure when knotted, offering little advantage over monofilament nylon.[87]

Monofilament (Dermalon, Ethilon). The monofilament nylon suture has characteristics similar to those of the braided form of nylon suture, although it is uncoated. Monofilament nylon suture is relatively inert and nonirritating to tissue, with smooth passage through the tissue. Nylon sutures are well suited for retention and skin closure because of their elastic nature. Nylon is widely used because of its favorable qualities, such as high tensile strength and low tissue reactivity. The sutures degrade at a rate of 15% to 20% per year by hydrolysis. They have some memory and will tend to return to their original linear shape over time. Because of this tendency, more throws in the knot are indicated to securely approximate the tissue during healing, even compared with braided nylon sutures.[62] Moistened nylon-monofilament sutures are more easily handled, and some types are packaged wet for use in plastic surgery procedures. A careful four-throw knot usually is sufficient.

Polyester: Braided (Tycron, Mersilene, Uncoated, Dacron, Ethibond, Coated)

Polyester sutures are constructed from multifilament fibers of polyester or polyethylene terephthalate. The polyester suture has excellent tensile strength, which is maintained indefinitely.[62] Mersilene is uncoated, is somewhat rougher and stiffer than the coated form, and has a significant amount of drag when passed through the tissue. Ethibond is a braided polyester suture that is coated with polybutilate, which provides a low infection rate, secure knot tying, smooth removal, low reactivity, and easy passage through the tissue. Ethibond is an excellent suture for skin surgery; however, it is more expensive than other sutures with similar indications for use. Polyester-braided sutures are stronger than nylon or polypropylene sutures but have an increased risk of contamination and therefore are not generally used for skin closure. When used in deeper layers, the polyester suture has been shown to last indefinitely.[88]

Surgical Cotton

Surgical cotton is the weakest nonabsorbable suture and, as the name implies, is composed of long stable cotton fibers. Cotton suture has good knot security but is associated with high tissue drag and reactivity and has been shown to produce a marked tissue reaction.[89] Surgical cotton sutures are unsuitable for use in contaminated wounds or in the presence of infection. These undesirable qualities and the fact that newer synthetic sutures provide superior performance have resulted in cotton sutures being rarely used in surgical procedures today.

Stainless Steel

Stainless steel sutures are monofilament strands of ferrous alloy that have desirable characteristics of strength and low tissue reactivity. However, this suture has the potential to corrode or break at points of twisting, bending, or knotting.[90] Stainless steel suture is hard to tie and the knot ends require special handling. Both

monofilament and twisted or braided multifilament stainless steel sutures are available. Stainless steel sutures offer the greatest amount of tensile strength, even in the presence of infected tissue, and are the most inert of all suture material. The difficulty in handling of stainless steel suture and its tendency to cut through tissue make it unpopular for cutaneous surgery, but it may be used successfully for closure of deeper layers that are infected.[62]

Silk

Silk sutures are braided, siliconized, proteinaceous thread spun of silkworm larval cocoons. Each silk filament is processed to remove the natural waxes and sericin gum. After braiding, the strands are dyed, stretched, and impregnated with a mixture of waxes and silicone. Silk sutures provide good knot security. Dry suture is stronger than wet silk suture but is not as strong as comparable sizes of synthetic materials. Silk sutures should not be used in the presence of infection.[62,87] Silk sutures represent the historical and material standard of performance against which newer types of suture materials are judged, although silk sutures offer little advantage over modern synthetic materials. Silk sutures may be braided or twisted; the braided form has better handling characteristics. Silk suture is useful in the periocular area, intraorally, and on other mucosal surfaces because it remains soft and pliable and does not easily cut through tissue.[3,63] Although characterized as a nonabsorbable material, studies have shown that silk sutures lose most of their tensile strength after 1 year and cannot be detected in tissue after 2 years.[91]

Polypropylene (Prolene)

Polypropylene suture is an isostatic crystalline stereoisomer of a linear hydrocarbon polymer permitting little or no saturation. The material is extremely inert and will retain its tensile strength for at least 2 years.[92] Polypropylene suture holds knots better than most other synthetic monofilament sutures and is indicated for use when minimal suture reaction is necessary, such as infected tissue and contaminated wounds. This suture does not adhere to tissue and is flexible, and thus is useful for pull-out types of sutures. Simple knots are ineffective, but carefully tied four-throw knots will provide adequate security.[87]

Polybutester (Novofil)

Polybutester is a monofilament, nonabsorbable suture made of polyglycol terephthalate and polybutylene terephthalate and is considered to be a modified polyester suture.[62] In contrast to polypropylene and nylon, this suture does not have significant memory, is easier to manipulate, and has greater knot security. A unique feature of polybutester sutures is their capacity to elongate or stretch with increasing wound edema. After tissue edema has subsided, the suture resumes its original shape, which theoretically makes polybutester an ideal suture material for lacerations secondary to blunt trauma.[93] The tensile strength of Novofil is high and lasts for an extended period of time. Novofil has minimal tissue reactivity as well. The popularity of this suture in cutaneous surgery has been gradually increasing.

It is important to note that even the least reactive nonabsorbable suture, nylon, tends to elicit some degree of infection in tissue contaminated with *Escherichia coli* or *S. aureus* bacteria. The incidence of gross infection in contaminated tissue containing nylon sutures has been shown to be significantly greater than the infection rate in contaminated needle puncture tracks not containing suture.[64] These results suggest that sutures should be avoided or the number of sutures minimized in infected tissue, whenever possible.

SURGICAL TAPE

Microporous tape for wound closure is useful alone or in conjunction with subcutaneous or skin sutures to decrease tension at the wound margin. Skin tape comes in ⅛-, ¼-, and ½-inch wide strips that may be reinforced with rayon filaments to increase the tensile strength of the strips. The skin margin is prepared with tincture of benzoin to provide better adhesiveness for the tape. The tape should be placed perpendicular to the wound on one skin side first; the wound margins are then pulled together with the fingers or by an assistant, and the tape is secured to the skin on the other side of the wound. Thus, tension over the wound is diminished. Before placement of the tape, a thin coat of antibiotic ointment may be placed along the wound margin to protect the wound from skin oils and bacteria. To remove the adhesive tape and prevent separation of the epithelial margins, the ends should be lifted equally toward the wound margin and then lifted evenly from the wound.[3]

SURGICAL NEEDLES

One of the earliest descriptions of needles used for surgical purposes appears in the Edwin Smith Surgical Papyrus, written approximately 3000 to 2500 BC. The twisted, or "harelip," suture in which a needle was inserted on either side of the defect and the suture material was intertwined about the needle in a figure-eight fashion (Fig. 21-6) has been described in surgical texts published in the late 1800s, although it has commonly been assumed to be a surgical technique used in ancient times.[94]

Needles today are manufactured from stainless steel wire, which initially is soft and then submitted to varying heat-treating techniques to provide strength and other desirable characteristics, such as temper, hardness, malleability, and sharpness. The needles can be shaped or milled into the various types commonly used today (Fig. 21-7). Needles may be eyed or swaged. Eyed needles require threading of the suture material before use, which results in pulling a double strand of suture material through the wound and an increased risk of losing the needle in the tissue. Tying the suture to the eye is not recommended because it increases the bulk of suture material drawn through the tissue. Swaged needles do not require threading and permit a single strand of suture material to be drawn through tissue. A new and undamaged needle is provided with each strand of suture, allowing for less trauma when passed through the tissue.[94,95]

One common type of suture needle used in closing facial wounds is the reverse cutting needle (Table 21-5).

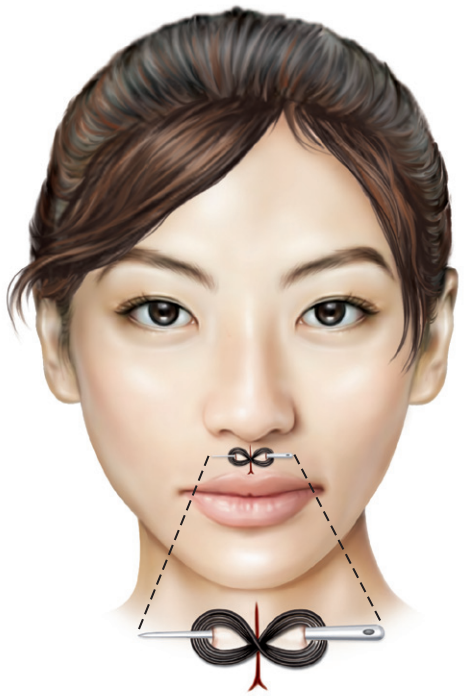


FIGURE 21-6 The twisted, or harelip, suture, in which suture material was used in a figure-eight fashion around a needle passed through a lip defect.

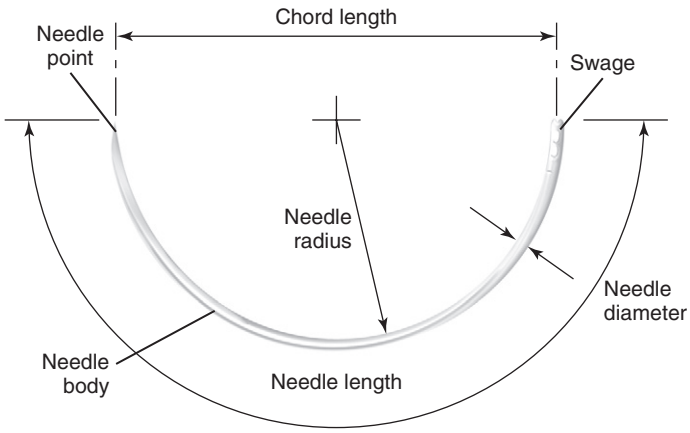


FIGURE 21-7 Anatomy of a surgical needle.

The reverse curved cutting needle has a cutting edge along the convex surface, rather than along the concave surface, as in the conventional cutting needle. There is less likelihood that the needle will cut considerable tissue in its path through the tissue, and thus its use will prevent needless tissue damage and wound enlargement. It has been calculated that the strength of the needle in the reverse cutting shape is increased 32%. The increased strength of the needle makes possible the use of a smaller diameter wire, resulting in a smaller wound produced by the needle.[62,94]

The most commonly used curved needles are three-eighths circle needles. These needles are easily manipulated in relatively large and superficial wounds, because their introduction, passage, and withdrawal require more space than the one-half curved needles and less wrist movement. Half-circular needles are recommended for suturing tissue in small wounds, deep wounds, and body cavities and orifices. They require less space relative to needle size because of their greater length, even though more pronation and supination of the wrist are necessary for passage of the needle. The most extreme are the five-eighths circle needles, which may be selected for use in confined areas, such as the oral cavity.

The two basic varieties of needles are cuticular and plastic. Cuticular needles are sharpened 12 times, whereas plastic needles are sharpened an additional 24 times and designed to produce less trauma during penetration of tissue for cosmetic closures. Needle terminology, created by manufacturers, has developed in a haphazard manner; Ethicon currently supplies approximately 80% of the suture market. Cuticular needles may be designated as C (cuticular) or FS (for skin). Plastic-type needles may be labeled as P (for premium or plastic) or PS (plastic surgery), and PC needles are coded for precision cosmetic use. Needles in the PC series are made of a stronger stainless steel alloy and have modified, flattened, and conventional cutting profiles. A number is used to indicate the size of the needle in the various manufacturers' series. The larger the number, the smaller the needle size in that specific series.[25]

WOUND CLOSURE

With repair of facial wounds, the following guidelines should be followed:

1. The injured tissue should be handled gently and minimally débrided to ensure an adequately clean bed.
2. Complete hemostasis must be obtained.
3. Incisions should be placed to follow tension lines and the natural folds of the skin.
4. The skin margins must be relaxed without tension.
5. Fine sutures should be used and removed as early as possible.
6. The wound edges should be everted.
7. Dead space must be obliterated.
8. The tissue should be closed in layers.
9. The scar tissue must be allowed to mature before revision procedures.[96,97]

Tissue is composed of multiple elements that are combined to produce properties related to its functions. Parameters used to characterize tissue properties are tensile strength, breaking strength, and burst strength. Tensile strength is measured as the load per unit cross-sectional area at the point of rupture. The tensile strength of the tissue is related to the nature of the tissue, rather than to tissue thickness. Although the skin and fascia are the strongest tissues in the body, they regain their tensile strength during slow healing compared with other tissue. Breaking strength is the load necessary to break a wound regardless of its thickness. The size, age, and weight of the patient, the thickness of tissue, and presence of infection or edema all affect strength characteristics.

With the closure of facial wounds, minimal tissue trauma during repair is important to prevent excessive scarring of the wound margins. Meticulous attention to detail is necessary to minimize tissue injury and prevent further scarring from poor suturing techniques. Skin

TABLE 21-5 Characteristics of Surgical Needles Commonly Used in Skin Closure

Needle Series	Type of Cutting Edge	Alloy	Point	Body	
FS	Reverse	420	▼	▼	Triangular to swage
P Prime	Reverse	Ethalloy	▼	■	Square-bodied
PS Prime	Reverse	Ethalloy	▼	■	Square-bodied
PC Prime	Conventional	Ethalloy	▲	■	Square-bodied

FS, For skin; *P*, plastic; *PC*, precision cosmetic; *PS*, plastic surgery.
Adapted from from Ethicon: *Wound closure manual*, Somerville, NJ, 1994, Ethicon, pp. 74-75.

hooks should be used for retraction and stabilization of tissue during débridement and repair and, when tissue forceps must be used, only those with multiple fine teeth, such as Adson-Brown forceps, should be used. Wound edges should be grasped only at the level of the subcutaneous tissue to prevent puncture marks on the skin surface.[31] The wound margins should be undermined slightly to prevent undue tension on the wound margins and permit closure of the wound in layers with subcutaneous tissue and eversion of the wound margin.[21] Unless necessary to elevate rotational flaps, excessive undermining of the facial tissue should be avoided to prevent unnecessary scarring and distortion of adjacent features, such as the ala of the nose, commissure of the mouth, and eyelid. Only sharp blades and scissors should be used for débridement and preparation of wound margins. Appropriate suture material on an atraumatic cutting needle is desirable for repairing facial wounds. The sutures should be placed to allow slight elevation of the wound margin, and with the tying of surgical knots it is important to remember to "approximate, not strangulate."[19]

The principles of knot tying include the following:

1. Using the simplest knot that will prevent slippage
2. Tying the knot as small as possible and cutting the ends of the suture as short as reasonable to minimize foreign body reaction
3. Avoiding friction as the suture passes through the tissue ("sawing" of the tissue inevitably results in further trauma to the wound)
4. Preventing damage to the suture material that may compromise the integrity of the tied suture
5. Avoiding excessive tension that may break sutures or cut tissue
6. Approximation of the tissue-tying sutures too tightly strangulates the tissue
7. Maintaining traction at one end of the suture after the first loop is thrown to prevent loosening of the knot
8. Placing the final throw as horizontally as possible to keep the knot flat
9. Limiting extra throws to the knot, because they do not add strength to a properly tied knot

Wound closure should follow examination, débridement, and preparation of the wound margins (Fig. 21-8), if indicated, to allow meticulous alignment of the tissue. Key landmarks, such as the eyebrows, mucosal margins of the lip and nose, eyelids, and other anatomic structures, must be aligned and repaired properly. Key sutures placed to approximate these landmarks before closure of the remaining wound margins will assist in proper orientation (Fig. 21-9). Irregularities in the wound should be noted and approximated. Straight line portions of the laceration may then be closed, with the first suture placed to bisect the wound into equal sections and subsequent sutures placed in a similar fashion to provide even closure and prevent creation of so-called dog ears at the end of the wound. If dog ears develop, the sutures should be removed and closure should be attempted again; a skin hook can be inserted in the end of the wound, the tissue can be elevated, and redundant tissue can be incised around the base on one side of the wound margin.[98] Every attempt should be made to accomplish closure without the necessity for removing tissue because of dog ears.

Wounds in the facial region should be repaired in layers to provide anatomic alignment and prevent dead space (Fig. 21-10). All deep lacerations must be inspected carefully. Divided muscles should be reapproximated. When muscle is severed, layered closure is essential; otherwise, the muscle will retract, with a hematoma filling the gap and eventually organizing to form a depressed scar. Deep layers should be approximated with 3-0 or 4-0 absorbable sutures and the skin should be repaired with 5-0 or 6-0 suture.

If the muscular layer is involved, the tissue should be approximated with absorbable sutures tied lightly to prevent crushing of the muscle.[5] Subcutaneous sutures placed in the subcutaneous layer and reticular dermal layer are useful for closing dead space, minimizing wound tension at the skin level, and assisting with eversion of the wound margins. Slight eversion of the wound margin is desirable to produce a scar that will be level with the adjacent skin after scar contraction is complete.[21,31] It is important that the knot on the subcutaneous suture be inverted, or buried, so that the knot does not lie between the skin margin and cause inflammation or infection. To bury the knot, the first pass of the needle should be from within the wound and through the lower portion of the dermal layer. The needle should then be passed through the dermal layer at approximately the same level in the opposite wound margin and should emerge through the subcutaneous tissue again at a level similar to that of the subcutaneous suture of the opposing wound margin. If the sutures are placed at different levels, the wound margins may not be level at the skin and may produce an unacceptable scar. Suturing that is not level in the subcutaneous layers may be indicated to level off an oblique wound through the tissue. The suture

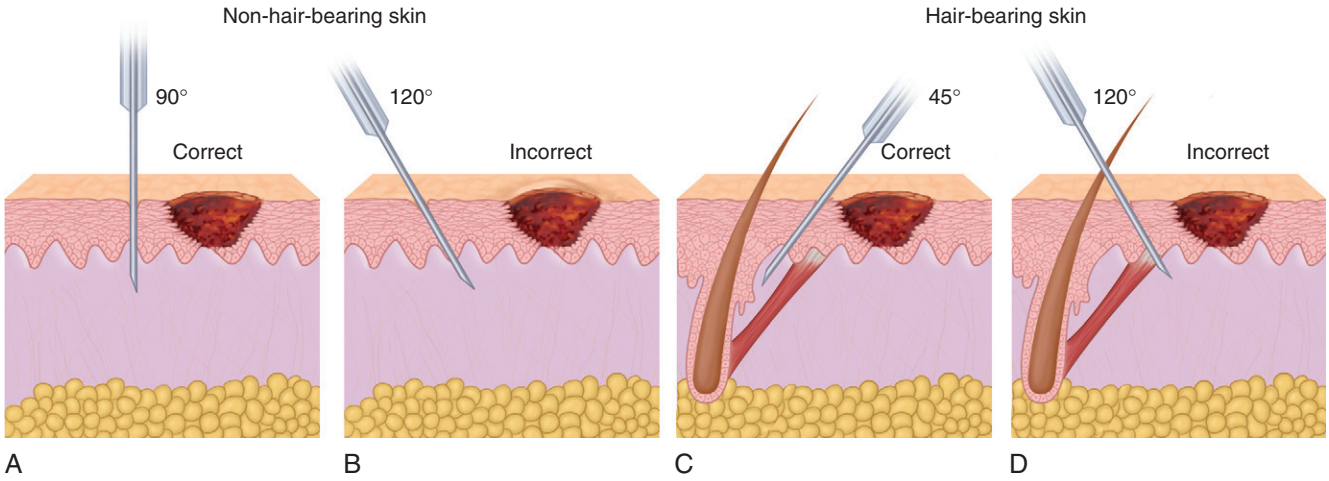


FIGURE 21-8 The angle of the blade with reference to the non–hair-bearing skin should be 90 degrees. If this angle is exceeded, the wound edge becomes beveled and interferes with wound closure. In hair-bearing skin, the blade is angled approximately 45 degrees parallel to the path of the hair follicle.

should be tied and the skin wound margins approximated under minimum tension.

Skin sutures are placed in an interrupted fashion or as a continuous subcuticular pull-out suture. Interrupted sutures should be of 5-0 or 6-0 strength, and nylon, polypropylene (Prolene), and polydioxanone (PDS) suture materials are indicated, but occasionally 6-0 chromic gut suture may be used. Sutures should be placed close to the wound margin and close enough to each other to relieve all wound margin tension.[21] Excessive numbers of sutures are unnecessary. The needle should enter the tissue at a 90-degree angle to the skin surface, approximately 2 mm from the wound margin. The needle should then be passed into the wound by rotation of the wrist along the arc of the needle and pass through the dermal layer to assist in eversion of the wound margin. The needle should then be passed through the tissue of the opposite wound margin at the same level in the dermal layer and should exit the skin at the same distance from the wound margin as that of the insertion.[19,31] The suture should be tied without undue tension to prevent suture marks. The two most common reasons for suture scars are closure under tension and delayed removal.

In lacerations without extensive tissue loss, meticulous attention should be paid to hair and eyebrow alignment, wrinkle continuity, and orientation of muscle movements to produce unobtrusive scars and restore normal anatomic function[99] (Fig. 21-11). Blanching of the skin indicates that the knot is too tight. Uneven wound margins with closure indicate that too deep a bite of subcutaneous tissue was enclosed in the suture. Scalloped edges with open wound margins between sutures are the result of bites of tissue that are too small.[31] A thin line of antibiotic ointment may be placed over the wound and adhesive strips placed over the sutures to minimize tension around the wound margins. One technique involves the placement of adhesive strips between sutures to allow for taking out the sutures without the necessity for removing the plastic strips.[100] The eventual width of the scar is proportional to the amount of tension necessary for closure.

Skin wounds regain tensile strength slowly. However, it is recommended that skin sutures be removed between 3 and 10 days, when the wound has gained only 5% to 10% of its final tensile strength, with most of the tension forces absorbed by the fascia, which holds the wound closed. Skin sutures in the face should be removed 4 to 6 days after placement. Sutures in thin-skinned areas, such as the eyelid, should be removed 3 to 5 days following placement.[13] Alternate sutures can be removed beginning at day 4 and the wound should be supported by adhesive strips. The remaining sutures may be removed 2 days later. Suture marks are usually caused by three factors: (1) skin sutures left in place longer than 7 days, resulting in the epithelialization of the suture track; (2) tissue necrosis from sutures that were tied too tightly or became tight from tissue edema; and (3) the use of reactive sutures in the skin.[5] Interrupted skin sutures should not be used in patients who are subject to hypertrophic scars.[101]

The continuous subcuticular suture is good for approximation of the skin margins and can be left in place for 3 to 4 weeks, without the formation of suture tracks.[21] Polypropylene or nylon synthetic monofilament materials of 4-0 strength are used for the subcutaneous suture. After absorbable sutures are placed to close deep tissue, the needle is passed through the skin 5 to 10 mm from the wound edge into the wound at the dermal layer. A small hemostat should be placed on the free end of the suture. The needle is passed back and forth through the dermal layer at the same level in the dermis on opposite margins of the wound and parallel to the epithelial layer. Skin hooks are useful for manipulating the wound margin during placement of the needle and for orienting the tissue for proper placement of the suture, which is critical for level approximation of the wound margins. It is also helpful to have an assistant follow, or keep a slight amount of tension on, the suture material already placed in the wound to facilitate proper orientation of the suture in the dermal layers. Removal of the suture in long wounds will be made easier if the suture is brought out through the epithelium near the



FIGURE 21-9 **A, B,** Wound closure should follow examination, débridement, and preparation of the wound margins to allow meticulous alignment of the tissue. Relevant landmarks, such as the eyebrows, mucosal margins of the lip and nose, eyelids, and other anatomic structures, must be aligned and repaired properly. Key sutures placed to approximate these landmarks before closure of the remaining wound margins will assist in proper orientation. Irregularities in the wound should be noted and approximated. Wounds in the facial region should be repaired in layers to provide anatomic alignment and prevent dead space. When muscle is severed, layered closure is essential; otherwise, the muscle will retract, with a hematoma filling the gap and eventually organizing to form a depressed scar. Deep layers should be approximated with 3-0 or 4-0 absorbable sutures and the skin should be repaired with 5-0 or 6-0 sutures. **C,** 6 months following repair of the facial injuries. Slight eversion of the wound margin is desirable to produce a scar that will be level with the adjacent skin after scar contraction is complete.

wound margin and is reinserted at the same level through the epithelium. The suture is continued through the dermal layer to the end of the wound. At the end of the wound, the needle is passed out through the skin 5 to 10 mm from the margin. The ends of the suture can be tied in a knot above the skin or secured to the skin with adhesive strips. A thin line of antibiotic ointment should be placed at the wound margin and adhesive strips should be used to relieve tension at the margin. If areas in proximity to the wound margin are not level on closure of the wound with continuous subcuticular sutures, interrupted sutures or adhesive strips can be placed to level the margin. The suture is removed by cutting one free end of the suture at the skin level or by cutting the epithelial loop and pulling the suture out the free end. As the suture is removed, support should



FIGURE 21-10 **A,** Careful examination of the facial injuries should include clinical and radiographic evaluation of the underlying facial bones for possible fracture. Repair of the soft tissue injury should be delayed until the facial fractures have been repaired. This patient has a nasal bone fracture associated with a complicated soft tissue injury that involves the nose, eyebrow, and scalp. **B, C,** The soft tissue would provide access to the fracture site; the fractured bones are stabilized with plate fixation. **D,** The soft tissue injury is repaired in layers with interrupted sutures to reapproximate the eyebrow and facial structures.



FIGURE 21-11 **A,** A scalp injury associated with a motor vehicle accident should first be débrided of all foreign material, without removal of tissue. Small pieces of tissue should be saved, as the blood supplied tends to be maintained and will heal. **B,** 5-0 or 6-0 skin sutures should be placed in an interrupted fashion. Nylon, polypropylene (Prolene), and polydioxanone (PDS) suture materials are indicated, but occasionally 6-0 chromic gut sutures may be used. Sutures should be placed close to the wound margin and close enough to each other to relieve all wound margin tension. **C,** 4 months following repair.

be given to the wound margin to prevent pulling the wound open. The wound should then be supported with adhesive strips.

Cyanoacrylate (Histoacryl) is a polymeric glue material that has been used successfully for closure of small linear lacerations, especially in children. Lacerations in one study were limited to those that were smaller than 3 cm in length, those with nonragged edges, and superficial nonhemorrhagic wounds away from the eyelids and the vermilion border of the lip.[102-105] The glue is applied by means of a glass capillary tubing with the skin edges held together. A thin film of glue is applied while the skin edges are held in approximation for 30 seconds, or until the glue becomes opaque. Histoacryl appears to be safe when used for skin closure, although the polymer should not be allowed to come into contact with tissue below the level of the skin, where it can cause inflammation and even tissue necrosis because of toxic byproducts.[106] Dressings are not applied unless the child is very young. Gluing is quick, atraumatic, and cost-effective, with good cosmetic results. This technique obviates injections, suturing, and postoperative suture removal for small lacerations. In one study, 98.6% of patients and patients' parents were satisfied and would prefer this technique over conventional wound closure. Because complications, infections, and unusual scarring have been reported in as many as 10% of patients, careful selection of simple wounds with a low risk of contamination is essential.

Fibrin tissue adhesives are made up of two components; component I contains fibrinogen, factor XIII, and calcium chloride, and component II is composed of bovine thrombin and an antifibrinolytic agent.[106,107] Different fibrin tissue adhesives have different characteristics. Raising the fibrinogen concentration increases the binding strength. Thrombin catalyzes the conversion of fibrinogen to fibrin and initiates the activation of factor XIII. Therefore, the speed of fibrin polymerization is directly related to the concentration of thrombin. Factor XIII is necessary to initiate the cross linking of the fibrin clot. Fibrin tissue adhesives can be prepared from autologous, single-donor, or multiple-donor sources. This feature may limit their use in emergency room settings but may be of value in delayed wound closure in certain patients. These adhesives are used commonly to fixate split-thickness skin grafts and skin flaps. They also have been used as a hemostatic and sealing agent in many patients with certain blood dyscrasias.

CLASSIFICATION AND MANAGEMENT OF SOFT TISSUE WOUNDS

CONTUSIONS

Contusions are usually produced by blunt trauma that results in edema and hematoma formation in the subcutaneous tissue. The hematoma will usually resolve without incident or necessity of treatment unless it is large or becomes infected. Usually, the overlying skin and mucosa are intact, but if the contusion is associated with a laceration, the contused margins should be excised before closure.[1,13,21] The margins should be undermined at the subcutaneous level to allow for closure of the mucosal or skin layer without tension. If the contused laceration involves vital structures that would not tolerate tissue removal, such as the eyelid or nose, débridement and primary closure should be delayed until the contusion resolves. During the resolution of contusion type of injuries, hypopigmentation or hyperpigmentation of the area is not unusual but is rarely permanent.

ABRASIONS

Abrasions result from deflecting type of trauma, such as sliding along pavement, dirt, or glass, that removes the epithelial layer and papillary layer of the dermis and leaves the raw, bleeding reticular layer of the dermis exposed. This type of wound may be painful because of exposed nerve endings in the reticular dermal layer. Care should be taken to clean small particles, dirt, grease, carbon, and other pigments from the dermal layer as soon as possible to prevent fixation within the tissue and formation of a traumatic tattoo. Local anesthesia should be used and the wound should be scrubbed clean with a mild soapy solution, followed by copious irrigation with saline. To prevent drying and desiccation of the exposed wound surfaces, the abrasion should be covered with a thin layer of antibiotic ointment, such as bacitracin, and dressed with cotton gauze or covered with an antibiotic-coated cellulose acetate gauze.[5,13,21,108]

Epithelialization is complete 7 to 10 days after injury, without notable scarring if the epidermal pegs have not been completely removed.[5,20] After 3 days, the epidermal cells begin to migrate onto the abraded dermis. At 14 days, fibroblast and capillary formation in the dermis increases and new elastic fibers develop by 3 months. Regeneration is not complete for 6 to 12 months.

If the wound extends deeply into the dermal layer, notable scarring from granulation tissue formation will result. Excision of the remaining dermal tissue or excision of secondary scar tissue, with primary closure of the skin wound with 4-0 chromic sutures in the dermal layer and 6-0 nylon sutures at the surface, is indicated.[2,16,20] Exposure of abraded skin wounds to excessive sunlight during the first 6 months after injury may cause permanent hyperpigmentation. These healing wounds may benefit from protection with a sun-blocking agent with a sun protection factor of 15 or higher.

LACERATIONS

Lacerations may be sharp, with little jaggedness or contusion of the wound margins; they may have contused, ragged, or stellate margins, as seen in a crushing type of injury; or they may involve partial avulsion of tissues that remain pedicled to surrounding structures. After examination, débridement, and irrigation, the wound should be repaired in layers.

Simple Lacerations

Simple lacerations may be clean, contaminated, or contused. Clean lacerations may be repaired with little débridement or preparation necessary. Contaminated wounds should be cleansed and closed primarily, even if a delay of up to 5 to 7 days after trauma is necessary. Contused wounds should be evaluated and tissue

removed about the margins of the wound if enough tissue is available, or treatment should be delayed until the contused tissue stabilizes enough to allow for primary closure of the wound. If the laceration is beveled and ragged, the beveled portion of the wound should be excised with supporting dermal tissue to provide perpendicular skin edges and to permit closure with some wound margin eversion to prevent excessive scar formation.[1,21] Undermining of soft tissue wound margins is helpful for suturing tissue without extensive tension at the wound margin. Excessive undermining should be avoided because natural wound contraction may lead to tissue elevation at the margins of the wound and to excessive scarring. Displaced tissue should be returned to the original anatomic position and orientation. Only occasionally is there an indication for changing the direction of the wound margins by Z-plasty or for making tissue allowance for scar contracture at the time of primary wound repair.[13] These procedures should be done as secondary revision procedures, if indicated.

Stellate Lacerations

Ragged lacerations usually have a contused portion because of the blunt crushing trauma that is commonly responsible for this type of injury. To facilitate closure, ragged edges should be trimmed with a scalpel blade to make beveled wound margins perpendicular.[21] Interrupted sutures should be used to close the wound as far as the stellate portion of the wound margin, when multiple lacerations meet. Strangulation of the flap tip is commonly encountered with placement of interrupted skin sutures through such small portions of tissue. A partially intradermal horizontal mattress suture placed through the dermal layer of the tissue flap and exiting the skin on the larger portion of the wound is useful for closing the triangular wound without impairing the blood supply in the tip of the flap[109] (Fig. 21-12). Adhesive strips will assist in relieving tension on the wound margin.

Flaplike Lacerations

Flaplike lacerations involve significant undermining of the soft tissue, usually at the subcutaneous tissue or supraperiosteal level, without loss of tissue. The techniques of wound débridement and preparation already discussed must be meticulously followed. It is not uncommon to find debris in deep tissue under the flapped tissue. Preparation should include minimal débridement of involved tissue and removal of beveled wound margins for perpendicular closure. The excellent blood supply to facial structures will support tissue on very small pedicles.[13,21,32]

In flap wounds, pressure dressings play an important role in minimizing dead space and limiting hematoma and fluid formation within the deep tissue. Hematoma and lymph pooling may become infected or may promote fibrin deposition and excess scar formation beneath the flap.[13] Semicircular flaplike wounds of the head and neck may have a Z-plasty or W-plasty incorporated to align the skin edges more along resting skin tension lines and prevent possible trap door deformities and scar formation (see Chapter 22).

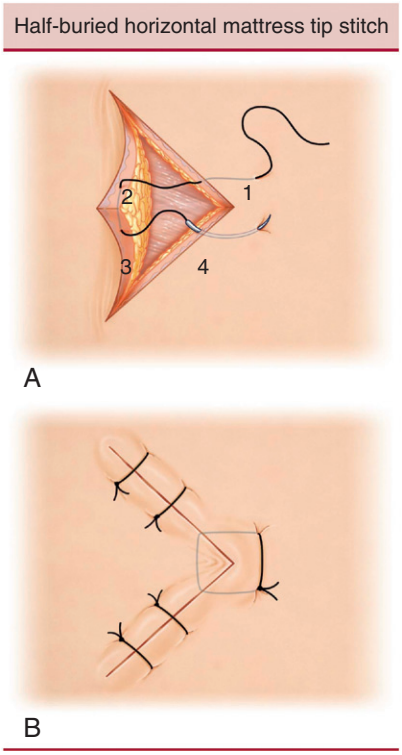


FIGURE 21-12 Half-buried horizontal mattress suture. Half of the suture lies beneath the skin in the subcuticular plane and the knot lies above the skin for easy removal. This suture is useful for approximating corners of irregular wounds. (From Robinson JK, Hanke CW, Siegel DM,, et al: *Procedural dermatology*, ed 2, St. Louis, 2010, Mosby.)

AVULSION INJURIES

The actual loss of tissue in facial wounds is fairly rare. Even if the initial evaluation suggests a loss of tissue, meticulous examination usually reveals that the tissue margins have been retracted or rolled under the wound margin. If small areas of tissue are missing, simple local undermining of the skin may provide for primary closure without tension on the wound margins. If there has been a notable loss of tissue and the wound cannot be closed free from tension with local undermining, the raw surface should be covered with a skin graft, local flaps, or apposition of the skin margin to the mucous membrane.[19,21,31] Under no circumstances should a wound on the face be allowed to heal by secondary granulation tissue because of excessive scar formation (Fig. 21-13).

DELAYED PRIMARY WOUND CLOSURE

Delayed primary wound closure is also indicated in patients with extensive facial edema or subcutaneous hematoma and when the wound margins are badly contused and tissues are devitalized. Primary repair in such damaged tissue is difficult and the possibility of bacterial infection with wound breakdown is increased. Limited débridement to remove devitalized tissue, moist dressings, and antibiotic therapy until resolution of the edema and control of infection are indicated until definitive treatment of the wound can be accomplished.

Open wound treatment, formerly universally accepted for contaminated wounds and bite injuries, is no longer



FIGURE 21-13 **A,** An avulsive scalp wound. **B,** The avulsed tissue should have as much of the subcutaneous layer and fat removed as possible to allow for adequate perfusion of the skin. **C,** The flap is then sutured into place and a compression dressing placed to prevent hematoma formation below the flap. **D,** The flap becomes revascularized. **E,** It eventually heals with hair-bearing tissue except for a small defect that will heal by secondary epithelialization.

practiced for facial wounds, except abrasive wounds.[100] Immediate definitive treatment of maxillofacial injuries was used in the Vietnam conflict. Wounds of the face and anterior cervical region were repaired with primary closure, when possible. Minimal, careful débridement was performed and the anatomy was restored to as normal a position as possible. Most of these patients could expect no major impairment of function[110] except when very destructive wounds with loss of important anatomic parts were present.

Open wounds allowed to heal with granulation tissue leave large unsightly scars on the face. These wounds should be mechanically débrided and closed primarily or, in the case of defects, closed primarily by local flaps or skin grafts. Delayed primary wound closure may be indicated if a patient is seen late with extensive soft tissue edema or has a large subcutaneous hematoma, a crushing type of injury, wound edges that are badly contused or devitalized, or both, or an increased risk of infection. Delayed primary closure is accomplished by limited débridement removing only gross foreign substances and overtly devitalized tissue. If the wound is to be treated with delayed primary closure because of contamination, edema, fractures, or other clinical findings, the patient should be treated with systemic antibiotics, the wound should be cleansed and débrided, and a sterile dressing should be placed until final treatment can be instituted.[19]

SKIN GRAFTS

Primary closure of defects with adjacent tissue and local flaps provides the most predictably successful results.



FIGURE 21-14 **A,** An avulsive injury was repaired primarily, but eventually the blood supply to the avulsed tissue was lost and the avulsed flap became necrotic and was lost. **B,** Because of the location of the lost tissue, the decision was made 10 days following the injury to allow the wound to heal by secondary epithelialization. **C,** 1 month after repair. **D,** 10 weeks after repair. **E,** 4 months after repair.

Loss of tissue may be so notable that primary closure is not possible. Closure of wounds following large tissue loss is usually secondary to epithelialization with epithelial migration and wound contraction (Fig. 21-14). Placement of skin grafts limits the amount of contraction and usually limits tissue deformity. Other indications for skin grafting include lining cavities, resurfacing mucosal deficiencies, and providing temporary coverage before definitive treatment.

Free grafting of tissue involves tissue transfer without preservation of blood supply. Therefore, vascularization and perfusion of the graft must occur rapidly to ensure graft survival. Cortical bone denuded of its periosteum cannot accept a skin graft. Tendons, nerves, or cartilage are unable to support skin grafts without their corresponding connective tissue sheaths. Other tissues, including muscle, fat, fascia, dura, and periosteum, are amenable to skin grafting if the wound surface is viable

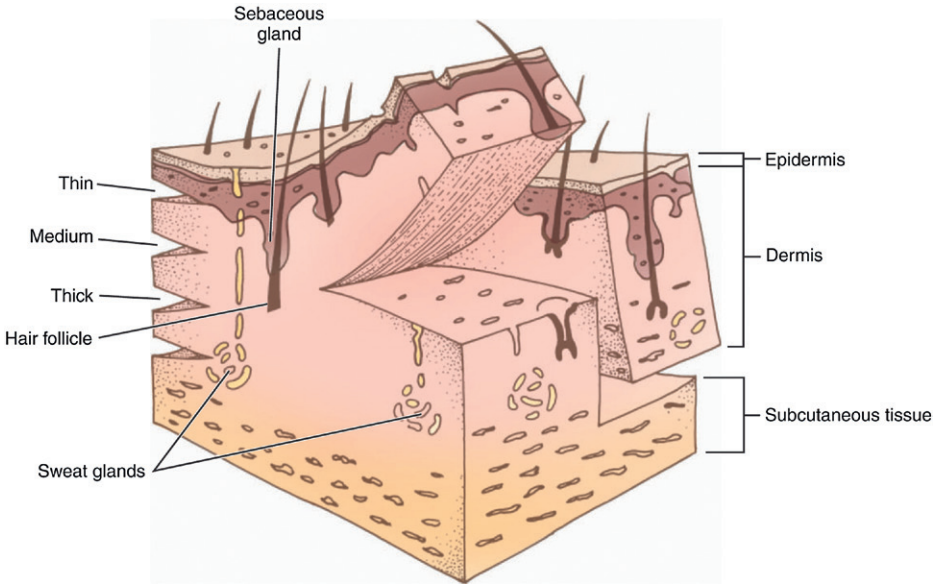


FIGURE 21-15 Free skin grafts are classified according to their thickness. Split-thickness grafts are divided into thin, medium, and thick. (From Rothrock J: *Alexander's care of the patient in surgery*, ed 13, St. Louis, 2007, Mosby.)

with good hemostasis, there is no infection, and the host is systemically healthy.

Free skin grafts are classified according to the thickness of the graft. Split-thickness skin grafts consist of the epidermis and a portion of the dermis and can be further classified as thin (0.008 to 0.012 inch), medium (0.012 to 0.018 inch), and thick (0.018 to 0.030 inch). Full-thickness skin grafts include both the epidermis and dermis (Fig. 21-15).[111] Thinner grafts rapidly vascularize and survive under less than optimal conditions. Split-thickness grafts can be expanded if necessary and have multiple donor sites that heal with minimal scarring at the donor site. Thin split-thickness grafts should be used on the face as a tissue dressing to prevent infection until repair with flap procedures can be used to reconstruct the defect.[31]

The thicker a split-thickness graft, the more closely it will resemble the qualities of color, texture, and limited contraction of a full-thickness skin graft. Thick split-thickness grafts are ideal and often provide definitive repair for large clean defects. The anterolateral area of the neck can serve as a donor site for skin grafts to the face, because the skin is similar in color and texture.[13]

Full-thickness skin grafts provide tissue of good color and texture match but are limited by their devascularization at the defect site. Optimal wound conditions are necessary and the donor site must be able to be closed primarily. Full-thickness skin grafts are usually used to repair small defects in the lip, nose, eyelid, or eyebrow (Fig. 21-16). In general, donor sites that are as close as possible to the defect should be selected to achieve the best possible color match and a texture approximating that of the surrounding skin. Such donor sites include the postauricular area, upper eyelid, supraauricular area, and antecubital fossa.[13] Free composite grafts can be obtained from the ear primarily to reconstruct avulsions of the nasal alar base. These grafts will survive if no part of the graft is more than 1.0 to 1.5 cm away from the nutrient bed.[21]

With free skin grafts, pressure dressings should be left in place for 7 to 10 days to prevent hematoma or fluid accumulation and to facilitate perfusion of the graft. The grafted skin should be kept well lubricated with oil-based lotion to prevent excessive drying and prolonged exposure to the sun should be avoided.[111]

In preparation, the wound should be débrided thoroughly and irrigated with a physiologic saline solution or Dakin's solution (10% sodium hypochlorite in saline). Many standard scrub solutions provoke an inflammatory response and may compromise the recipient bed. Moist dressings may be applied until the graft is placed. Full-thickness grafts may be harvested by dissection, with the physician carefully separating the skin from the underlying fat, which may act as a barrier between the blood supply and graft tissue. Donor sites should be closed primarily. Split-thickness grafts may be harvested with a dermatome, with the physician carefully maintaining the correct angle with a pulling motion.

Graft immobilization is key to the success of skin grafting. Grafts are held in place with sutures, staples, or tape and should be tension-free, because even minimum shearing forces result in tearing. Tie-over dressings may be best for ensuring graft immobilization. Initially, the graft survives by the plasma exchange of nutrients, called plasmodic imbibition; this process is responsible for graft nutrition for the first 48 hours. Fluid is passively absorbed by the graft, which leads to edema within 2 to 3 days. Inosculation is the vascular supply of the graft spreading to the host bed. Vascular ingrowth by vascular beds from the recipient site also occurs. Revascularization is complete within 4 to 7 days, and lymphatics are established after 4 to 5 days. A wound will contract after grafting. Full-thickness grafts show minimum contracture, whereas a split-thickness graft may contract by up to 30%



FIGURE 21-16 **A,** An avulsive injury involving the upper eyelid, eyebrow, and scalp with loss of soft tissue so that primary closure was not possible. **B,** A split-thickness graft was obtained from the lateral portion of the neck to cover the defect. The anterolateral area of the neck provides a donor site for skin grafts to the face because the skin is similar in color and texture. **C,** The thinner the graft used, the quicker the graft will vascularize and survive. The graft is secured with interrupted 6-0 sutures and a pressure dressing is applied. **D,** The pressure dressing is secured to the area to prevent hematoma or fluid accumulation and to facilitate perfusion of the grafted skin. The grafted skin should be kept well lubricated to prevent excess drying and avoid prolonged exposure to the sun. **E, F,** 6 months following reconstruction, with good function of the upper eyelid maintained. Hair grafts may be considered in the future to restore the lost portion of eyebrow.



FIGURE 21-16, cont'd

to 35%.[112,113] Most grafts will also undergo pigmentation changes as a result of damage to pigmented cells. Usually, the wound goes through a period of hypopigmentation early and then a period of hyperpigmentation before it returns to normal color. Reinnervation is complete in most grafts but appears to be superior in split-thickness grafts compared with full-thickness grafts.[114,115]

The most common causes of graft failure are hematoma formation and failure of immobilization. Careful hemostasis and periods of proper immobilization prevent failure and the necessity for revision procedures. Careful treatment planning, taking of the patient's history, and examination of the recipient bed increase the chances of graft success. Improper orientation of the grafted tissue, such as securing a graft upside down or wrong side down, may lead to graft failure.

FLAPS

Local or regional flaps provide one-stage repair of avulsion defects with similar tissue that has its own vascular supply and is not dependent on the perfusion of damaged tissue, as with free skin grafts. The disadvantages include additional incisions, elevation of tissue on the face, and increased scarring. In the design of all flaps, the blood supply and venous drainage are of prime concern. The method of closing the secondary defect must be planned before the procedure. Facial flaps do best when based laterally or inferiorly, with the incisions following normal skin folds and lines of expression.[13] The basic skin flaps used on the face are advancement, rotational, transpositional, and microvascular anastomotic flaps.

Advancement flaps involve making two parallel incisions from the defect and undermining the tissue until the flap can be advanced into the defect under minimal tension. Dog ears created at the base of the flap should be carefully excised. Rotational or rotational-advancement flaps use a semicircular flap to rotate tissue into a defect, with primary closure of the secondary defect. This flap requires careful planning to keep the incision from crossing too many natural skin folds. The circumference of the circle should be eight times the size of the defect and at least twice the diameter.[19,31]

Transpositional flaps involve swinging flaps into areas of defect over healthy tissue, with a secondary defect at the donor site that is closed primarily by undermining adjacent tissue or by coverage with a free skin graft. The donor site is usually in the neck or scalp region and closure is within the natural creases or in the hair. With large defects, a flap may have to be swung up from the chest region.[31]

Microvascular anastomotic flaps are usually not indicated in defects of the face, although some favorable results have been reported with the reconstruction of scalp wounds.[116] With facial avulsive defects, adequate arteries or veins may not be close enough to the defect for successful anastomosis. When successful, these flaps provide excellent coverage but are often bulky on the face and may require multiple revisions to thin. Conventional coverage with a free skin graft or local skin flaps may provide a superior result, with a much simpler operative procedure.[13]

Local flaps can be divided into two groups, those that rotate about a fixed point to reach the defect (rotational, transpositional, and interpolated) and those that advance into the defect (single pedicle, bipedicle, and V-Y advancement). The fundamental motion of the tissue should be a straight line from donor site to recipient site, with minimal rotational or lateral movement.

Rotational Flap

The movement of the rotational flap is in an arc around a fixed point, primarily within one plane. The act of rotation results in less reliance on tissue elasticity for flap movement, allowing these flaps to be useful in areas of inelastic skin, such as the scalp and nasal dorsum.[117,118] An example is the semicircular flap (Figs. 21-17 and 21-18). When possible, the flap should be designed so that it is inferiorly based, which promotes lymphatic drainage and reduces flap edema. The disadvantages of rotational flaps are relatively few. The defect itself must be somewhat triangular or modified by removing normal tissue to create a triangular defect. As with all pivotal types of flaps, rotational flaps develop cutaneous deformities at their bases that may not be easily removed

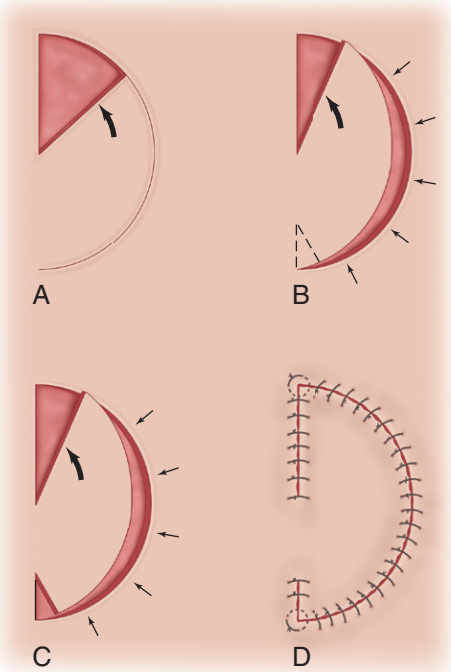


FIGURE 21-17 The rotational flap is useful in soft tissue surgery of the face. **A,** The flap is planned with judicious removal of tissue about the defect, allowing for rotation into this space. **B,** The flap and adjacent tissue are undermined, allowing for movement. **C,** To gain further rotation into the primary defect, it is sometimes useful to make a small incision to release the posterior portion of the base. **D,** The flap is then secured over the defect and the secondary defect is then occasionally able to be closed primarily.



FIGURE 21-18 Double-rotation (O-Z) flaps. **A,** A circular defect may be closed with the double-rotation flap, with the primary tension shown at the junction of the two flaps. **B,** A square defect may be divided into two triangular defects. Each triangular defect is closed with its own advancement flap.

without compromising the vascularity of the flap. Thus, a second-stage removal of the deformity may be necessary.

Transpositional Flap

The transpositional flap is a rectangular flap that rotates about a pivot point (Fig. 21-19). Transposition is the most common method of moving tissue into local defects of the head and neck region. The length of the flap should not exceed three times the width, although the abundant vascularity of the head and neck often enables the development of flaps that exceed this 3:1 ratio. The more it is rotated, the shorter the flap becomes. Like rotational flaps, these flaps tend to be pushed rather than pulled over the defect by forces created by closure of the secondary defect. They also tend to drape into place with very little tension. Careful planning is necessary because once flaps have been incised, they cannot be enlarged. Frequently, dog ears are created; these are best removed after the flap is fixed into place so that a precise amount of tissue can be removed.[117-119]

Interpolated Flap

With the interpolated flap, the donor site is separated from the recipient site and the pedicle of the flap must pass over or under the tissue to reach the recipient area. A second surgical procedure is usually necessary to release and modify the flap (Fig. 21-20). An advantage of the interpolated flap is the use of distant tissue with aesthetically pleasing characteristics, such as skin texture, thickness, and color match. An example is the nasolabial flap for reconstruction of the nose.

Single-Pedicle Advancement Flap

With the single-pedicle advancement flap, a rectangle of skin is pulled forward on the basis of the elastic properties of the skin. The design of the flap should take advantage of local skin tension lines, cosmetic borders, and regional blood flow patterns. Generally, inferiorly or laterally based flaps are best. Excision of deformities caused by flap design, such as Burrow's triangles, may facilitate movement of the flap and help prevent of tissue bunching and dog ears. It is unwise to move advancement flaps toward free margins, such as the lip and eyelid, because of the increased risk of ectropion and eclabium. Movement across or parallel to free margins is recommended.[118,120] Single-pedicle advancement flaps work well in certain areas, such as the forehead, helical rim, upper and lower lips, and medial cheek. Mucosal advancement flaps are also useful for vermilion reconstruction.[119]

Bipedicle Advancement Flap

With the bipedicle advancement flap, or H-plasty, the same principles apply as for the single-pedicle advancement flap. An incision is made parallel to the defect and the flap is undermined and advanced. The length of each flap is 1.5 to 2.0 times the width of the defect. The equal and opposite motion of the two flaps minimizes the impact on the surrounding tissue. Because each flap covers only half of the defect, they are smaller and move less, which results in better relative blood flow and less tension on the closure.[119,120] Often, this type of advancement requires skin grafting to close the donor site.

V-Y Advancement Flap

With the V-Y advancement flap, an elliptical incision is planned over the defect using a 3:1 ratio, in which the length of the ellipse is three times the length of the defect. The advancing edges of the two flaps are trimmed and subcutaneous dissection is started. Each V-shaped

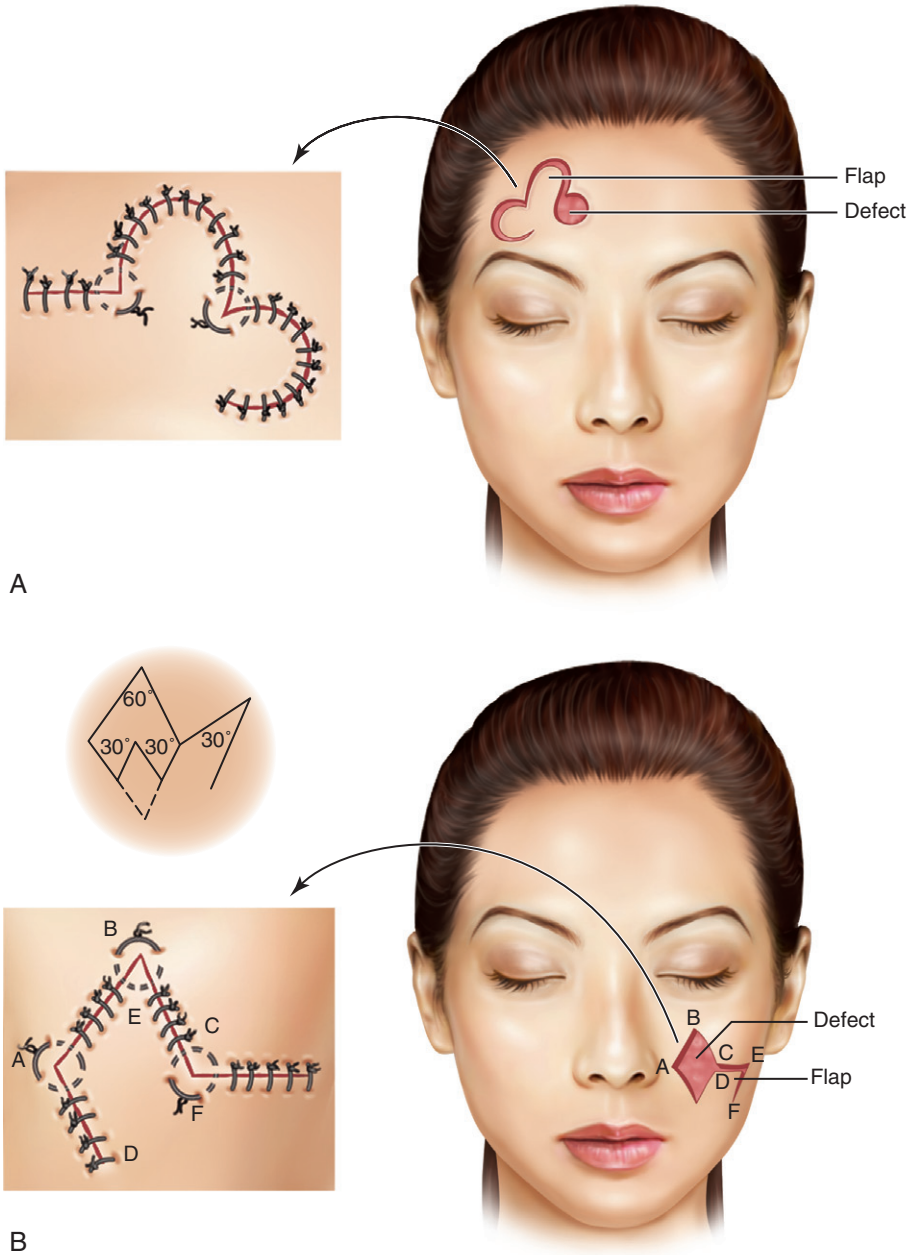


FIGURE 21-19 Transpositional flaps. The base of the flap is parallel to the lines of maximal extensibility. The secondary defect is closed primarily. **A,** A bilobed transpositional flap is used to close a scalp defect. **B,** The classic rhomboid transpositional flap (Limberg flap) and a modified 30-degree rhombic flap (*inset*).

flap is undermined until a small central vertical base of tissue is formed. The two island flaps are advanced and approximated over the primary defect. The secondary defects are then closed primarily (Fig. 21-21). When flaps are constructed on the lip or cheek, more advancement is possible compared with flaps on the forehead because of the thicker layer of subcutaneous fat.[119,121]

APPOSITION OF THE SKIN MARGIN TO MUCOSAL MEMBRANES

Full-thickness defects in the cheek, nose, or lip—commonly seen with gunshot wounds—usually cannot be repaired primarily by skin grafting or flap procedures. The mucous membrane and skin margin should be undermined and closed primarily. With primary closure of the wounds, infection, delayed healing, and scar contracture will be prevented.[119,121] There will be no distortion of local tissue around the defect, which will allow for secondary reconstruction of the defect, with stable anatomic landmarks.

ANIMAL BITES

Approximately 1 to 2 million animal bites are treated annually; dog and cat bites alone account for approximately 1% of all emergency room visits in the United States annually.[122] Usually, 10 to 20 dog bite–related fatalities occur annually. Dog bites are most common, accounting for 63% to 93% of reported animal bites to

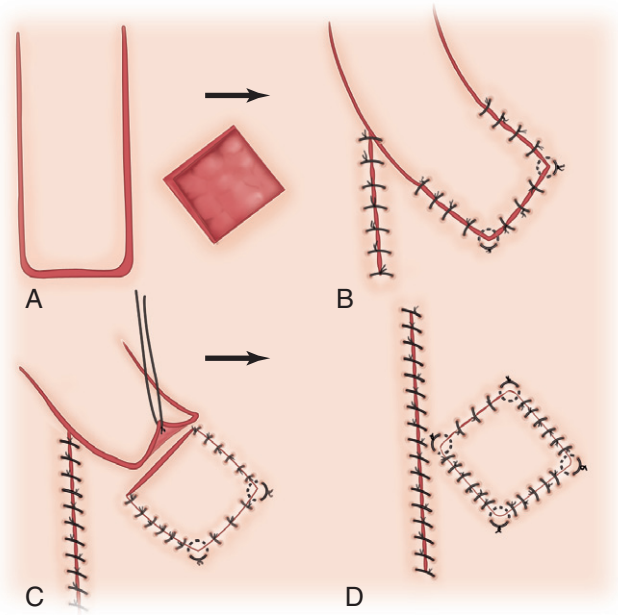


FIGURE 21-20 Interpolated flap. **A,** The flap is planned. **B,** The flap is elevated and closed over the defect, and the donor site is closed. **C,** Once the flap is revascularized, its pedicle is divided. **D,** The base is closed primarily.

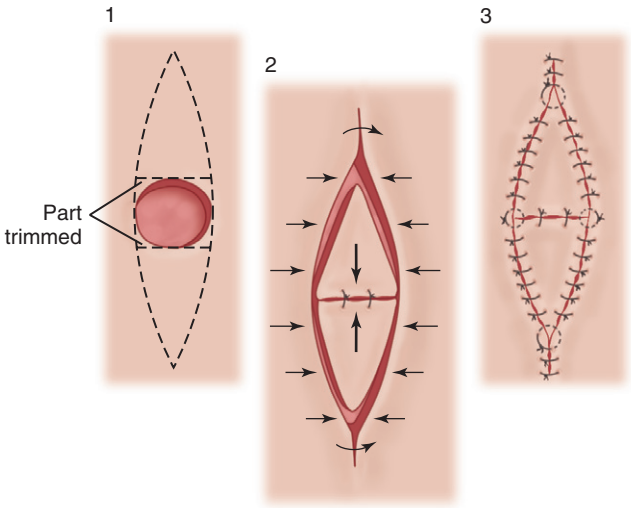


FIGURE 21-21 The V-Y plasty, or island advancement flap. *1,* The four rounded tips of the advancing flaps are trimmed and the 3:1 dimension of the total length of the ellipse is planned. *2,* The V-shaped island of tissue is raised and dissected from its base until a small central line of tissue is formed in the center of the island. *3,* The first suture is placed to align the advancing edges of the two flaps. The tip sutures are placed to align the tails. Next, the subcutaneous sutures are placed to approximate the corners and closure is completed.

humans, primarily children. Head and neck bites are more common in younger age groups, in children younger than 10 years.[123-125] Although infections resulting from animal bites are polymicrobial, *Pasteurella* spp. *(P. multocida)* are most frequently cultured from dog bite wounds, followed by *S. aureus*. The feline oral flora is more likely to harbor *P. multocida*.[126,127] Infections that occur with *Pasteurella* species are most likely to be seen within 24 hours after the incident whereas, after 24 hours, wounds are more likely to contain *Staphylococcus* or *Streptococcus* species.[128]

With animal bites, the wounds should be evaluated, with the patient receiving local anesthetic. Radiographs should be obtained if there is considerable edema about the wound or if bony penetration or foreign bodies are suspected. Canine jaws are capable of compressive forces of 200 to 450 psi and skull fractures with cranial penetration have been reported[122,129,130] (Fig. 21-22).

Wounds should be thoroughly débrided and irrigated. Historically, animal bite wounds were not closed primarily, although some investigators supported closure.[124,131] In most laceration types of injuries, it is safe to close the wounds primarily after proper wound preparation without increased risk of the development of infection. Puncture types of wounds should not be closed primarily because it is difficult to clean and prepare the wound adequately. Bite wounds with extensive crush injury and wounds requiring a considerable amount of débridement are best treated with delayed primary closure.[122,132]

Proper antibiotic therapy is important for the prevention and treatment of infections caused by animal bite wounds. For prophylaxis of uninfected wounds, dicloxacillin or Keflex (cephalexin monohydrate) is recommended.[122] For infections presenting within the first 24 hours of the incident (implicating *P. multocida*), penicillin or amoxicillin is recommended. If the infection develops beyond 24 hours after injury (implicating *Staphylococcus* or *Streptococcus*), an antistaphylococcal penicillin, such as dicloxacillin or cephalexin, is suggested for antibiotic coverage.[132] Animal bites should be considered prone to tetanus and treated accordingly.

Rabies is a serious consideration with any animal bite. Immediate and thorough washing of the wound with a soap or scrub solution is probably the best prevention against rabies. Postexposure rabies prophylaxis is recommended for patients attacked by suspected animals or in geographic locations in which the incidence of rabies is particularly high. It consists of human diploid cell vaccine (HDCV) or rabies immune globin (RIG). If necessary, captured animals may be tested to determine the necessity of treatment.[132] For more detailed information, see Chapter 23.

INJURIES TO STRUCTURES REQUIRING SPECIAL TREATMENT

LIP

The lip provides special challenges to repair following trauma because of the anatomy of the region of the vermilion border, which involves the transition of mucosal tissue to skin, associated edema of the tissue after trauma to the area, and aesthetically displeasing and difficult to correct puckering defect that results from an irregular vermilion margin (Fig. 21-23). A mismatch of even 1 mm at the vermilion may be readily noted by an observer (Fig. 21-24). Scars or defects that affect the sphincter activity of the orbicularis oris muscle produce drooling, functional difficulties in eating, and alterations in speech.

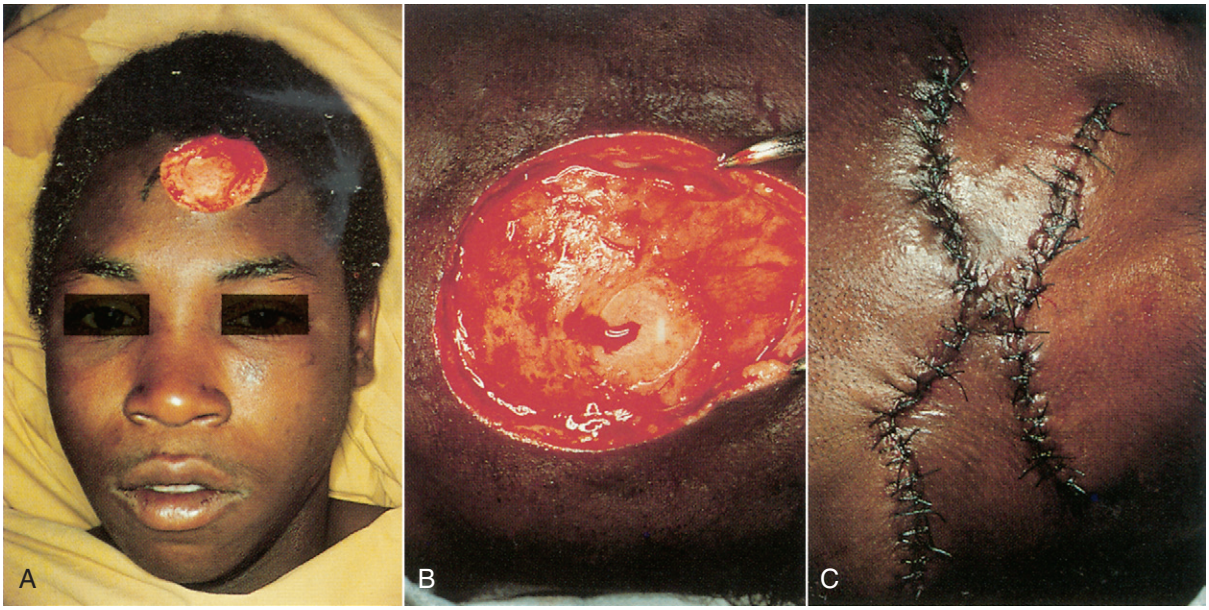


FIGURE 21-22 A large avulsive forehead wound with loss of the pericranial tissue and exposed bone. The wound is closed primarily with rotational advancement flaps.



FIGURE 21-23 **A,** The lip provides special challenges to repair following trauma because of the anatomy of the region of the vermilion border. A mismatch of even 1 mm may be readily noted at the vermilion by an observer. **B,** After examination, a single 5-0 suture should be placed at the mucocutaneous line, or gray or white line, to reorient this important junction. The muscular layer is reapproximated with 3-0 or 4-0 chromic sutures; the dermis and subcutaneous tissues are closed with 4-0 or 5-0 chromic sutures. The skin should be carefully approximated with 6-0 sutures placed evenly and the mucosal layer is loosely reapproximated with 4-0 sutures. **C,** 3 months after repair.

After examination, a single 5-0 nylon suture should be placed at the mucocutaneous line (gray or white line) to reorient this important junction (Fig. 21-25). The wound should be débrided, hemostasis achieved, and local anesthesia achieved in the surrounding tissue or via mandibular or mental nerve blocks. Blocks are preferred to prevent unnecessary edema in tissue to be approximated. The wound should then be closed in layers. Proper reapproximation of the orbicularis oris muscle is important if the muscle has been disrupted (Fig. 21-26). Severed muscle fibers tend to retract, which may lead to later depression of the scar with maturation of the wound. The muscular layer is reapproximated with 3-0 or 4-0 chromic sutures, the dermis and subcutaneous tissue are closed with 4-0 or 5-0 chromic sutures, the skin should be carefully approximated with 6-0 nylon sutures placed evenly, and the mucosal layer is loosely reapproximated with 4-0 chromic suture (Fig. 21-27).

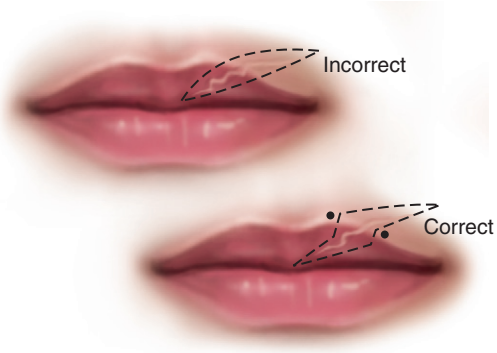


FIGURE 21-24 The vermilion skin junction should be crossed at 90 degrees so that correct alignment may be achieved.



FIGURE 21-26 Large lip laceration with concomitant maxillary fracture and tooth avulsion. Proper reapproximation of the orbicularis oris is essential for preventing later depression of the scar.



FIGURE 21-25 Closure of the commissure of the mouth presents an unique challenge with this child, who suffered a dog bite. The wound should be débrided, hemostasis achieved, and a local anesthetic administered in the surrounding tissue. The wound should then be closed in layers. Proper reapproximation of the orbicularis oris muscle is important with any lip repair if the muscle has been disrupted.

In avulsive injuries to the lips, 25% of the upper lip and up to 25% of the lower lip can be lost without resultant functional or aesthetic defects.[133] The tissue margins should be straightened, with removal of a full-thickness wedge of lip tissue to facilitate closure. If there has been an extensive avulsive injury, an Abbe-Estlander flap between the affected lip and the opposite lip can be used to rotate tissue into the avulsed area. Another type of rotational flap is the Karapandzic flap, which uses full-thickness perioral tissue about the oral stoma. The lips are advanced along with the orbicularis oris, neurovascular bundle, and underlying mucosa to close the defect. The major complication is the reduced size of the oral stoma[3] (Fig. 21-28). Distinct cutaneous creases outline



FIGURE 21-27 Flaps of tissue, no matter how small, should be maintained if still attached. The island of vermilion border is secured with multiple interrupted 5-0 nylon sutures, with care taken to maintain the alignment of the gray line of the vermilion border junction with the skin.

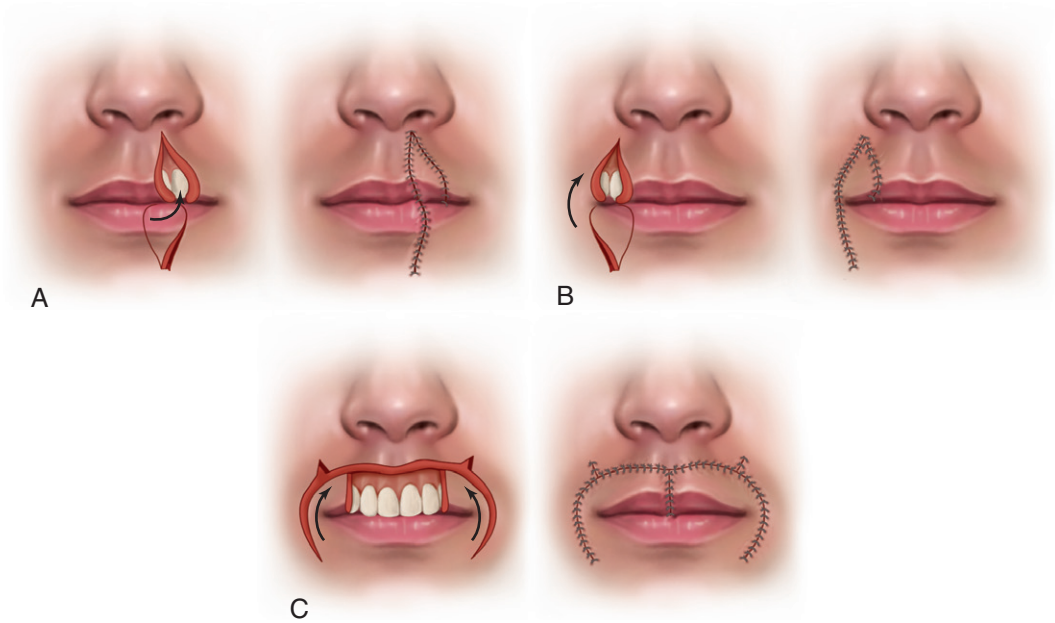


FIGURE 21-28 Reconstructive flaps used in avulsive lip injuries. **A,** The Abbe flap. **B,** The Abbe-Estlander flap. **C,** The Karapandzic flap.

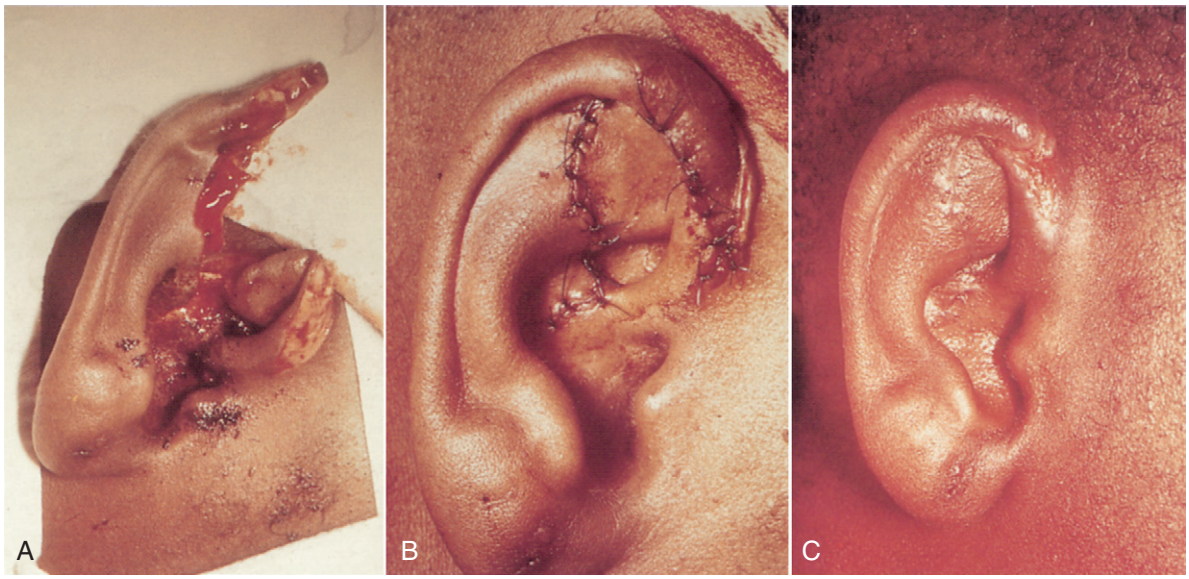


FIGURE 21-29 The excellent blood supply to the ear can support large portions of tissue on very small pedicles. The first sutures should reapproximate known landmarks and secondary sutures should reapproximate adjoining tissue. Conservative débridement should be used to maintain as much tissue as possible.

FIGURE 21-30 **A-C,** The pinna consists of a thin central area of relatively avascular cartilage that depends on the thin overlying layer of skin for its blood supply. The ear has a good blood supply and can maintain large portions of tissue on very small pedicles. Conservative débridement and manipulation should be used to maintain as much tissue as possible. Sutures should first be used to reapproximate known landmarks and then should be placed to reapproximate adjoining tissue. The skin should be approximated with 6-0 or 7-0 nylon sutures or other fine suture material. Suturing of the cartilage is usually unnecessary and may lead to devitalization of the region of cartilage, or may provide a pathway for infection. **D,** 3 months after repair.

the anatomic lip unit. The mental crease divides the lip and chin, the nasolabial crease defines the lateral borders, and the base of the nose serves as the superior limit. These dominant features of the lower face are important in facial aesthetics because they are excellent locations for camouflage of scar lines in lip repair. Distortion of these lines may be significantly deforming.[134]

EAR

In the assessment of injuries to the ear, a complete examination of the external ear, pinna, tympanic membrane, and hearing should be performed and documented before treatment (Fig. 21-29). The external ear consists of the pinna, external auditory meatus, and tympanic membrane. The pinna consists of a thin central area of relatively avascular cartilage that depends on the thin overlying layer of skin for its blood supply.[32] The ear has a good blood supply and can maintain large portions of tissue on very small pedicles. Conservative débridement and manipulation should be used to maintain as much tissue as possible. Sutures should first be used to reapproximate known landmarks and then should be placed to reapproximate adjoining tissue. The skin should be approximated with 6-0 or 7-0 nylon sutures or other fine suture material. Suturing of the cartilage is usually unnecessary and may lead to devitalization of the region of cartilage or may provide a pathway for infection. If sutures in the cartilage are necessary, fine chromic sutures are recommended.[135] Torn cartilage should be repaired with a minimum number of sutures (Fig. 21-30).

In avulsive injuries involving segmented portions of the external ear that are missing or attached only with a small pedicle flap, the tissue should be returned to proper anatomic position and secured with sutures to the skin. The skin from the dorsum of the ear should be dermabraded and attached to a skin flap elevated from the mastoid region for a vascular bed.[136] Postoperative treatment should include bed rest, use of a supportive bandage, application of ice to cool the replanted part and decrease the metabolic rate within the segment, heparin anticoagulant treatment, and antibiotics to cover gram-positive bacteria.

Total amputation of the external ear is a difficult repair and reconstruction problem. Plastic surgery grafting procedures to reconstruct the external ear with rib cartilage, skin flaps, or Silastic or silicone implants have had variable results[137,138] and are rarely satisfactory. Some success has been reported with microvascular techniques. The superficial temporal artery or posterior auricular arteries are used, but there are problems with artery size and poor venous drainage that make salvage difficult.[136]

Prosthetic rehabilitation of the external ear and other specialized facial structures has been greatly improved with the development of silicone and plastic materials for more reliable and stable color match, comfort, and durability.[35,139] Attachment of the prosthesis to eye-


A
B
C
D

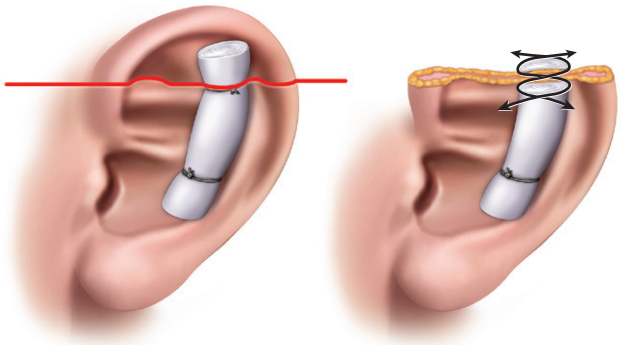


FIGURE 21-31 After aspiration or incision and drainage of an otohematoma, the ear may be dressed by using dental cotton rolls bolstered with sutures through the pinna, as shown above. This technique provides compression to prevent recurrence.



FIGURE 21-32 A significant deformity may develop if hematoma formation is not well controlled over the cartilage of the ear. The hematoma may become fibrosed and result in a thickened ear, known as a cauliflower ear. Placement of compression sutures eliminates dead space and prevents recurrence.

glasses is the most common method used, although some prostheses are held with glue to the skin margins.[101] The disadvantages with gluing are that the glue may not tolerate sweat or oils, and allergic skin reactions are possible. Retention and good fit with an eyeglass-supported prosthesis are also difficult to achieve. With the development of osteointegrated implants and techniques for placement of the implants in the mastoid region, zygomatic buttress, and other supporting bone, prosthetic devices can be anchored to replace missing external ears, orbits, and noses.[137]

Sometimes, the skin of the external ear is lost but the cartilage is preserved. If only a small defect exists or if the perichondrium still covers the cartilage, a skin graft should be used to cover the defect.[140] A good donor site is the retroauricular skin of the contralateral ear. When the perichondrium is missing, the best treatment is coverage with a retroauricular skin flap.[136]

Hematomas of the ear should be aspirated with a fine needle or small incisions in dependent drainage areas. A hematoma that is not removed may become fibrosed and cause a thickened ear, known as a cauliflower ear. Dressings are extremely important and should be molded to the shape of the ear to support the ear and provide gentle pressure to prevent recurrence of the hematoma[140] (Fig. 21-31). One technique is to place cotton balls soaked in antibiotic solution along the area involved. A mixture of rubber base or silicone impression material is pressed into the ear, with a cotton ball in place to protect the external auditory meatus. The material is allowed to set and acts as an excellent compressive bandage. The dressing should be left in place for 5 to 7 days.

Otohematoma is one of the more common injuries of the external ear. A significant deformity may result from improper initial management (Fig. 21-32). Many initial treatments, such as needle aspiration or simple incision, tend to be unsuccessful and require several attempts because of the reaccumulation of fluid despite meticulous dressing application. Fibrosis and new haphazard cartilage may develop if the hematoma is not completely drained. Simple compression sutures yield superior results by eliminating dead space and preventing recurrence. This technique uses a small incision over the hematoma, with decompression and primary skin closure. Horizontal mattress sutures are then passed through the entire thickness of the ear overlying the area of hematoma formation. These sutures should incorporate a small bolster dressing on either side of the ear (see Fig. 21-31). Usually, two to three sutures of this type are necessary. A precise head dressing with moist gauze or heavy-bodied impression material is molded into the ear interstices. Dry cotton gauze or fluffs are placed over the pinna and between the external ear and mastoid region. Finally, a soft gauze bandage is firmly wrapped around the patient's head. The head dressing is removed in 2 to 3 days and the tie-through dressing and sutures are removed in 7 to 10 days.[141-143]

Anesthesia of the auricle is achieved with a solution of 1% lidocaine or 2% bupivacaine , without vasoconstrictor, injected subcutaneously at the base of the auricle to block the nerves providing sensation to the external ear. These nerves consist of the auriculotemporal branch of the mandibular nerve, lesser occipital nerve, great auricular nerve, and auricular branch of the vagus nerve (Arnold's nerve; Fig. 21-33).

Exposed cartilage presents a special problem. Perichondritis may develop and, if left untreated, necrosis of the cartilage may occur. For this reason, conservative débridement of devitalized tissue and complete coverage of all cartilage with skin are indicated, if possible.

NOSE

The nose is the most prominent structure on the face and is commonly traumatized. Many injuries result in fractures to the bony structure, with or without soft tissue involvement. As in the treatment of any bone fractures, the underlying bones must be repaired before soft tissue wounds are closed. The bone and cartilage substructure of the external nose are covered by muscle, subcutaneous tissue, and skin and have an internal lining of mucous membranes and glandular structures. Like other

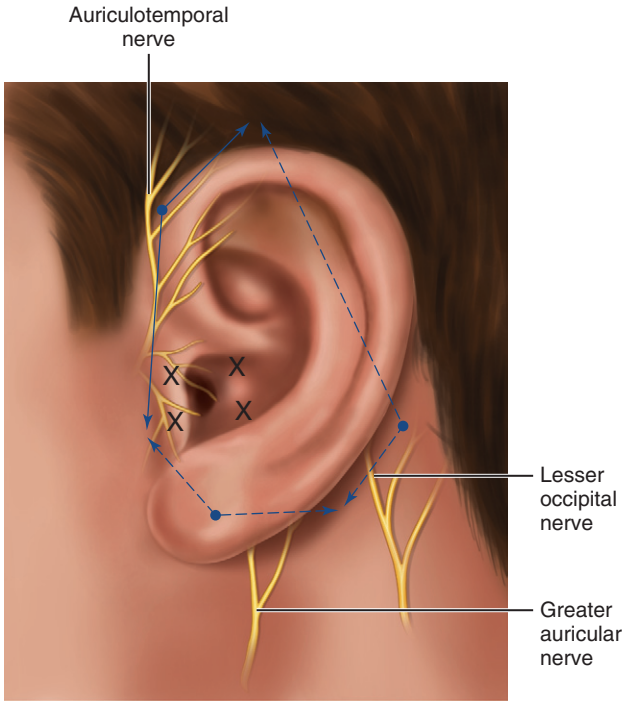


FIGURE 21-33 Ear block. The dots are the site of needle placement. A suggested trajectory for the needle *(arrows)* is shown for placing the anesthetic in relation to the major nerves that innervate the ear. This method will not anesthetize the external canal adequately and must be augmented (X) if it is necessary to include this area.

structures on the face, the entire nose has an excellent blood supply and most lacerations, wounds, and incisions of the skin of the nose heal readily and rapidly.[144] Examination of the internal nose requires a nasal speculum, excellent lighting, and suction. Anesthesia with a 4% or 10% cocaine solution and lidocaine with epinephrine may be necessary for an adequate examination. The mucosa is inspected for evidence of lacerations, and exposure of the cartilage is noted (Fig. 21-34). The septal cartilage should be evaluated for displacement, buckling, and possible fracture. The septum must be assessed for the presence of hematoma, which would appear as a boggy blue elevation of the mucosa.[145] Once diagnosed, a septal hematoma should be evacuated through small mucosal incisions or by needle aspiration. A running 4-0 chromic suture is placed through and through the septum to prevent recurrence.[136] If untreated, the septal hematoma may become infected and septic necrosis of the cartilage is possible. With destruction and collapse of the septum, the nose loses its supporting framework, resulting in retraction of the columella and saddling of the middle third. The septum may also become thick secondary to subperichondrial fibrosis.[13]

Mucosal tears may be associated with bone fractures or penetrating objects. Suturing should be attempted with thin absorbable sutures. Exposed septal cartilage does not pose any difficulty as long as the mucosa is intact on the other side of the septum. If the cartilage is divided, a mucosal flap should be designed to cover the area, at least on one side.[144]

Lacerations of the skin of the nose should be closed after inspection and débridement with 6-0 nylon or other nonabsorbable sutures. Partial avulsions and through and through lacerations should be closed by suturing the mucosal layer with fine absorbable sutures, placing the knots so that they are in the nasal cavity. Key sutures should be used to align landmarks to ensure proper orientation, especially about the nasal rim. Repair should then continue with approximation of the cartilage with 5-0 chromic sutures and closure of the skin with 6-0 nylon sutures. Because of the thick sebaceous skin over the nasal tip and high content of bacteria, suture abscesses are common and the skin is prone to developing scars.[5,144] Sutures should be removed after 4 days and reinforced with adhesive strips (Fig. 21-35).

Avulsive wounds of the nose may require skin grafts. Skin grafts are ideally done with full-thickness postauricular grafts, which give the best possible match of color and texture. Split-thickness skin grafts can be used if necessary and are best obtained from exposed body areas, such as the neck or forearm.[13] Davis and Shaheen[19] noted that almost 50% of composite grafts will fail even in ideal conditions; these grafts should be used only if the following conditions apply:

1. The wound edges are cleanly cut and viable.
2. There is no prospect of infection.
3. Primary repair is not delayed.
4. No part of the graft is more than 0.5 cm from the cut edge of the wound.
5. All bleeding is controlled.

Abscess formation in the nasal septum is rare. If an infection develops, it is usually from *S. aureus* invasion of a nasal septal hematoma following nasal trauma. Both hematoma and nasal septal abscess formation can lead to cartilage destruction, with severe functional and cosmetic sequelae, consisting of septal deformity, perforation, and saddle nose deformity. A hematoma of the nasal septum will separate the mucoperichondrial blood supply from the septal cartilage. Cartilage necrosis results within 3 days from ischemia and pressure but can rapidly occur with abscess formation within 24 hours.[146-148] Potentially life-threatening complications from the spread of infection include osteomyelitis, orbital and intracranial abscesses, meningitis, and cavernous sinus thrombosis.

The typical clinical presentation includes nasal obstruction from a tender swelling of the anterior nasal septum. When two applicator sticks are pushed against the septum bilaterally, the septum should feel firm. If a hematoma is present, a soft fluctuant consistency will be noted. Prompt drainage of a nasal septal hematoma not only prevents abscess formation but also prevents cartilage destruction that can occur even without infection. Drainage and antibiotic therapy minimize the damage caused by a septal hematoma, but treatment does not reverse the damage to the cartilage. Needle aspiration or staggered incisions are made bilaterally in a dependent location to prevent septal perforation. Penrose drains, nasal packing, or both, are used to prevent the reaccumulation of blood or pus. An alternative is application of a septal stent that consists of two pieces of Silastic



FIGURE 21-34 **A,** An injury to the nose with a large laceration through the skin and mucosal tissue. **B,** The mucosal tears were repaired with thin absorbable sutures. Exposed septal cartilage does not pose any difficulty as long as the mucosa is intact on the other side of the septum. If the cartilage is divided, a mucosal flap should be designed to cover the area at least on one side. **C,** The lacerations of the skin of the nose were closed after inspection and débridement with 6-0 sutures and a splint placed to prevent hematoma formation **(D).**



FIGURE 21-35
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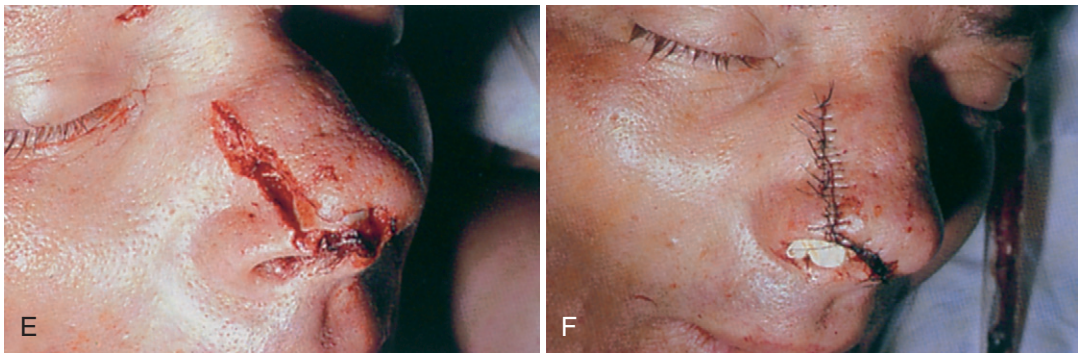


FIGURE 21-35, cont'd **A-D,** Through and through lacerations should be closed by suturing the mucosal layer with fine absorbable sutures, placing the knots so that they are in the nasal cavity. Key sutures should be used to align landmarks to ensure proper orientation, especially about the nasal rim. Repair should then continue with approximation of the cartilage with 5-0 chromic sutures and closure of the skin with 6-0 nylon sutures. Because of the thick sebaceous skin over the nasal tip and high content of bacteria, suture abscesses are common and the skin is prone to developing scars. Sutures should be removed after 4 days and reinforced with adhesive strips. **E, F,** Fracture of the nasal bones with concomitant laceration. The nasal fractures were reduced, the nose was packed, and the laceration was closed in layers.

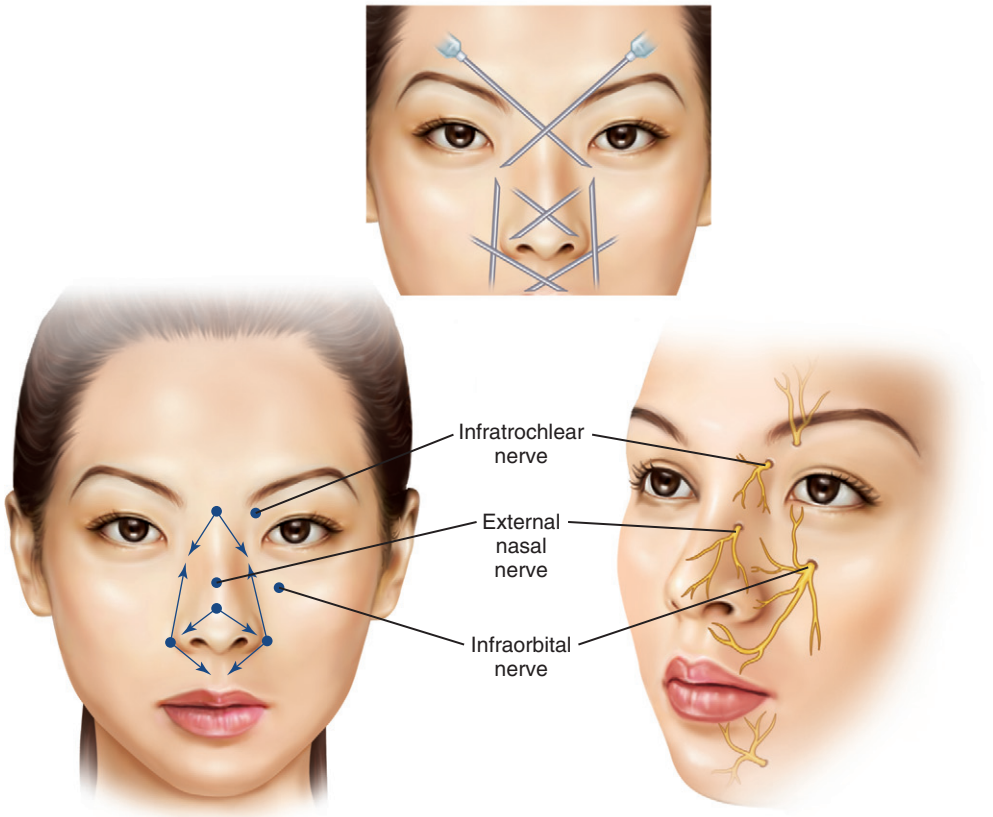


FIGURE 21-36 Field block of the nose. The sites of needle insertion *(dots)* and line of infiltration *(arrows)* are shown in relation to the sensory nerves of the face.

material placed bilaterally against the septal wall. These stents are secured by large, trans-septal Prolene sutures passed through both Silastic stents and septum in a horizontal mattress fashion. Stents may be combined with nasal packing and removed in 7 to 10 days.[147,149]

Anesthesia (Fig. 21-36) may be achieved with 10% cocaine in 1:10,000 epinephrine or a 1:1 mixture of 4% lidocaine and phenylephrine (Neo-Synephrine) soaked in cotton rolls, which are packed into the nares bilaterally. Next, 1% to 2% lidocaine in 1:100,000 epinephrine is injected bilaterally at the nasal bridge to block external branches of the anterior ethmoid and supratrochlear nerves. Infiltration with lidocaine or bupivacaine at the vestibule, ala, and floor of the nose will block external

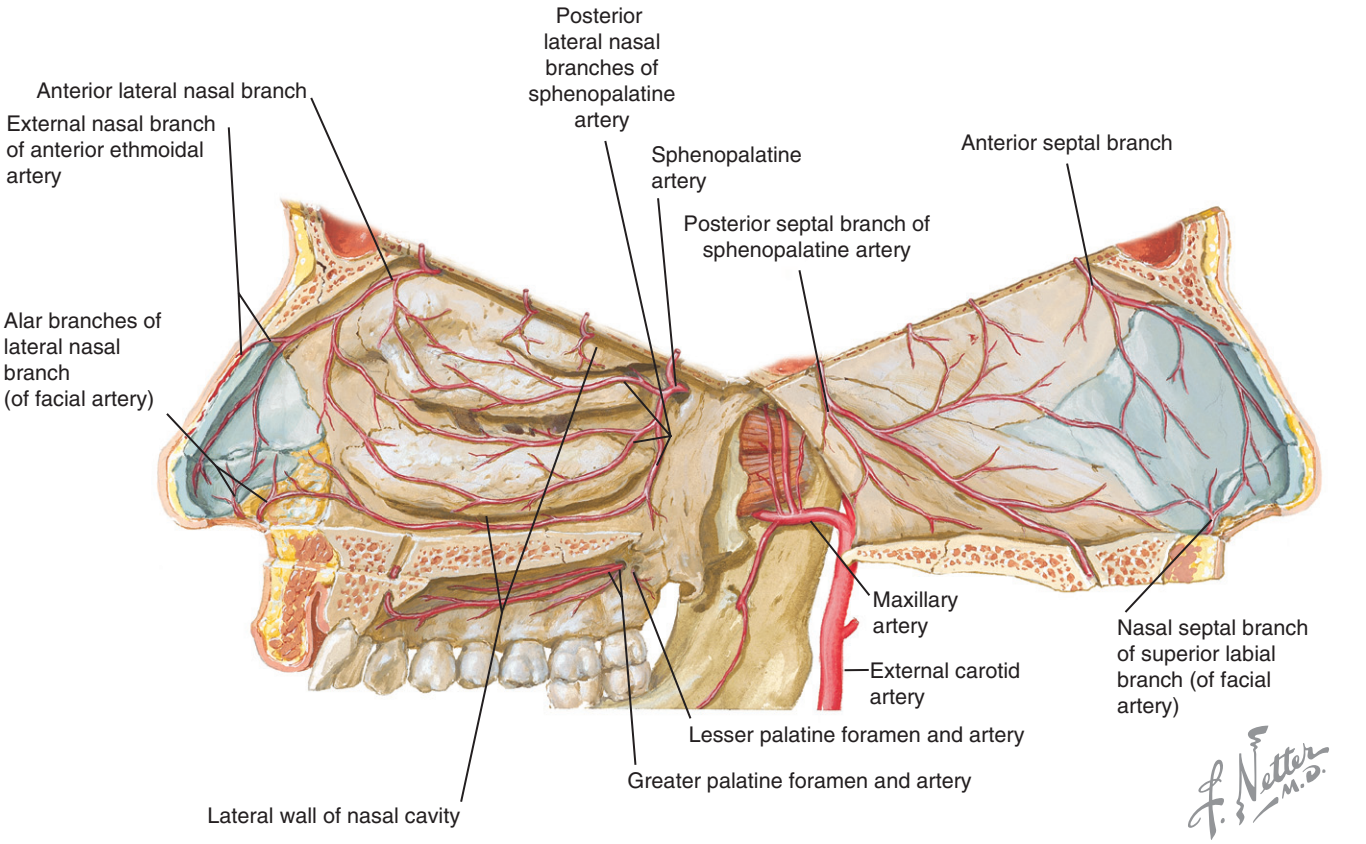


FIGURE 21-37 Blood supply to the lateral nasal complex. (Netter illustration from www.netterimages.com; © Elsevier Inc. All rights reserved.)

nasal branches of the infraorbital nerve. A standard infraorbital nerve block is also beneficial. Avoid solutions with vasoconstrictors injected near the septal cartilage so as not to compromise the blood supply.[150] Any intranasal mucosal laceration should be repaired and the nasal cavity packed to assist in preventing any post-traumatic nasal adhesions. Lacerations involving skin overlying the nose are common. Superficial lacerations can be treated with adhesive tape to prevent suture marks. Deeper lacerations require layered closure. Cartilage does not always require suture repair, but if it is unstable or has to be aligned, a fine absorbable suture, such as 5-0 fast-absorbing gut suture, is recommended. Use of a limited number of sutures in cartilage is desirable. Skin sutures should be removed from the nose in 3 to 5 days and adhesive tape should be used to maximize the cosmetic result.

Nasal bleeding from trauma usually stops spontaneously without requiring therapeutic intervention. On rare occasions, treatment may be necessary. Anterior epistaxis is more common than posterior nosebleed and usually involves hemorrhage from Kiesselbach's area, also referred to as Little's area (Fig. 21-37). Packing this area with cotton soaked in phenylephrine and 4% lidocaine will provide hemostasis and some topical anesthesia, and often will be effective. A nasal speculum should be used to visualize the areas of bleeding that persist and have to be cauterized. When using electrocautery or silver nitrate, caution must be exercised to avoid excessive cautery on both sides of the nasal septum because septal perforation or exposure of the cartilage may result. Cauterization with silver nitrate and packing usually stops most anterior nosebleeds. Placement of an anterior nasal pack, an 8.0-cm Merocel or 10-cm Pope nasal pack, may be useful if the bleeding persists. The most common method of anterior nasal packing is performed by the insertion of 0.5-inch petroleum jelly (Vaseline) gauze soaked in antibiotic ointment in a layered manner (Fig. 21-38). Careful packing will allow tamponade of the bleeding areas. This packing can be left in place for 2 to 5 days; the patient should receive broad-spectrum antibiotic coverage.

If the patient has a posterior nosebleed, these efforts will be useless. The most practical definition of a posterior nosebleed is epistaxis that cannot be treated with an anterior nasal pack. Inspection of the posterior pharynx often reveals bright red blood that the patient may be coughing up frequently. Posterior nasal packing is then indicated. There are several methods for packing a posterior nosebleed. A reliable method uses a Foley catheter. First, the nose is adequately anesthetized. As described earlier, the patient is sedated with an IV agent. Next, a Foley catheter is inserted into the offending naris until it is seen in the oropharynx. Approximately 10 mL of sterile water or normal saline is used to inflate the cuff of the Foley catheter, with firm tension applied on the catheter to secure it in the posterior nasopharynx. Next,

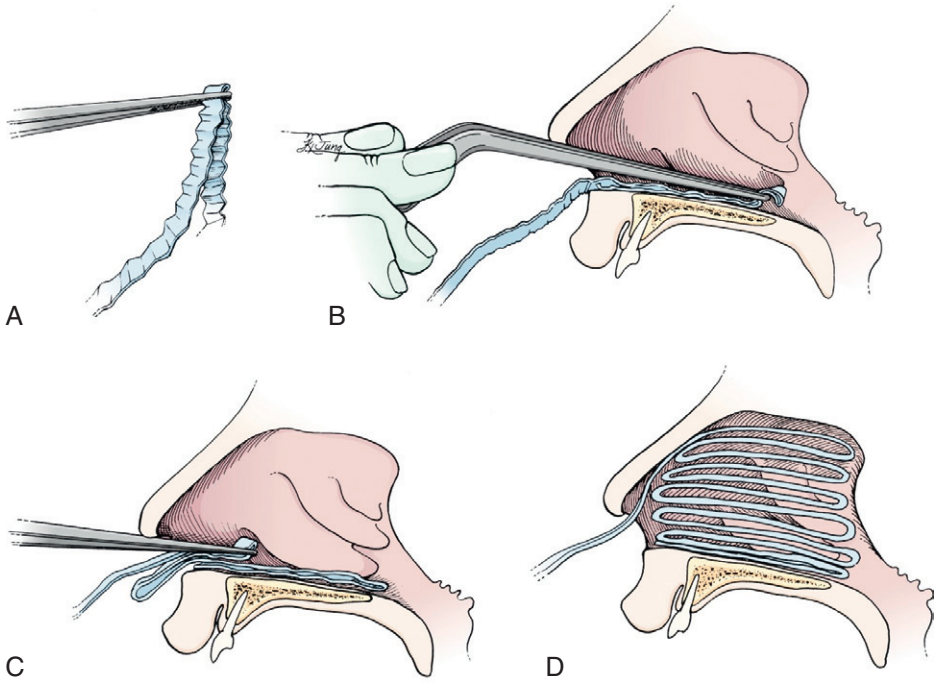


FIGURE 21-38 Correct technique for anterior packing of the nose. **A,** The gauze is gripped 4 to 6 cm from the end. **B,** The first layer is placed along the floor of the nose. **C, D,** Subsequent layering of the gauze packing. (From Roberts J, Hedges J: *Clinical procedures in emergency medicine*, ed 5, Saunders, Philadelphia, 2010.)

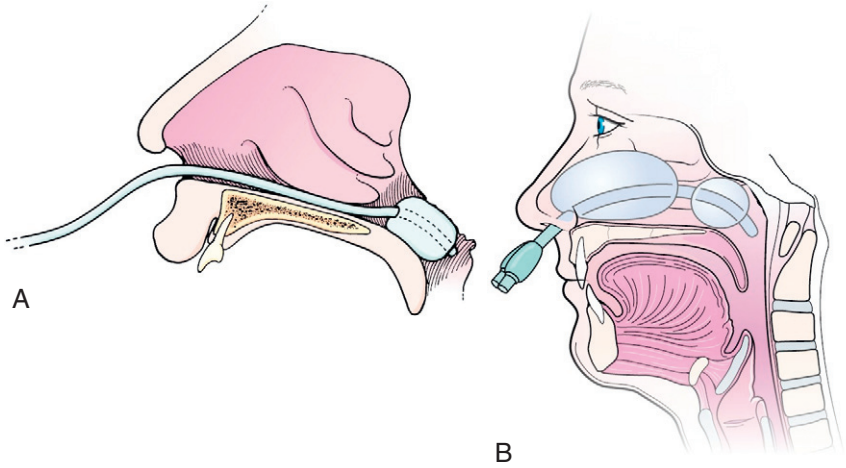


FIGURE 21-39 The Foley catheter is an effective tool used in the management of posterior epistaxis. **A,** It is placed in the nares and inflated to tamponade the posterior nasopharynx. The nose is then packed with gauze in a layered manner to provide adequate hemostasis. **B,** Another alternative is a commercially available packing device in which posterior and anterior balloons are used to pack a posterior nosebleed. (From Roberts J, Hedges J: *Clinical procedures in emergency medicine*, ed 5, Saunders, Philadelphia, 2010.)

0.5-inch petroleum jelly gauze is packed into the naris around the catheter. In the contralateral naris, 0.5-inch petroleum jelly gauze is packed to prevent septal trauma. An umbilical clamp is then used to secure the catheter in the desired position by applying it at the level of the entrance of the catheter into the nose (Fig. 21-39*A*).

The patient should be given IV antibiotics and necessary pain medication after admission. The balloon on the catheter may be decompressed in 3 days, allowing for assessment of hemorrhage. If successful hemostasis has been attained, the Foley catheter and packing can be removed before patient discharge. Another common method of posterior nasal packing is achieved with a silicone dual-cuffed catheter (Epistat [Medtronic, Minneapolis]). This double-balloon tampon is placed along the floor of the nasal cavity and a smaller posterior balloon is inflated with saline to occlude the nasopharynx. The larger anterior balloon is then inflated to create pressure to control hemostasis (see Fig. 24-39*B*). Although patients may find this method more uncomfortable, it is useful when emergent control of hemorrhage is necessary.[151]

EYEBROW

Reconstruction of the eyebrow is extremely difficult and transplants to the eyebrows are not always cosmetically satisfying[100] (Fig. 21-40). Therefore, efforts to repair eyebrows without resultant distortion or defects are



FIGURE 21-40 **A-C,** The eyebrow is maintained and the laceration is closed in an interrupted fashion, with good cosmetic result.

important. The eyebrow should not be shaved but should be lightly clipped if necessary to assist the surgeon in proper alignment of the eyebrow. The wound should be inspected and underlying fractures of the frontal sinus or supraorbital rim repaired before closure. As little tissue as possible should be removed and sutured into place. If nonvital tissue must be removed, incisions should be made parallel to the hair follicles to injure as few as possible. Special care should also be taken to avoid tight constricting sutures in the area, because hair follicles are sensitive to decreases in blood flow.[5,145]

The muscular layer should be closed with fine absorbable sutures to prevent spreading of the tissue and scar formation. The skin should be approximated with 6-0 nylon sutures. Vertical displacements that may develop can usually be corrected with a Z-plasty procedure, and horizontal displacements can be corrected with scar revision and realignment of the parts. These procedures should be performed 6 to 12 months following the accident, after the scar tissue has softened.[152]

EYELID

In the treatment of injuries to the eyelid, it is important to restore not only the appearance of the individual but also, and more importantly, the vital function of the structure. The major function of the eyelid is to protect the globe and prevent drying of the cornea and adjacent tissue. Eyelids aid in removal of tears through the canalicular system. With any type of injury to the orbit, eyelids, and globe, an ophthalmology consultation is mandatory to assess for global injury and defects in vision. Even if no defects are initially apparent, early baseline records are necessary.

As in any surgical procedure, a detailed understanding of the anatomy of the region is necessary to assist in the proper repair of traumatic injuries to the eyelids. In order, from the skin to the conjunctival layer, the eyelids are composed of the skin, alveolar tissue, orbicularis oculi muscle, tarsus, septum orbitale, tarsal (meibomian) glands, and conjunctiva.[153] The lid margin is formed by the junction of the skin and mucous membrane and is delineated by a gray line. The superior tarsus is of a semilunar shape to conform to the configuration of the globe; it assists in keeping the conjunctival mucosa intact with the cornea. The tarsal plates are long thin plates of connective tissue that also aid in maintaining the form and support of the eyelid.[154]

The levator muscle inserts into the skin of the upper lid and upper margin of the tarsus and is responsible for elevation of the upper lid (Fig. 21-41). The muscular layer over the tarsus is also anchored to the medial and lateral aspects of the orbit by the medial and lateral canthal ligaments. The orbital septum is peripheral to the tarsus and forms a fibrous sheet attached to the periosteum about the circumference of the orbital rim. The septum maintains the orbital contents in the proper position.[153,154]

Lacerations of the eyelids can be divided into two categories, wounds that involve the lid margin and those that do not. Simple lacerations that do not involve the



FIGURE 21-41 **A, B,** Injury to the upper eyelid. Disruption of the upper lid margin disrupts the superior tarsal plates, which are long thin plates of connective tissue that also aid in maintaining the form and support of the eyelid. The levator muscle inserts into the skin of the upper lid and the upper margin of the tarsus and is responsible for elevation of the upper lid. **C, D,** Lacerations of the upper eyelid must be explored to identify damage to the levator muscle. At the point at which the levator attaches to the superior portion of the tarsus, an upper lid fold is normally created. If the fold is violated, it should be restored by repair of the muscle-tarsus junction and suture of the subcutaneous layer to the deep structures. Sutures should be removed in 48 to 72 hours to prevent suture tracks of epithelium.

margin should be closed primarily. Following evaluation for possible injury to the orbit, globe, and punctal and canalicular systems, débridement with minimum tissue removal should be accomplished. Lacerations should be closed in layers, restoring the integrity and orientation of the skin, muscle, tarsal, and conjunctival layers. Deep sutures are not recommended in the lower eyelid because the orbital septum may be inadvertently sutured, creating a cicatricial ectropion as the wound heals.[136] Lacerations of the upper eyelid must be explored to identify damage to the levator muscle. At the point at which the levator attaches to the superior portion of the tarsus, an upper lid fold is normally created. If the fold is violated, it should be restored by repair of the muscle-tarsus junction and suture of the subcutaneous layer to the deep structures. Sutures should be removed in 48 to 72 hours to prevent suture tracks of epithelium.[153]

Marginal lacerations must be repaired carefully and accurately to prevent functional and cosmetic defects. The most common identifiable structures are the lash line, meibomian gland orifices, and gray line (junction between the conjunctival mucosa and skin).[136] Three 6-0 nylon sutures should be placed at the marginal rim to align and properly orient these structures on either side of the laceration. The sutures should not be tied. Fine absorbable sutures are placed to close the fascial border, but no other deep sutures are placed because of the risk of ectropion in the lower lid. A slight eversion of the lid margin must be obtained with the marginal sutures to allow for wound contraction of the lid margin. The transmarginal sutures at the gray line and lash line are left long and are secured to the skin surface to prevent corneal abrasion.[153]

Avulsive injuries to the eyelids are treated with full-thickness skin grafts from the postauricular region or the other upper eyelid.[154] With avulsive injuries of the lid margins, carefully placed pedicled tissue will usually be maintained because of the excellent blood supply in the region. Full-thickness eyelid avulsions of less than 25% of the lid length can be approximated primarily as a simple laceration.[5] Lateral canthotomy to dissect the skin and conjunctiva free from the lateral canthal tendon,

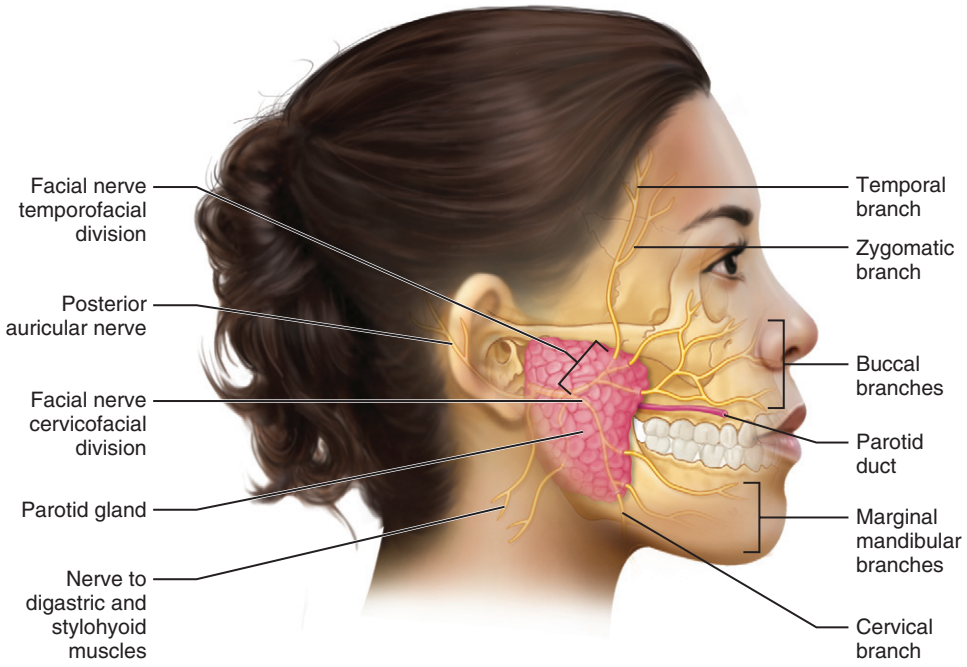


FIGURE 21-42 Lacerations to the parotid duct frequently damage the buccal branch of the facial nerve because of the close approximation of the two structures.

free all structures between the conjunctiva and the skin, and allow for release of horizontal tension can be used. Ordinarily, 5 to 10 mm may be gained in lid length in the horizontal direction.[153] Larger defects require grafts or flaps, such as an Abbe-type rotational flap from the unaffected eyelid.[136]

Injuries to the conjunctiva require no treatment if they are small. Large lacerations or punctures may require fine absorbable sutures to control the tissue. Corneal abrasions are commonly associated with facial trauma. Pain and irritation of the injured eye with a sensation of a foreign body present in the eye are common complaints. Fluorescein dye testing and slit-lamp examination will confirm the damage to the cornea. Treatment with cycloplegics, ophthalmic antibiotic ointments that do not contain corticosteroids, and patching usually relieves discomfort while the injury heals. Because of rare but devastating infections (e.g., *Pseudomonas*), patients with corneal abrasions should be referred to an ophthalmologist within 24 hours.

Povidone-iodine (Betadine) solutions should be used to prepare the wound for closure of lid lacerations. Tissue débridement is discouraged. The use of 6-0 silk or fast-absorbing 6-0 gut sutures at the lid margin and skin and 7-0 Vicryl interrupted sutures at the tarsal plate is recommended.[155] A transverse laceration of the upper lid may completely sever the levator aponeurosis and Müller's muscle from their attachments to the tarsal plate. A profound ptosis with minimal levator function will appear. The wound has to be inspected with the patient under local anesthesia; in most patients, the orbital septum has also been violated. If it has not, the septum should be incised and orbital fat allowed to prolapse forward. Proper identification of orbital fat is important for establishing a landmark. The fat is retracted superiorly and the underlying aponeurosis identified. The patient should be instructed to look up and down, because this maneuver will aid in identification of the structure. The aponeurosis should be reapproximated to the distal cut edge or tarsal plate with interrupted 5-0 Vicryl sutures in a horizontal mattress fashion. Lacerated skin edges are then reapproximated with 7-0 nylon or 6-0 fast-absorbing gut sutures. No attempt should be made to close the orbital septum.

ORAL MUCOSA AND TONGUE

Lacerations of the oral mucosa and tongue should be inspected, especially for pieces of teeth or restorations, and débrided as for other wounds. The wounds should be thoroughly irrigated with normal saline and sutured loosely. Mucosal wounds should be sutured with 3-0 or 4-0 chromic gut suture. Deep lacerations should be closed in layers, with chromic gut sutures in the muscle layers to prevent formation of a hematoma. The tongue should be closed in layers, with 4-0 Vicryl (dyed) sutures in the superficial layers.

The tongue has a rich blood supply and injuries to the tongue or the floor of the mouth may cause serious hemorrhage that could threatens the airway. The airway may become compromised some time after trauma to the tongue or lacerations of the floor of the mouth if veins are damaged, resulting in swelling of the tongue into the oropharynx.[108,110]

SALIVARY GLANDS AND DUCTS

With injuries to the parotid area, an understanding of the anatomy is essential to proper treatment (Figs. 21-42 and 21-43). The facial nerve exits the stylomastoid foramen, where it divides into five branches within the substance of the gland. The temporal and zygomatic branches run over the zygomatic arch, the buccal branch

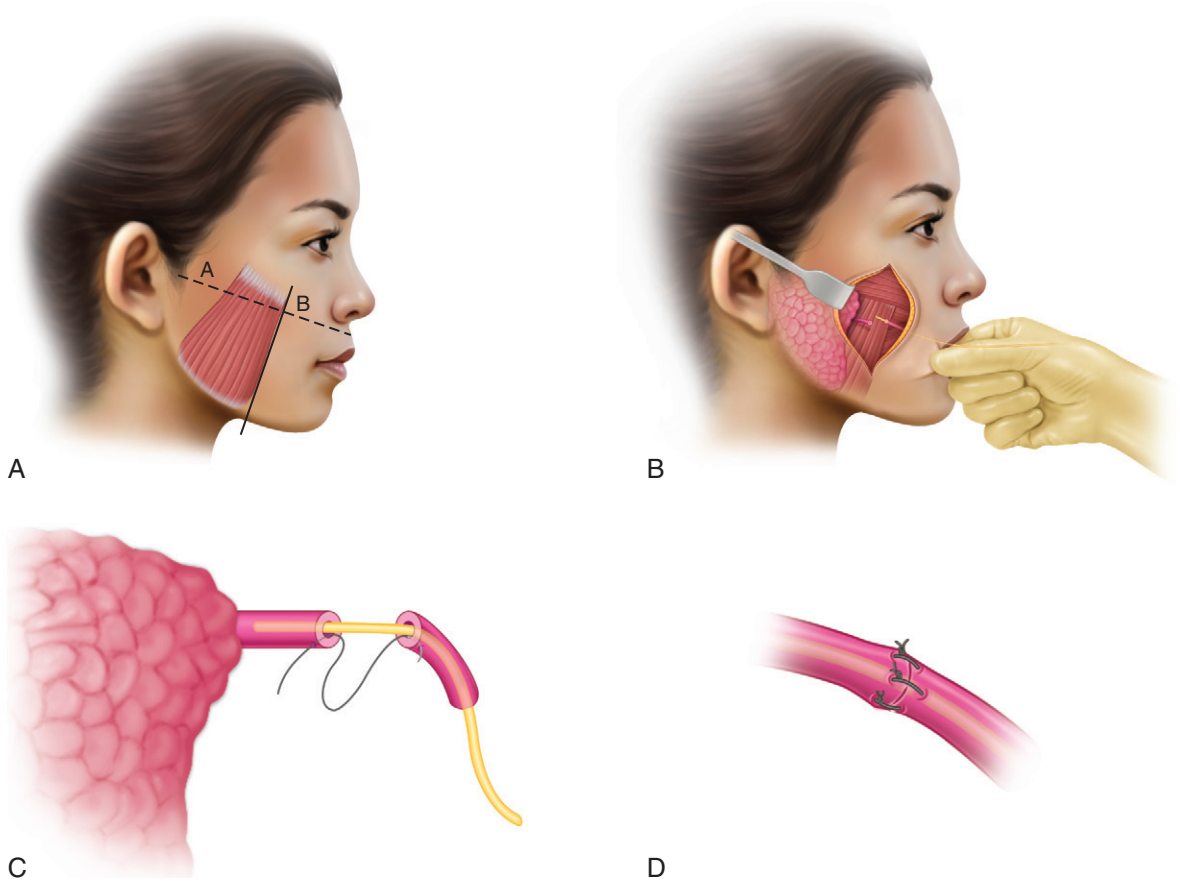


FIGURE 21-43 The parotid duct is typically found along the plane from the tragus of the ear to the middle of the upper lip.

courses over the superficial aspect of the masseter muscle along with the parotid duct, the mandibular branch crosses superficially to the facial vessels at the angle of the mandible, and the cervical branch runs down the neck. The parotid gland is a single-lobed gland, with superficial and deep portions of the gland determined by their relationship with the seventh cranial nerve. The superficial part of the gland is lateral to the facial nerve and extends anteriorly to the border of the masseter muscle. The deep portion, which comprises approximately 20% of the gland, lies medial to the nerve in the retromandibular fossa. The parotid duct exits the gland anteriorly, runs along the superficial portion of the masseter muscle, and penetrates the buccinator to enter the oral cavity opposite the upper second molar. The presence of multiple structures in such a small region explains the high morbidity associated with these injuries. Treatment of a parotid duct injury depends on the site of the injury. If the injury is anterior to the masseter and the distal portion of the duct cannot be located, the duct may be drained directly into the mouth. If the injury is over the masseter muscle, the distal and proximal portions may be connected using a stent. If the injury is within the parotid gland, treatment should include closure of the parotid capsule and application of a pressure dressing.

Injuries distal to the parotid gland and medial to the lateral canthus of the eye rarely result in severe disfigurement. A rich anastomotic network of the branches of the nerve allows frequent return of function in this area. Repair of the forehead and mandibular branches should be considered because cross innervation in these areas is less predictable.

Injuries to the parotid or submandibular gland should be evaluated and repaired, if possible. Injuries to the parotid or submandibular ducts must also be assessed. If the duct has been transected, repair around a thin polyethylene tube is necessary. From the anterior border of the gland, the parotid duct extends forward approximately 1 cm below the zygoma. The location of the duct on the face may be visualized as the middle third of a line from the tragus of the ear to the middle of the upper lip. The duct is approximately 4 to 6 cm in length and 5 mm in diameter. The parotid (Stensen) duct runs transversely through the buccinator muscle to empty into the oral cavity at the buccal mucosa, directly across from the maxillary second molar. Lacerations involving the parotid duct frequently damage the buccal branch of the facial nerve because of close approximation of the two structures.

When the parotid duct has been lacerated, both ends of the duct must be located and sutured together. The distal portion of the severed duct is usually located first by placing a lacrimal duct probe or polyethylene cannula through the Stensen's duct orifice in the mucosal wall of the oral cavity, just lateral to the second maxillary molar,

and passing the probe through the laceration site. The proximal segment can then be located by expressing saliva from the parotid gland. A Silastic catheter should be placed through the severed segments and repair should be made over the catheter. The repair should be done with 6-0 nylon interrupted sutures. Approximately 2 cm of stent should extend from the orifice, and the stent should be secured to the oral mucosa with one or two nonabsorbable sutures. The Silastic stent should remain in place for 5 to 7 days and the patient should be given sialagogues, such as lemon drops, to prevent scar formation at the anastomosis site.[19,136]

After repair of the duct, the overlying soft tissue is closed in layers to prevent formation of a fistulous tract and a sialocele. If a sialocele does form, it should be treated with aspiration and a pressure dressing over the area to eliminate fluid collection.

A swelling over the course of the duct that slowly increases in size after trauma to the area may be suggestive of an injury to the duct that was not detected during the initial examination. If the diagnosis is made within 48 hours of the injury, an exploratory operation with repair is indicated.[19] With injury to the salivary ductal system, prophylactic antibiotics, such as penicillin or cephalothin, should be used.[136]

The submandibular duct runs laterally and superiorly from the gland to its orifice in the oral cavity behind the mandibular incisors. The mandibular duct is approximately 5 cm long and courses near the lingual nerve before the nerve enters the tongue. A small polyethylene catheter is placed in the orifice and through the distal segment. The proximal segment is located by massage of the gland, so that saliva from the transected duct is found. The catheter is then advanced into the proximal segment. Several 4-0 chromic sutures should be placed to secure the transected ends of the duct about the catheter. The catheter should then be cut in the oral cavity and secured to the mucosal tissue by sutures. The catheter should remain in place for 5 to 7 days and removed only after ensuring that the duct will remain patent without the catheter.

Lacerations of the parotid gland that occur without major ductal lacerations do not require any special treatment, other than the routine management and repair of the soft tissue injury. Drain placement, however, may be necessary. Permanent fistulas are rare in parotid glandular lacerations in the absence of major duct involvement. Lacerations of the gland frequently result in delayed fluid accumulation, even after drain removal, and the fluid collection may be treated with intermittent aspiration, compression, and reinsertion of a drain.[156,157]

If the parotid duct cannot be repaired after traumatic laceration, several treatment choices are available. Ligation of the duct is an option that will produce a temporary swelling and may develop as a chronic source of infection. If possible, the proximal duct stump should be mobilized and diverted into the mouth in the oropharynx. Irradiation of the gland to destroy its function is a last resort. All these options are less desirable than primary repair.

Repair of a lacerated submandibular duct is usually unnecessary because a fistula into the mouth usually develops and allows the duct to drain. Scarring with obstruction of the duct may eventually require reestablishment of the duct opening. Chronic obstruction or inflammation is usually best treated with removal of the submandibular gland.

LACRIMAL APPARATUS

Tears produced by the lacrimal gland drain across the surface of the cornea to the medial portion of the eye, where they enter the puncta of the upper and lower lid margins and proceed to the canaliculi in the nasolacrimal apparatus. The tears then drain into the inferior meatus of the nose. More than 50% of the tear drainage volume is normally evacuated through the inferior canaliculus; when this pathway is traumatically interrupted, it is important that it be repaired, when possible (Fig. 21-44).

Any lacerations of the medial third of the lower lid should immediately raise the suspicion of injury to the inferior canaliculus. Establishing hemostasis of the laceration is mandatory for finding the injury. The canaliculus is a fairly large white-walled tube and may be located by placing a lacrimal duct probe through the punctum and into the wound. The canaliculus begins at the punctum and proceeds perpendicular to the eyelid margin for approximately 2 mm and then turns medially and proceeds to the nasolacrimal apparatus. Magnifying loupes of 2× or 3× power should be available to help locate the lacerated ends of the canaliculus. Another method of locating the lacerated canaliculus is to infiltrate the upper canaliculus with air and instill sterile water or saline solution into the laceration line, allowing the liquid to pool. Air will pass through the canalicular apparatus and bubble through the saline or water, demonstrating the site of the laceration.

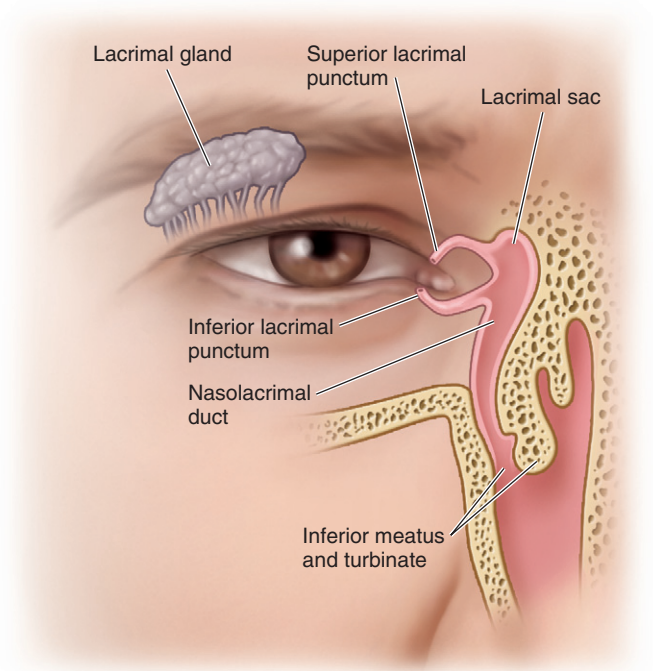


FIGURE 21-44 Anatomy of the nasolacrimal system.

When lacerated ends of the canaliculus have been located, they can be repaired using a Veirs stainless steel rod wedged onto black silk. The rod is passed through the punctum into the laceration site and then into the medial portion of the canaliculus to align the cut ends. The laceration is stabilized with small chromic gut sutures and the rod is left in place for 4 to 6 weeks. The free end of the silk material of the Veirs rod is tied into place to help stabilize the rod and is used to retrieve it at the time of removal.

Through and through margin injuries to the eyelid must be repaired in at least three layers to prevent notching as healing progresses. The deep layer contains the conjunctiva and tarsus and should be closed with 4-0 or 5-0 chromic gut interrupted sutures; the knots should be tied into the wound so that they do not irritate the cornea. The middle layer is the orbicularis oculi muscle, which is closed with interrupted 5-0 chromic gut sutures, and then the skin is finally closed. Great care should be taken to approximate the tarsus and ciliary margin accurately. Once these structures have been sutured, the remainder of the eyelid can generally be properly repaired without difficulty. Any laceration involving the medial portion of the upper or lower eyelid should be presumed to involve the lacrimal canaliculi until proved otherwise. Because the dense fibrous tissue of the tarsal plate is notably stronger than the medial canthal tendon, an avulsing force placed anywhere along the lid margin will preferentially tear the medial soft tissue, causing disruption of the lacrimal apparatus.

SCALP

The scalp and forehead are portions of the same highly vascularized anatomic unit responsible for protection of the skull (Fig. 21-45). They consist of five layers, which can best be remembered by the mnemonic SCALP (Fig. 21-46). In order, from the skin to the cranial bone, the layers are skin (S), subcutaneous tissue (C), aponeurosis layer (A), loose subepicranial space (L), and pericranial layer (P).[32]

The thickness of the epidermis and dermis of the scalp varies from 4 to 8 mm, so the scalp is one of the thicker regions of the body; only the back, soles, and palms are

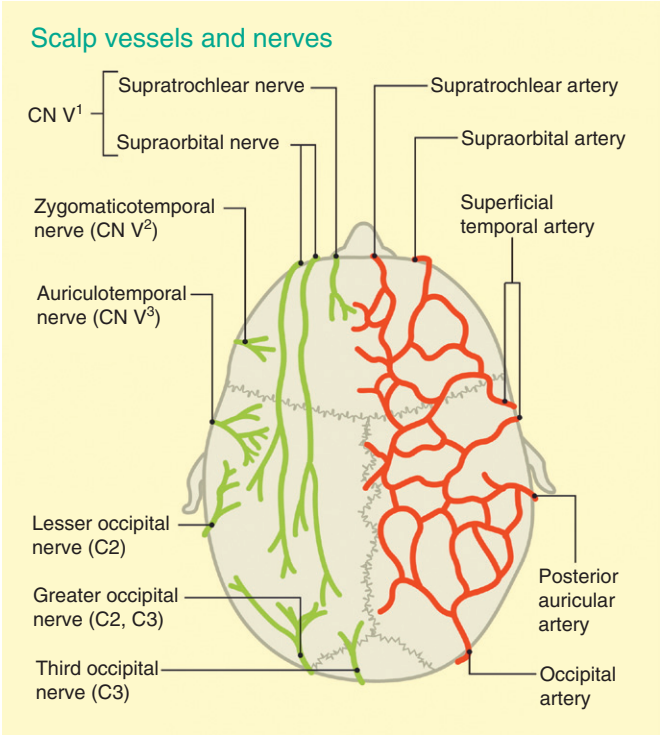
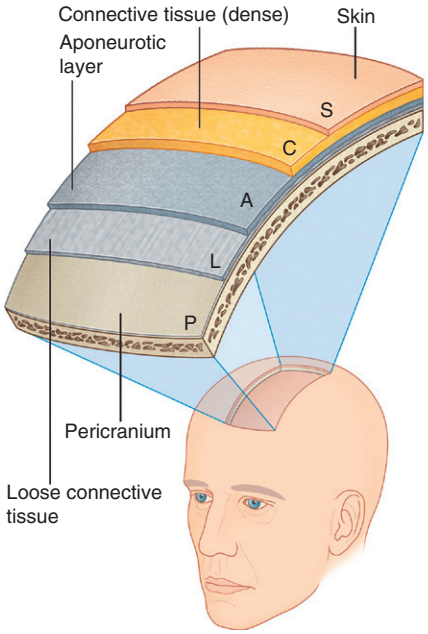


FIGURE 21-45 Approximate location of the arteries and sensory nerves that course through the dense connective tissue layer of the scalp. (From Ellis H: Anatomy of head injury. *Surgery (Oxford)* 25:505, 2007.)



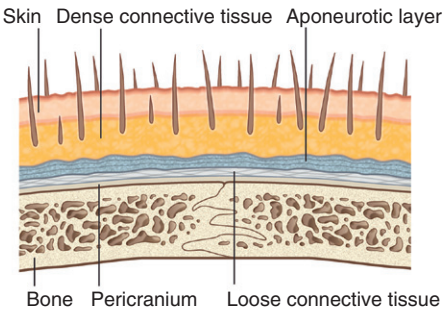


FIGURE 21-46 The layers of the scalp can be remembered by the mnemonic SCALP: Skin, Connective tissue, Aponeurotic layer, Loose connective tissue, and Periosteal or Pericranial layer. (From Aehlert BJ: *Paramedic practice today*, St. Louis, 2009, Elsevier.)

thicker.[32] The subcutaneous layer has many large vessels that anastomose freely. Because this layer is so inelastic, the blood vessels minimally contract when severed and tend to bleed easily. Thus, large amounts of blood can quickly be lost.[99]

The aponeurosis layer consists of the galea aponeurotica, which connects the paired occipital muscles and paired frontal muscles. The galea is a tough inelastic tissue that is attached firmly to the skin and subcutaneous layers and moves freely over the subepicranial space below.[19] The subepicranial space lies between the galea aponeurotica and pericranium. It is traversed by small arteries and emissary veins connecting the scalp veins with the venous sinuses of the skull. Infections and thrombosis in this layer may be passed to the cranium or sinuses via these vessels.[32]

The pericranium is very vascular and can be easily stripped from the cranium. The pericranium will accept a free graft readily because of its excellent vascularity. The outer table of the skull will not accept a free graft, so the pericranium is therefore extremely important in any avulsive injury to the scalp.[19] Avulsed scalp flaps are replaced if the tissue is not badly damaged, and most survive. Free graft survival depends on the presence of the pericranium over the skull.

Injuries to the scalp should be evaluated for possible accompanying skull fractures and intracranial trauma. A simple laceration should be treated like other lacerations on the face, with evaluation, hemostasis, débridement, and primary closure. It is unnecessary to shave the hair from the region, but scissors should be used to trim the hair in the area around the laceration. Closure is easy if the scalp defect is less than 2.5 cm wide. If larger defects are encountered, flaps should be used. (Fig. 21-47).[116]

With avulsion of the skin of the scalp, the examiner must not only carefully ascertain the status of the pericranium, but also look for evidence of fractures of the cranium. As with other avulsive defects of the face, healing by secondary granulation must be prevented because of the cosmetic and functional defects that result from excess scar formation and contracture. The scalp offers unique challenges because the cranial bones depend on the pericranial tissue for their blood supply.[32] The scalp has an excellent blood supply in the subcutaneous tissue and pericranial layers that support avulsed tissue, skin grafts, and various flaps.

If the pericranial tissue is intact and the cranium has not been fractured, defects in the scalp that cannot be closed primarily should be covered with a split-thickness skin graft. After stabilization and healing of the defect, the area can be reconstructed by various advancement or rotational flaps to bring similar tissue into the defect, with or without atraumatic tissue expansion. Tissue expansion is an alternative for the closure of wound defects because it provides donor tissue of the same color, texture, and thickness, with minimal scar formation and minor donor site morbidity.[98] Tissue expansion involves developing donor tissue without depriving the donor site of tissue.

If the pericranium has been lost, the exposed cortical cranial bone will not support a skin graft. When bone is exposed in large avulsive injuries of the scalp, primary closure with flap procedures is indicated.[19] The flap procedures used with scalp defects include advancement flaps, transpositional flaps, rotation-advancement flaps, and microvascular free scalp flaps.[13,99]

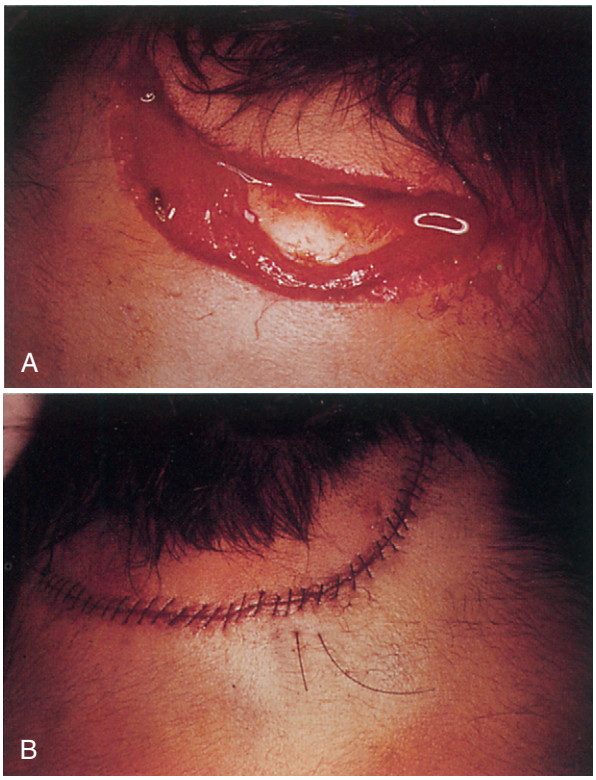


FIGURE 21-47 **A, B,** Oblique laceration through the scalp on the forehead, with the pericranium intact. The laceration is closed primarily in layers.

Hair Apposition Technique

A technique of treating scalp lacerations has been termed the *hair apposition technique* (HAT).[158] After standard cleaning and débridement, hair on both sides of a laceration is apposed with a single twist. It is then held into position with tissue adhesives. Severely contaminated wounds, actively bleeding wounds, and patients with hair strands shorter than 3 cm may not be suitable for this technique. In comparison with standard suturing, patients were found to have less scarring, lower pain scores, shorter procedure times, less wound breakdown, fewer overall complications, and greater satisfaction.[159] The authors concluded that HAT is equally acceptable and perhaps superior to standard suturing for closing suitable scalp lacerations. This procedure also avoids clipping or shaving of hair (to be avoided to limit infection rate) to improve visualization and access for suturing. However, in full-thickness wounds, only the superficial margin of the skin may be apposed. There is potential for creation of dead space, which can increase complications such as infection and wound breakdown. Also, there may be a temptation to avoid the use of local anesthetics, which may limit adequate cleaning and débridement.[160] In conclusion, in small superficial scalp wounds in the appropriate clinical situation, the HAT technique may be desirable.

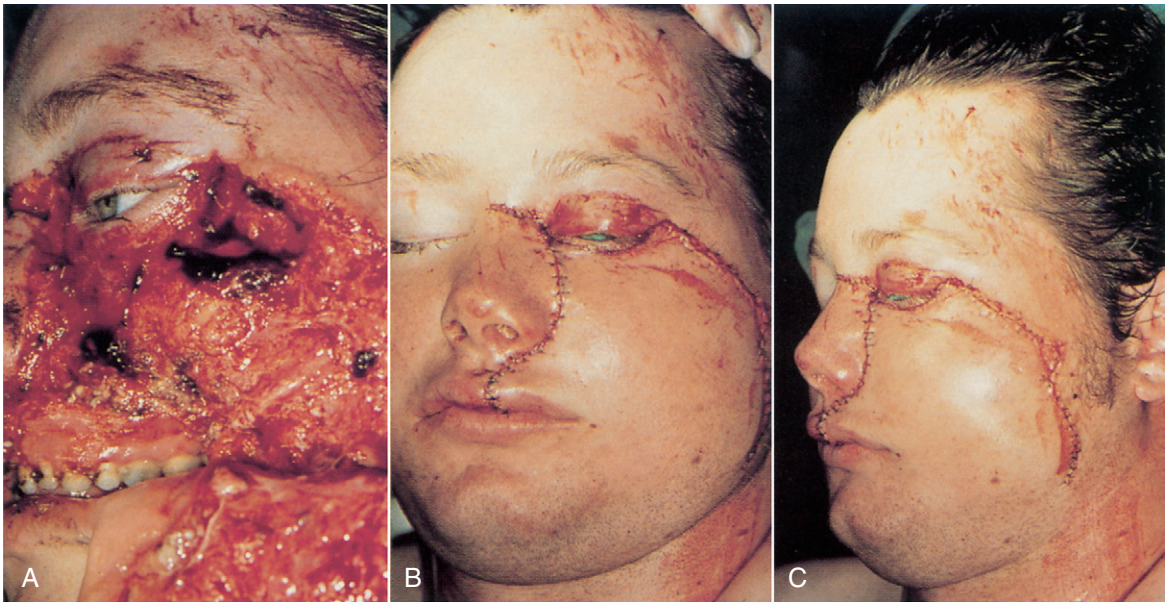


FIGURE 21-48 **A-C,** Laceration involving the eyelid, cheek, and intraoral structures. The laceration is sutured from the inside out. Intraoral closure should take place before the extraoral facial closure. The lacrimal apparatus is identified and repaired about a Silastic catheter.

Advancement Flaps

Advancement of the scalp into defect wounds is difficult because the galea aponeurotica is thick and not very elastic and does not stretch under traction. The flap must be widely undermined in the subepicranial space; multiple longitudinal incisions are made in the galea parallel to the skin margin to facilitate closure of the defect. Split-thickness skin grafts should then be placed over the exposed pericranium.

Transpositional Flaps

Transpositional flaps involve the transfer of tissue from one section to the defect, with acceptance of the fact that a secondary defect will be left in the donor area.[19] The donor area is usually in a less aesthetic region, such as the occipital area of the head, and is covered with a skin graft.

Rotation-Advancement Flaps

A rotation-advancement of local flaps to cover defects is the treatment of choice when the pericranium has been destroyed.[21] To assist with stretching of the tissue, the galea should be released with multiple incisions parallel to the longer axis of the flaps. Orticochea[116] has devised a technique for closing cutaneous defects of the scalp with a three-flap procedure that mobilizes large flaps based on the superficial temporal, occipital, posterior auricular, and supraorbital arteries, which supply blood to the scalp.[99] These studies described the cutaneous covering of the skull and used five different applications of the three-flap rotation-advancement technique for reconstruction of the forehead, right temporal region, left temporal region, central region of the skull, and nape of the neck.

Microvascular Flaps

In 1972, McLean and Buncke described a microsurgical reconstruction of a bare cranial defect with a free omental transplant anastomosed to the superficial temporal artery and vein and covered with a free split-thickness skin graft, and Ohmori has described the use of a microsurgical free scalp transfer, which permits the grafted scalp to have a natural hair direction at the recipient site.[21] Four types of free flaps based on the superficial temporal, posterior auricular, and occipital vessels have been described. Reconstruction with microvascular free flaps is seldom indicated in emergency situations.

If the pericranium has been destroyed and small scalp defects cannot be covered with local flaps, burr holes should be made through the outer table until pinpoint bleeding is found in the bone. A split-thickness skin graft can be placed over the bleeding bone. If the grafted skin is lost or the outer table of cranial bone undergoes necrosis, an osteotome or burr should be used to expose the diploe and a split-thickness skin graft should be reapplied.[21]

SCAR FORMATION

Hypertrophic contracted scars are produced when inflammation is present in healing wounds (Figs. 21-48 and 21-49). The main factors responsible for inflammation within wound surfaces are contamination, foreign bodies, hematoma formation, crush injury, devitalized necrotic tissue, dead space, and operative trauma[99] (Table 21-6). Hypertrophic scars are often mistaken for keloids (Table 21-7). Because they are histologically indistinguishable, the clinical picture and medical history are the only clues to diagnosis. Keloids are true benign tumors that extend into skin that was not involved in the original wound. Keloids are less common than hypertrophic scars. They are the result of an overactive production of fibrous tissue during wound healing, perhaps caused by increased tension around the wound margins. Keloids are defined as scars within

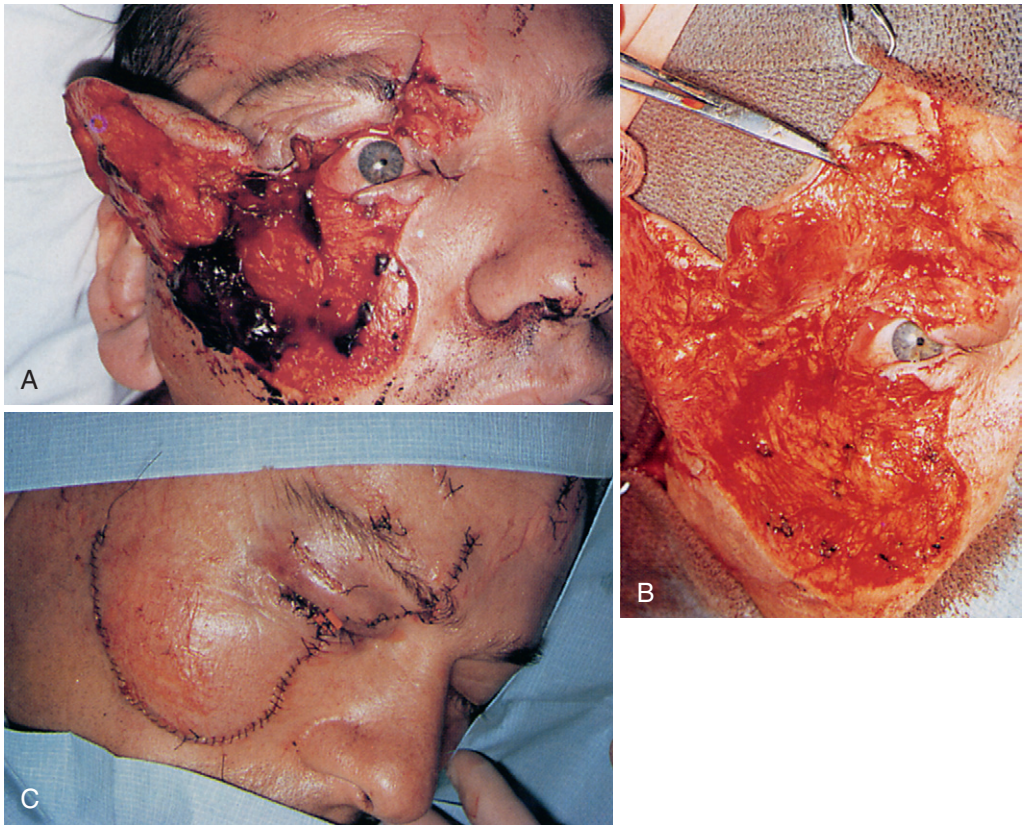


FIGURE 21-49 **A-C,** Significant laceration to the eyelid and cheek region. Careful débridement and examination must be done to identify important structures such as the parotid duct and facial nerve. Structures must be repaired primarily and the wound closed in layers.

TABLE 21-6 Factors That Increase Scarring and Compromise Wound Healing

Contributing Factors	Methods to Minimize Scarring
Direction of wound (i.e., perpendicular to lines of static and dynamic skin tension)	Layered closure; proper direction in elective incisions of wound
Infection necessitating removal of sutures, resulting in healing by secondary intention and a wide scar	Proper wound preparation; irrigation, débridement, and use of delayed closure in contraindicated wounds
Wide scar secondary to tension	Layered closure; proper splinting and elevation
Suture marks	Remove all pericuticular sutures within 7 days.
Uneven wound edges, resulting in magnification of shadows	Careful, even approximation of scar by wound edges and top layer closure to prevent differential swelling of edges
Inversion of wound edges	Meticulous placement of sutures or use of horizontal mattress sutures
Tattooing secondary to retained dirt or foreign body	Proper wound preparation and débridement
Tissue necrosis	Use of corner sutures on flaps, splinting, and elevation of wounds with marginal circulation or venous return; excise nonviable wound edges before closure
Compromised healing secondary to hematoma	Use of proper conforming dressing and splints
Hyperpigmentation of scar or abraded skin	Use of no. 15 or higher SPF sunblock for 6 mo
Superimposition of blood clots between healing wound edges	Proper hemostasis and closure; H_2O_2 frequent swabbing; proper application of compressive dressings
Failure to align anatomic structures properly, such vermilion border	Meticulous closure and alignment; marking as or placement of alignment suture before distortion of wound edges with local anesthetic; use of field block

TABLE 21-7 Comparison of Abnormal Scar Formation

	Keloid	Hypertrophic Scar	Widespread Scar
Genetics	Significant familial predilection	Less familial incidence	Not inherited
Race	Blacks more than whites	Less race-related	Not ethnic
Sex	Females more than males	Equal gender ratio	Unknown
Age	Most common at 10-30 yr of age	Any, but mostly <20 yr	Any age
Borders	Outgrows its boundaries	Remains within wound boundaries	Wide, flat, often depressed
Natural history	Months after injury, rarely subsides	Soon after injury, subsides with time	Within 6 mo of injury
Location	Mostly face, earlobe, anterior chest	Across flexor surfaces	Arms, legs, and abdomen
Etiology	Possible autoimmune phenomenon	Tension and timing of closure	Tension and mobility of edges
Treatment	Often worse after surgery	Improves with correct surgery and/or pressure	Scar excision or layered closure

Adapted from Brody GS, Peng ST, Landel RF: The etiology of hypertrophic scar contracture: Another view. *Plast Reconstr Surg* 67:678, 1981.

the skin that grow beyond the confines of the original wound and tend to be darker than the normal skin, whereas hypertrophic scars are raised scars that remain within the boundaries of the wound.[90] Certain individuals are predisposed to keloid formation and the patient usually can report a history of keloid formation. Keloids occur more frequently in younger age groups in dark-skinned individuals and in areas of thick skin rather than in areas in which the skin is thin. The incidence of keloids in dark-skinned persons is estimated to be 15 to 20 times that of light-skinned individuals.

An abnormality in melanocyte-stimulating hormone (MSH) may be responsible. Keloid growth is increased during puberty and pregnancy, when production of MSH is increased. Infection may also promote the development of keloids at sites of skin injury. A family predisposition is apparent; however, the exact cause of keloids is unknown. Patients who tend to form keloids usually produce reactions at other sites, such as vaccination sites. Keloids generally form in areas of increased skin tension in individuals.

Approximately 12 hours following injury, epithelial migration begins. In primary closed wounds, complete epithelialization can occur within 48 hours. If wound margins are open, secondary repair begins with migration of epithelial cells from the margin. Epithelial cells will not cover necrotic tissue or highly inflamed tissue. Epithelial cells can migrate from 1 to 3 cm, but closure occurs after 5 to 7 days by the process of contracture of the wound margins.[161]

The contracture of facial wounds should be prevented because the resultant scar usually leaves a considerable functional and cosmetic defect. Wound contracture is caused by fibroblasts that migrate to the wound margin from surrounding tissue and rapidly synthesize collagen at the wound margin. Rapid collagen synthesis lasts 2 to 4 weeks. The immature scar tissue is usually irregular, raised, and purplish because of the increased number and size of capillaries, collagen, and fibroblasts. The final phase of wound repair, the maturation phase, involves resorption of excess collagen, decreased number of fibroblasts, and a decrease in the number or size of capillaries. This phase may last for 6 months to 2 years and, during this phase, the scar contracts and widens and may undergo hypertrophy or form a keloid[3,162-164] (Fig. 21-50).

Hypertrophic scars are usually self-limited and, over time, soften, fade, and flatten. Scars should be revised secondarily only after they have undergone maximal maturation for as long as 12 months.[3,165] If a scar crosses the favorable lines of tension lines that follow underlying muscle contraction on the face, tension on the skin wound margins will tend to pull the wound margins apart. Poor operative technique is generally the cause of hypertrophic scars. Uneven closure of wounds, failure to close the wound in layers and relieve tension at the wound margin, and improper débridement and preparation of the wound before closure all result in excessive scar formation.[5]

In general, wounds that are within favorable lines on the face heal without incident. If the scar is noticeable and less than 2 cm in length, the scar tissue can usually be totally excised and the wound simply closed primarily. If the scar is longer than 2 cm and is easily visible, the scar line should be broken up from a straight line into multiple small segments to break the scar line visually and alter the forces of tension on the wound margin to limit contracture[164] (Fig. 21-51). Broken line closure techniques include the W-plasty, zigzag-plasty, and Z-plasty.[3,21,99]

The W-plasty involves excision of the scar tissue with multiple small triangles, with as many incisions made within the favorable lines of tension as possible. The base and apex of the triangles should be perpendicular to the scar line. The incisions should also be made

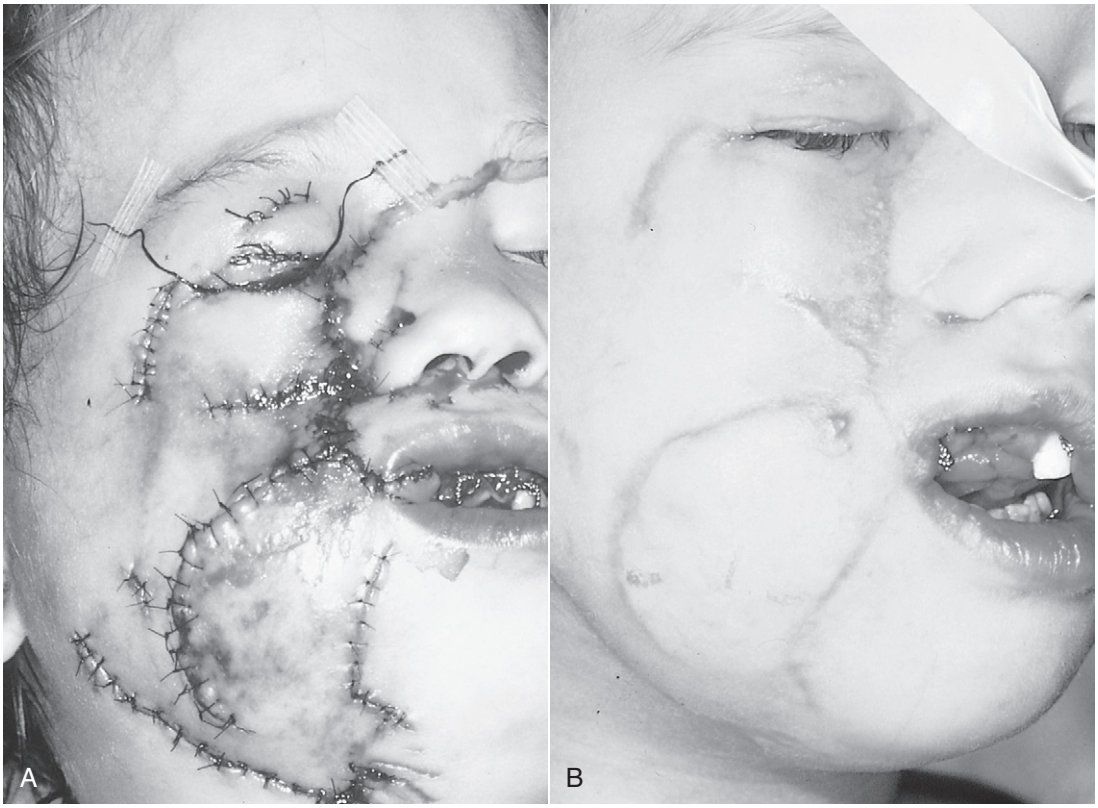


FIGURE 21-50 **A,** Dog bites to the head and neck region are common reasons for emergency room visits, and patients can have significant injury to the soft tissue and also fractures of underlying bone and dentoalveolar structures. Proper antibiotic coverage and tetanus control are important with animal bite wounds. The wounds should be thoroughly débrided and irrigated. Most laceration and tear wounds can be closed primarily, but puncture-type wounds and wounds with excessive crushing of the tissue that requires considerable débridement should be delayed for several days before definitive treatment. Wounds that have had significant crushing injury or heal by secondary intention may develop significant scarring. **B,** 3 months following injury, the immature scar tissue is irregular, raised, and purplish because of increased vascularity, collagen, and fibroblasts. Maturation of the scar may require 6 to 24 months as the scar softens, fades, and flattens. Scars should be revised secondarily only after the tissue has undergone maximal maturation for as long as 12 months.

perpendicular to the skin to allow for even closure. The wound margins should be undermined and meticulously closed in layers.[99,164]

The zigzag-plasty gives better results for breaking up linear scars because of the more random nature of the scar line (Fig. 21-52). The zigzag-plasty consists of a series of rectangles and squares between the triangles of the W-plasty. The procedure requires more operative time and technique but tends to camouflage the scar line better.[164] The wound margins should be undermined and closed meticulously in layers.

The Z-plasty was first described by Horner (1837) and further developed by Denonvilliers (1854) for correction and alteration of scars to a more favorable position.[99] It can alter the direction of scars across the lines of tension to fall more closely within the favorable lines. The Z-plasty is a local transpositional flap consisting of a central limb, with limbs extending from each end in opposite directions at an angle from 40 to 60 degrees. The change in direction of the central limb depends on the size of the angles of the Z-plasty. If both angles of the Z-plasty are 60 degrees, the central limb will rotate 90 degrees when the flaps are transposed. Smaller angles cause less transposition. The Z-plasty also increases scar length by up to 200%.[164]

The Z-plasty should be used for scars that are more than 40 degrees from the line of tension because the technique can change the axis of the scar, relieve the tension in the tissue of the area, and prevent linear scar contracture.[99] The W-plasty and zigzag-plasty procedures, along with simple excision of small wounds, are indicated for scar revision of most facial wounds.[164]

Combination therapy of surgical excision of the scar tissue with corticosteroid injections and pressure dressings is the mainstay of successful treatment. Surgical excision with primary skin closure and no additional therapy results in recurrence in more than 50% of patients. With the development of a hypertrophic or keloid scar, the patient should be instructed to massage the scar at least three to four times daily with a corticosteroid cream. If the scar develops into a keloid, steroid injections should be initiated (Table 21-8). Farrior[3] has recommended a preoperative regimen of triamcinolone acetonide (Kenalog-40), 40 mg/mL, and lidocaine 2%, 1:100,000 epinephrine times, one dose, followed by interlesional injections of triamcinolone mixed with lidocaine 1%, 1:100,000 epinephrine at a 1:4 dilution at a 3-week interval for three injections. The injections should be made evenly throughout the lesion; improvement may be seen during this period with steroid therapy alone.



FIGURE 21-51 **A-C,** A large scalp laceration associated with a motor vehicle accident in which the patient suffered significant intracranial and neurologic injuries. The scalp wound was débrided and closed primarily with an understanding that the wounds would have to be revised once the patient had recovered from her neurologic injuries. **D,** Several months following the initial accident, the patient had a large scar and hair loss. **E, F,** The scar tissue was excised with multiple small triangles, with the base and apex of the triangles perpendicular to the scar line. The incisions are made perpendicular to the skin to allow for even closure and the wound margins undermined and meticulously closed in layers. **G, H,** 3 months after repair of the scar.



FIGURE 21-51, cont'd

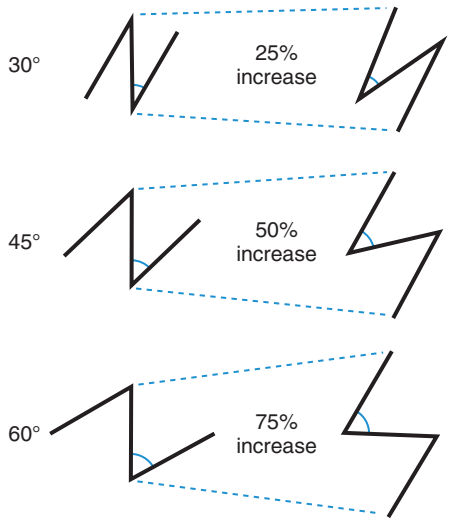


FIGURE 21-52 The classic Z-plasty has limbs with a 60-degree angle from the vertical portion. Changing this angle will alter the amount the scar length will increase.

TABLE 21-8 Steroid Injection Regimens for Treatment of Keloids

Medication	Dosage
Betamethasone acetate–sodium phosphate and 1% lidocaine mixed in equal volumes	Preoperative administration of 1-5 mL around the circumference of the keloid; no postoperative injection given
Triamcinolone acetonide (5 mg/mL) mixed with equal volume of 1% lidocaine with epinephrine (1:100,000)	Preoperative local administration of 1-5 mL, followed by 0.5-1.0 mL every 2-4 wk postoperatively
Triamcinolone acetonide (40 mg/mL), given only when signs of excessive scarring develop	0.2 mL given as an intralesional injection every 3 wk for 3 mo
Triamcinolone acetonide (10 mg/mL) mixed with equal volume of 2% lidocaine	1 mL injected every 2 wk, for a total of four injections preoperatively; repeat the same dose at surgery and every 2-3 wk, for a total of four injections postoperatively

Adapted from Zuber TJ, DeWitt DE: Earlobe keloids. *Am Fam Physician* 49:1835, 1994.

After 3 months without acceptable improvement, the lesion should be excised. The excision should be made just within the margin of the keloid to prevent creation of a new tissue reaction. An intralesional injection of the triamcinolone solution should be given before excision of the lesion to also inhibit tissue reaction. The wound margins are slightly undermined to limit wound surface tension. The wound is closed in layers, with deep synthetic absorbable sutures, subcutaneous polypropylene sutures, and multiple adhesive skin tapes to minimize wound tension. The suture and skin tapes should remain for 3 to 4 weeks; triamcinolone injections should begin 2 weeks postoperatively. If hyperplastic changes occur following removal, steroid injections should continue on a biweekly basis for 2 months. If no hyperplastic changes are noted, the steroid injections should be tapered off over 6 months.[3,164]

In patients with a tendency to form hypertrophic scars, the application of a pressure dressing and splinting may be preventive. A firm pressure dressing may disrupt the synthesis of collagen by decreasing circulation to the area. It may take months of splinting and pressure to aid in the prevention of hypertrophic scars.[166]

BURNS

Each year, in the United States, approximately 2 million individuals experience burns severe enough to require medical attention. Approximately 100,000 require hospitalization and 6,000 to 20,000 die as a result of complications of burn injury.[167] Burn injuries can be categorized into thermal (flame and scalding), chemical, radiation, and electrical types. Children younger than 5 years are particularly prone to burn injuries.[168] Electrical burns caused by high-voltage electrical current are unique because the underlying tissue destruction is frequently more extensive than the skin injury. Low-voltage electrical current results in potentially reversible physiologic changes, such as arrhythmias, without notable damage to the skin and underlying tissue. The exception to this rule is the electrical burn to the mouth, which is usually caused by a 120-V, 60-cycle alternating current.[169] Chemicals continue to produce local skin damage as long as they are in contact with the skin. Acids denature tissue by coagulation necrosis, with protein precipitation. The wounds may initially appear to be deeper than they actually are. Alkalies cause liquefaction necrosis. Generally, alkali burns are more serious than acid burns because the alkali penetrates much deeper into the tissue, producing progressive necrosis for several hours after contact. The severity of radiation burns, such as sunburn, is related to time of exposure and wavelength characteristics, which determine penetration.

The resuscitation of a burn patient should follow the same systematic evaluation as that for patients with other types of trauma, including primary and secondary surveys followed by definitive care. The most common cause of head and neck burns is flame or flash burns. It is particularly important with thermal injury of the head and neck to consider inhalational injury. Inhalational injury with resulting hypoxia is the most common cause of death during the first hour after the burn.[170] When the patient history and physical examination suggest a possible inhalational injury, fiberoptic nasopharyngoscopy is indicated to evaluate the upper airway.

The following factors and signs suggest inhalational injury[171]:

1. History of confinement at a burning building
2. History of exposure to an explosion
3. History of decreased level of consciousness
4. Carbon deposits around the mouth or oropharynx
5. Inflammatory changes of the oropharynx
6. Carbonaceous sputum
7. Singed facial hair
8. Respiratory distress

BOX 21-3 American Burn Association: Grading System for Injury Severity

MINOR BURNS

- First- and second-degree burns that cover <15% of body surface area in adults and <10% in children <6 yr
- Third-degree burns covering <2% of the body surface area in adults

MODERATE BURNS

- Second-degree burns that cover 15% -25% of body surface area in adults and more than 10%-20% in children
- Third-degree burns that cover <10% of body surface area

MAJOR BURNS

- Second-degree burns covering >20%-25% of body surface area in adults and 20% in children
- Third-degree burns that cover at least 10% of body surface area
- Burns of the hands, face, eyes, ears, feet, or perineum
- Inhalation burns
- Electrical burns
- Burns complicated by fracture or major trauma
- In general, all burns in infants and older adults
- Burns in which patient is a poor risk because of preexisting conditions, head injury, cerebrovascular accidents, psychiatric disabilities, or closed-space injuries

Adapted from American Burn Association: Appendix B to hospital resources document. Guidelines for service standards and severity classifications in the treatment of burn injury. *Bull Am Coll Surg* 69:24, 1984; and from Griglak MJ: Thermal injury. *Emerg Med Clin North Am* 10:369, 1992.

Another immediate concern is carbon monoxide poisoning caused by smoke inhalation. The most common cause of death in fires is because of the accumulation of carbon monoxide; carbon monoxide poisoning should be considered in any burn patient. Carbon monoxide has an affinity for hemoglobin 200 times that of oxygen and reversibly displaces oxygen on the oxygen- hemoglobin dissociation curve. The half-life of carboxyhemoglobin is 4 to 5 hours in room air. In an individual breathing 100% oxygen, the half-life is reduced to 45 to 60 minutes.[172] Therefore, it is prudent to administer oxygen to all patients with a history of exposure to excessive smoke.

The severity of burn injuries is related to the body surface area involved and depth of the wound. The amount of body surface area involved can be estimated by using the rule of nines. Any patient with burns covering more than 20% to 25% of their total body area requires support with IV fluids, Foley catheter to monitor urine output, and placement of a nasogastric tube because of the risk of intestinal ileus.*

After resuscitation and stabilization of the patient, the wounds are evaluated for extent and depth. Typically, burn wounds are classified as first-, second-, and third-degree burns (Box 21-3). First-degree burns (e.g., sunburn) are the most superficial and involve the outer epithelium, sparing the dermis. The skin becomes erythematous because of local vasodilation, remains dry, lacks blisters, and is painful to touch. These burns usually cause no permanent injury and resolve in 3 to 6 days.[168] Desquamation or exfoliation of the superficial epidermis is common 3 to 5 days after injury.

Second-degree or partial-thickness burns are classified as superficial or deep. Superficial partial-thickness burns are typically bright red or mottled, have a moist surface caused by oozing of serous fluid from damaged tissue, frequently form bullae, and are exquisitely painful. Pressure over the affected area causing blanching will be followed by rapid capillary refill. Because the epidermis and outer dermis are affected, epithelial elements are retained. Healing usually occurs with minimal scarring in 10 to 21 days.[175] First-degree burns, by definition, do not form bullae; however, because blisters may take 12 to 24 hours to develop, those initially appearing as first-degree burns may later be diagnosed as superficial second-degree burns.[167] Deep partial-thickness burns are caused by immersion in hot fluids or exposure to flame and are dark red or yellow-white, with a moist surface. Sensation to pressure remains over the wound but not to pinprick because of damage of the superficial cutaneous nerve endings. Only the epidermal cells lining the hair follicles and sweat glands remain and serve to reepithelialize the damaged skin surface. These burns heal in 3 to 9 weeks and are predisposed to hypertrophic scar formation. Thus, they are usually treated with excision of eschar and autologous skin grafting.

In third-degree or full-thickness burns, the epidermis and entire dermis are damaged. Electricity, concentrated chemicals, and prolonged contact with hot objects, liquid, and flame frequently cause full-thickness burns. These wounds are dry and appear pearly white or charred, or resemble parchment. Resurfacing and grafting are necessary for wound closure. The necessity for analgesia in burn patients is generally inversely proportional to the depth of the burn. Cutaneous afferent nerve endings are completely destroyed in full-thickness burns, resulting in minimal pain. However, partial-thickness burns, particularly superficial partial-thickness burns, are usually painful.

In burns of the head and neck, massive facial edema may result from even minor burns because of the general laxity of the skin in these areas. If deep full-thickness injury is present with inelastic necrotic tissue, edema formation may be directed toward internal structures. Upper airway obstruction may occur suddenly and should be anticipated.[175]

Only after full examination of the total body surface area and depth of the burn can treatment alternatives be considered. Although various areas of the country have slightly different requirements, the following conditions warrant transferring patients to a specialized burn facility[167,169,172,176]:

1. Second- and third-degree burns on more than 10% of body surface area
2. Second- and third-degree burns on the face, hands, genitalia, or perineum, or over major joints
3. Third-degree burns on more than 5% of the body surface area
4. Electrical burns
5. Inhalation injury

*References 167,168,170,171, 173-175.

6. Circumferential burns of any extremity
7. Suspected child abuse

The anatomic units in the head and neck region have different qualities that influence the outcome of a burn injury. For example, the hair follicles of the scalp and beard extend deep into the underlying dermis; therefore, epithelial repair is more likely. Nasal burns, which occur in 70% of facial burns,[169] tend to be partial-thickness burns because of the skin thickness and rich vascular supply. The thin skin of the eyelids predisposes them to full-thickness burn injury. The exposed position of the ears results in more than 90% of patients with burns of the head and neck also sustaining burns of the ear,[169] which are usually full-thickness injuries.

Successful treatment of small burns (2% to 3% of the body surface area) is often accomplished with the use of cold water soaks. These can reduce the pain of partial-thickness burns and, if instituted within 10 to 15 minutes of injury, may reduce the extent of tissue damage.[168] Direct tissue cooling is contraindicated in larger burns because hypothermia is common and may cause cardiac arrhythmias and failure of the patient to respond to resuscitation. More severe wounds are treated with topical antimicrobials and wet to dry dressings, with frequent débridement.

The nonviable necrotic tissue that forms over a burn wound is a fertile medium for bacterial growth. Therapy is directed toward prevention of infection and supportive care during wound healing. Although gram-positive bacteria, fungi, yeast, and viruses have all been associated with primary infection of burn wounds, the most serious offenders are gram-negative bacteria, with *Pseudomonas aeruginosa* being the most frequent.[172,176] Partial-thickness burns can be converted to full-thickness burns in the presence of infection.[169] Systemic antibiotics are of little value because of their inability to reach the wound effectively. Therefore, topical antimicrobials are the mainstays of treatment. Topical agents commonly used in burn injuries include 0.5% silver sulfadiazine, 0.5% silver nitrate, and mafenide acetate. The agent of choice is silver sulfadiazine. It demonstrates moderate penetration of the eschar and is painless to administer. A common side effect of silver sulfadiazine is leukopenia, which may develop 24 to 72 hours after application.[173] Mafenide acetate is the only agent that penetrates the eschar effectively; thus, it is more effective in the presence of bacterial contamination. A disadvantage of using mafenide acetate is that pain may last 20 to 40 minutes after application. Silver nitrate is painless, penetrates the eschar poorly, and is not an effective topical antimicrobial after the wound has been colonized. Dressings are usually saturated with silver nitrate and applied over the wound. Silver nitrate is less popular, although it is used in patients who cannot tolerate sulfa drugs.[168,174,175] Other topical agents used to limit colonization of moderate burns include polymyxin B, bacitracin, neomycin, povidone-iodine, and gentamicin cream. It is also important to note that tetanus prophylaxis is mandatory in all burn patients.

The different types of wound dressing used to treat burn wounds are generally open or closed. The most common method for more moderate burns is closed occlusive dressing,[176] which may be used with or without topical antibiotics. It allows for wound protection and good hygiene and allows the wearing of clothing. Early treatment of burns of the head and neck includes cleansing, débridement of disrupted blisters, and application of moist dressings to prevent large eschar formation. When necrotic debris or eschar persists despite these moist dressing changes, the use of wet to dry dressings should be initiated until healthy granulation tissue appears to serve as a graft recipient bed. Removal of the dry dressing aids in débridement of the dry coagulum and necrotic debris.[169] Wounds of the head and neck can also be treated with the open method, in which no dressing covers the wound. Topical antibiotic cream is applied in a ⅛-inch layer over the burn and washed and reapplied twice daily. For a more detailed discussion of burns, see Chapter 28.

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CHAPTER

22

Secondary Revision of Soft Tissue Injury

Michael R. Markiewicz | R. Bryan Bell

OUTLINE

Soft Tissue Wound Healing
Principles of Wound Management
Preoperative Consultation
Scar Analysis
Incision Placement
Facial Aesthetic Units
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Scar Location
Sun-Reactive Skin Type Classification
Skin and Aging
Timing of Scar Revision
Surgical Options
General Principles of Linear Scar Revision
Procedures
Complications
Hematoma
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Resurfacing Procedures
Preoperative Considerations
Patient Preparation
Specific Techniques
General Postoperative Care for All Ablative Techniques
Adjunctive Scar Revision Techniques
Tissue Expansion
Steroids
Filler Materials
Botox
Summery

Scarring is a natural progression of healing following soft tissue injury.[1-3] The primary and immediate secondary management of head and neck wounds will inevitably affect the long-term aesthetic and functional outcomes of the resulting scar. Therefore, meticulous planning is needed from the initial patient encounter to ensure successful outcomes in scar healing, maturation, and revision. This chapter will review the evaluation and management of various post-traumatic scarring, with an emphasis on commonly encountered clinical scenarios. It is meant to provide the reader with a literature-based rationale for treatment.

SOFT TISSUE WOUND HEALING

A basic understanding of wound healing is needed because of its relevance to scar formation and subsequent effects on revision.[3-5] Wound healing is classified into three main phases (Fig. 22-1). These include the immediate inflammatory phase (1 to 3 days), which is characterized by a vascular and inflammatory response including local vasoconstriction for the first 5 to 10 minutes followed by a local vasodilatory response. The inflammatory phase takes place the first few days after injury, during which wound strength relies mainly on the fibrin clot. The second phase, the proliferation phase (3 to 12 days), begins within 24 hours after injury and is characterized by the recruitment of blood vessels and fibroblasts, resulting in granulation tissue. Epithelialization may be completed in 24 to 48 hours in primarily closed wounds or be delayed for as long as 3 to 5 days in wounds healing by secondary intention The final phase of wound healing is the maturation phase, during which the scar gains strength and volume and erythema decreases. Complete scar maturation and final tensile strength generally take 12 to 18 months (Fig. 22-2).

In any wound, even one with adequate primary closure, the gap between wound edges is temporarily repaired in the form of a clot consisting of platelets engrossed in a mesh of cross-linked fibrin fibers derived from fibrinogen cleaved by thrombin. The platelet clot serves as a reservoir of cytokines and growth factors that are released as platelets degranulate. Growth factors and cytokines recruit inflammatory cells, fibroblasts, and capillary ingrowth to the wound site, which invade the clot to form granulation tissue, leading to contraction of wound margins. A connective tissue scar remains when wounds are closed by secondary intention—the larger the wound gap, the wider the connective tissue scar.

Scar consists of a poorly constructed collagen matrix in dense parallel bundles, which is contrasted to the efficiently cross-linked meshwork of collagen in the native dermis. It occurs in three phases—epithelialization migration, collagenization, and angiogenesis–granulation tissue formation. Epithelialization occurs by migration of keratinocytes over the dermis and under the clot (Fig.

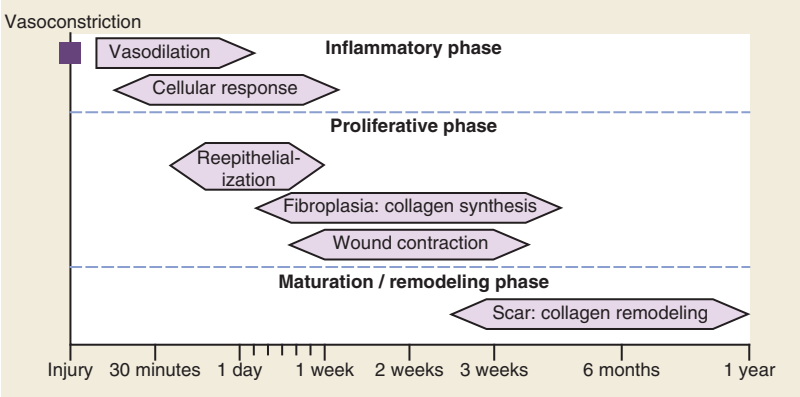


FIGURE 22-1 Phases of inflammation.

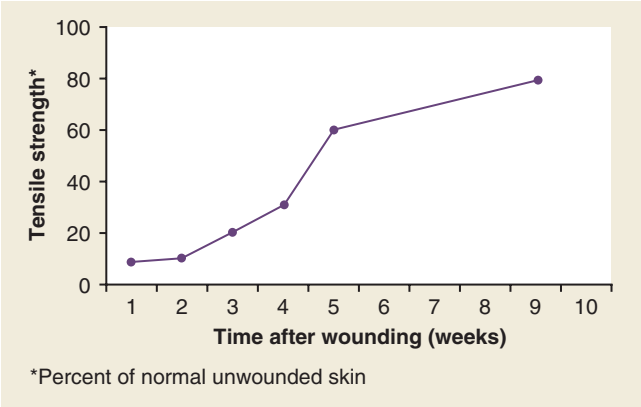


FIGURE 22-2 Maximum scar tensile strength is obtained approximately 12 to 18 months following injury. (Adapted from Goslin JB: Wound healing for the dermatologic surgeon. J Dermatol Surg Oncol 14:959–972, 1988.)

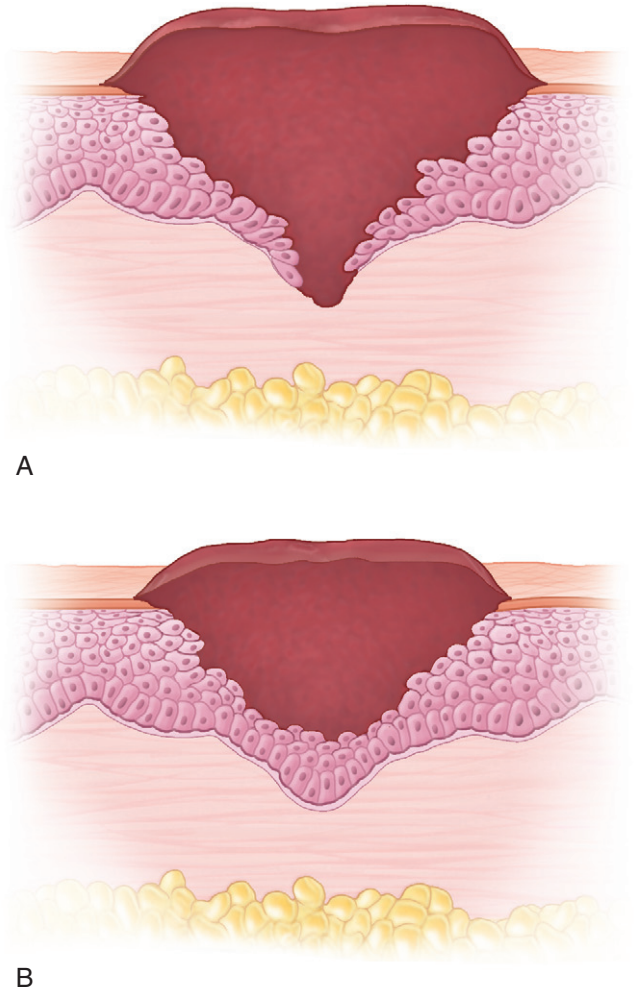


FIGURE 22-3 **A,** A leading front of basal epithelial cells divides and migrates beneath a dried clot. **B,** Contact inhibition and further differentiation and epithelial stratification.

22-3), which is facilitated by growth factors such as epidermal growth factor (EGF) and transforming growth factor α (TGF-α).[6-8] Once a monolayer of keratinocytes covers the denuded wound surface, epithelial migration ceases and a new stratified epidermis with underlying basal lamina is reestablished from the margins of the wound inward. All aspects of a skin flap rarely touch along all the length of the wound; migration is aided by contraction of the underlying connective tissue. Within 3 to 4 days of injury, dermal fibroblasts around the wound begin to proliferate and lay down a collagen-rich matrix, a process modulated by platelet-derived growth factor (PDGF) and transforming growth factor β (TGF-β).[9-12] Finally, angiogenesis of the wound and the incorporation of pink, capillary-filled granulation tissue is mediated by fibroblast growth factor 2 (FGF2) and vascular endothelial growth factor (VEGF) released at the site of the wound.[13] Connective tissue contraction ultimately closes embryonic and adult wounds. The major difference between scar-free healing and scar formation is that a scar-free wound site has significantly less inflammation present.[5]

PRINCIPLES OF WOUND MANAGEMENT

General principles of wound management should always be followed in the traumatic setting. These include an initial thorough removal of foreign bodies, débridement, and cleansing of the wound with copious pulsatile irrigation. Formal scrubbing of the wound may be undertaken if necessary but should be done sparingly to spare further damage to soft tissues. All devitalized soft tissue should be débrided and excised and sharp healthy wound margins should be obtained to facilitate closure, with minimal scarring.[14] Devitalized tissue increases the local

inflammatory response and subsequent scarring. Undiluted hydrogen peroxide use in open wounds should be avoided because it inhibits wound healing and may cause tissue necrosis. Normal saline with or without bacitracin is the preferred irrigant for soft tissue trauma. Although delayed closure may be necessary in some wounds caused by edema and/or high-velocity injuries such as gunshot wounds, in wounds with a significant amount of devitalized tissue, or because of other, more urgent medical and surgical interventions that precede the treatment of head and neck injuries, primary closure should be attempted, if feasible. In the case of avulsed tissue or the inability to reapproximate local tissue under minimal tension, local flaps, regional flaps, or free tissue transfer may be needed to restore tissues adequately to previous form and function.[15] Wounds should be closed in layers, if possible, to place minimum tension on the superficial skin closure and aid in the eversion of skin edges to minimize scar widening.[16] Tension across a wound decreases blood flow, which leads to necrosis of wound edges and increased connective tissue growth in the proliferation phase, and to elongation of scars in the remodeling phase.[17,18] Skin flaps should be appropriately undermined so that wound edges can be everted, which will minimize scar depression.[19]

Meticulous attention to detail and gentle tissue handling are important to optimize wound healing and minimize scarring.[1] A multivariate analysis has shown that characteristics associated with suboptimal cosmetic appearance following laceration and surgical incision closure are associated tissue trauma, use of electrocautery, incomplete wound edge apposition, and increased wound width (healing by secondary intention).[20] If permanent sutures are used for wound closure, they should be removed in 5 days or less to prevent track marks. Another option is to use fast, resorbing plain gut suture (5.0 to 6.0). In a study that compared 5.0 or 6.0 nylon sutures versus plain gut sutures for skin closure in a population of pediatric patients who suffered lacerations, no difference in infection or wound dehiscence was found between the two groups; however, slightly better cosmesis was found in the plain gut suture group.[21] Similar studies,[22] including a meta-analysis,[23] have confirmed these results, demonstrating no difference in long-term cosmesis, scar hypertrophy, infection rate, wound dehiscence, and wound erythema or edema between lacerations closed with nylon versus plain gut suture. For scalp incision and laceration repair, the use of staples as an alternative to suturing has been demonstrated in a randomized control trial to reduce procedure time significantly while not demonstrating any difference in cosmesis by a blinded physician at 1 week and 6 to 18 months postrepair.[24]

Adhesive bandages such as Steri-Strips (3M, Maplewood, Minn) should be used to reduce wound tension at the time of closure and to reduce the need for long-term suture placement in those wounds under increased tension. The use of 2-octyl cyanoacrylate–based fast-acting skin adhesives has been shown to be just as effective as Steri-Strips in reducing hypertrophic scar formation,[25] although other study results have demonstrated superior cosmesis in wounds dressed with Steri-Strips.[26]

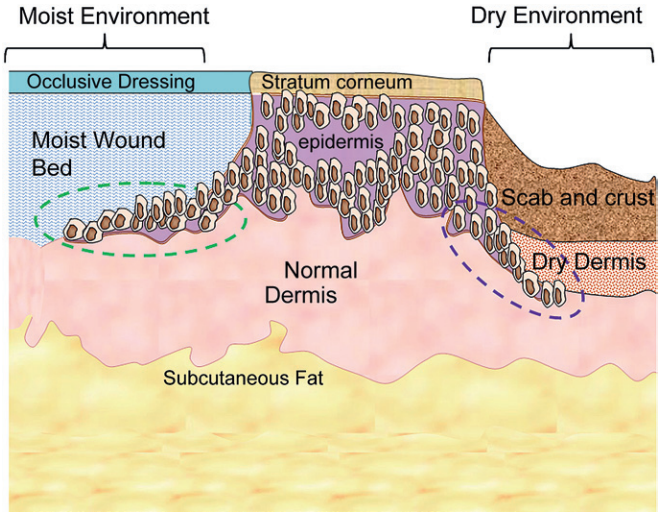


FIGURE 22-4 The effects of a moist occlusion dressing on stimulating more rapid epithelialization *(left)* than under a dried clot *(right).* (From Brenner MJ, Perro SA: Recontouring, resurfacing, and scar revision in skin cancer reconstruction. Facial Plast Surg Clin N AM 7:469–487, 2009.)

A review comparing the use of tissue adhesives versus standard wound closure in patients with traumatic lacerations found that pain and procedure time were significantly reduced in the tissue adhesive group and wound dehiscence and erythema were significantly reduced in the standard wound closure group; however, there was no difference in final cosmesis or scar appearance between the two groups.[27]

Desiccated and crusted wounds heal slower than those kept moist.[28] Epithelial migration follows a path of moisture and humidity, even if this route is longer (Fig. 22-4). This often requires more energy expenditure by the cell. In addition, an apoptosis-mediated decrease in granulation tissue has been shown to occur in vitro in flaps that are covered postoperatively.[29] Therefore, occlusive dressings and antibiotic ointment use are recommended so that epithelial migration proceeds in a direct and efficient manner. The use of occlusive or semiocclusive dressings has also been advocated as a means of promoting wound healing. Commercially available dressings, such as Tegaderm (3M), Opsite (Smith & Nephew, London), or similar polyurethane dressings minimize the amount of atmospheric oxygen that is absorbed directly through an open wound[30,31] and have been shown to decrease epithelial closure time by as much as 50%.[28,31-33] The moist environment is optimal for epithelial migration, with minimization of connective tissue formation, therefore expediting healing and reducing scar formation. These effects, however, have not been verified in a randomized control trial.[34] Antibiotic ointment should be applied to the wound until epithelialization occurs. Bacteria delay healing by direct cell damage, prolonging the inflammatory phase of wound healing by competing for oxygen; therefore, in addition to providing a moist environment, antibiotic ointment is beneficial for reducing bacterial colonization.[35]

Wounds should be cleaned daily during the immediate postoperative period using a mixture of 50%

hydrogen peroxide and 50% water. Sunblock should be applied to the area of wound closure to reduce irritation and minimize the inflammatory response. The patient should be instructed to massage the wound once epithelialization occurs using a simple base cream twice a day, 10 minutes at a time.[36,37] The effects of scar massage, however, have been questioned.[38] In addition, silicone sheets or gels may be used after epithelialization occurs and may assist in better scar maturation of hypertrophic and keloid scars.[39-44] Although a barrier, silicone sheets do allow the passage of oxygen. It has been shown in an animal model that silicone sheets cause hydration of keratinocytes within the stratum corneum of the epidermis, which has a suppressive effect on the metabolism of underlying fibroblasts and results in decreased capillary activity, hyperemia, and collagen deposition, and therefore decreases dermal thickness and subsequent scar hypertrophy.[45] Oxygen tension, pressure, or silicone leakage into dermis, however, has not been shown to be mechanisms for silicone effectiveness.[46-49] Furthermore, silicone sheets have been found to improve pigmentation, vascularity, and reduce the height of scars after 3 months of postoperative use.[50,51] Reduction of pain and pruritus has also been demonstrated to be a benefit of silicone sheets, but these benefits are not seen until after 6 months of use.[52] In a study that used the Vancouver Scar Scale to assess the effects of silicone gel, silicone gel sheets, and allantoin in improving postburn scarring less than 6 months from injury, the investigators found no significant difference in the silicone gel and gel sheet group, but there was a significant difference in improvement when comparing the allantoin groups with the silicone sheet and silicone gels group.[53] All three groups showed a significant improvement 6 months from baseline. The effects of silicone sheet use have been shown to last up to 6 months after removal; therefore, their benefits are maintained once they are discontinued[54] Onion extract, another popular topical treatment for scars, has shown only to improve scar color while not having an effect on scar height.[55]

PREOPERATIVE CONSULTATION

Detailed explanation of possible and realistic outcomes at the initial encounter will help circumvent any unreasonable expectations that the patient might have and prepare the patient for the reality that further procedures will be needed. Although the surgeon may have a good idea of which scar revision procedure the patient will need at the time of initial repair, it may not be until weeks to months later, after scar maturation occurs, that the surgeon will be able to formulate a definitive treatment plan. Depressed scars tend to contract initially and then relax as they mature, whereas hypertrophic scars and keloids will often grow as the patient ages. On follow-up examination, the surgeon should note scar tension and adjacent tissue laxity of the scar while attempting to predict the outcome of tissue rearrangement. In addition, the patient's medical history should be revisited because much may be left out in the emergent setting. Hereditary, vascular, metabolic, and immune deficiencies may be detrimental to wound healing. Finally, the patient must be compliant with postoperative care. This not only includes local care to the wound, but maintaining proper nutrition, because vitamin and trace mineral deficiencies such as deficiencies of vitamin A, C, E, zinc, and iron can be detrimental to wound healing.[31,56]

SCAR ANALYSIS

The purpose of scar analysis is to use a reliable and valid measure for quick and efficient scar assessment, which requires that the surgeon have a firm understanding of scar terminology (Table 22-1).[57] Documentation of a standardized assessment is necessary for developing a treatment plan and for serial evaluations. Photographic

TABLE 22-1 Scar Terminology

Scar Classification	Features
Mature scar	A light-colored, flat scar.
Immature scar	A red, sometimes itchy or painful, and slightly elevated scar in the process of remodeling. Many of these will mature normally over time, become flat, and assume a pigmentation that is similar to the surrounding skin, although they can be paler or slightly darker.
Linear hypertrophic (e.g., surgical or traumatic) scar	A red, raised, sometimes itchy scar confined to the border of the original surgical incision. This usually occurs within weeks after surgery. These scars may increase in size rapidly for 3-6 mo and then, after a static phase, begin to regress. They generally mature to have an elevated, slightly ropelike appearance with increased width, which is variable. The full maturation process may take up to 2 yr.
Widespread hypertrophic (e.g., burn) scar	A widespread, red, raised, sometimes itchy scar that remains within the borders of the burn injury.
Minor keloid	A focally raised itchy scar extending over normal tissue. This may develop up to 1 yr after injury and does not regress on its own. Simple surgical excision is often followed by recurrence. There may be a genetic abnormality involved in keloid scarring. Typical sites include earlobes.
Major keloid	A large, raised (>0.5 cm) scar, possibly painful or pruritic and extending over normal tissue. This often results from minor trauma and can continue to spread over years.

Adapted from Mustoe TA, Cooter RD, Gold MH, et al: International clinical recommendations on scar management. Plast Reconstr Surg 110:560–571 , 2002.

assessment using standardized lighting, spacing, positioning, camera settings, and background is beneficial for initially assessing and serially tracking the progress of the scar maturation.[58-65] Imaging software can be used to analyze, measure, and follow scars and can aid in surgical planning by almost performing the surgery before the actual procedure.[66] Immediate preoperative and postoperative photographs should be taken.[67] It is useful to review photographs after the patient has left. It is then that the surgeon will often notice subtle abnormalities that could affect the management of the scar. Also, photographs will document changes in following appointments often overlooked by the patient and surgeon.

Several factors must be considered when developing a treatment plan for scar revision, such as scar width, length, relationship to relaxed skin tension lines (RSTLs), neighboring anatomic landmark distortion, tissue loss, scar type, patient age and race, scar location (visible versus nonvisible with clothing), skin type (Fitzpatrick), tissue tension and laxity, and local tissue reservoirs (Box 22-1). Figure 22-5 illustrates a basic template for the surgeon to use for scar revision, but this must be tailored to the specific patient and scar.

The most widely used scarring index is the Vancouver Scar Scale, which gives the surgeon an objective measure of burn scars and assists in prognosis and management. Although some have described it as a valid research tool with good interrater reliability (Table 22-2),[68] others have not.[69] Another scale, the patient and observer scar assessment, has an objective component comparable to the Vancouver scale, but relies on patient opinion (Table 22-3).[70] A number of other subjective and objective assessment tools and devices have been reported in the literature.[71-76] Most indices include the clinical assessment of vascularization, pigmentation, thickness, surface, pliability, size, reflection, distortion, texture, functional deficits, and pain. These characteristics are often a clue to the cause of the scar. For example, postinflammatory hyperpigmentation is often seen in scars associated with acne, inherited and acquired diseases, temperature

BOX 22-1 Factors Influencing Selection of Technique in Scar Revision

Medical comorbidities
Scar width
Scar length
Relationship to RSTLs
Distortion of anatomic landmarks
Tissue or volume deficit
Type of scar
Age of patient
Race of patient
Patient expectations
Scar location
Skin type (Fitzpatrick)
Local tissue laxity
Adjacent tissue reservoir

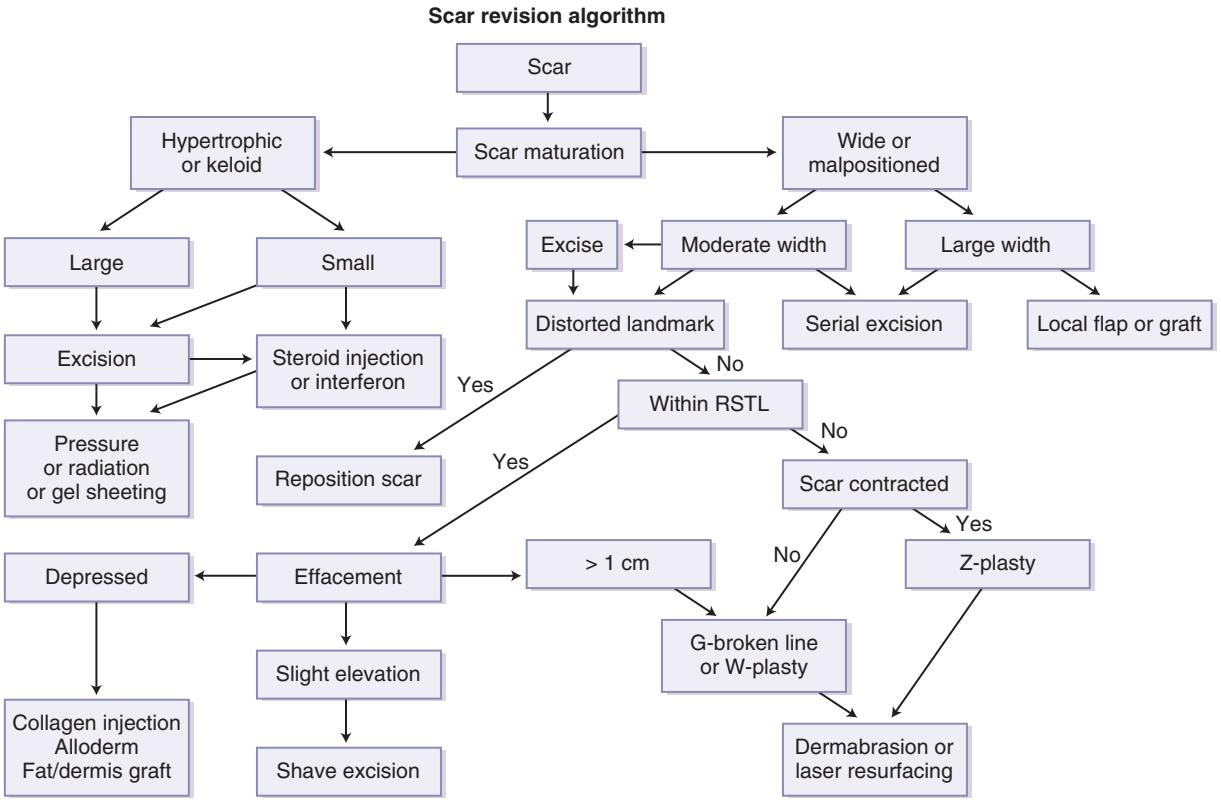


FIGURE 22-5 Flow diagram for management of optimal scar revision. (Adapted from Thomas JR. Facial scars. In Thomas JR, Holt GR, editors: Facial scars: Incision, revision, and camouflage, St. Louis, 1989, CV Mosby.)

TABLE 22-2 Vancouver Scar Scale

Scar Characteristic	Rating
Vascularity	
Normal	0
Pink	1
Red	2
Purple	3
Pigmentation	
Normal	0
Hypopigmentation	1
Mixed	2
Hyperpigmentation	3
Pliability	
Normal	0
Supple	1
Yielding	2
Firm	3
Ropes	4
Contracture	5
Height	
Flat	0
<2 mm	1
2-5 mm	2
<5 mm	3

extremes, sites of allergic reaction, radiation, and sun exposure.[77] Hypopigmentation often has iatrogenic causes, such as chemical peel, dermabrasion, laser resurfacing or, with burns, trauma and infection.[78,79]

Scar contour exists on a continuum, from the depressed scar to the hypertrophic scar and keloid. Hypertrophic scars typically remain confined to the borders of the inciting injury and usually retain their shape, whereas keloids are defined as scars that extend beyond the borders of the original wound, do not regress spontaneously, and tend to recur following excision.[80] Keloids present a special problem in treatment (see later). Scar depression often results from excessive tension across the wound. Scar texture and reflection are mainly concerns of aesthetics. Scar contracture may have functional and aesthetic ramifications in the head and neck. Pertinent to the craniomaxillofacial skeleton, scar tethering to underlying fascia, muscle, or bone may occur, resulting in aesthetic and functional deficits, mainly the failure to animate or move the face and neck.

Painful scars, especially those associated with burns, can severely debilitate the patient. The patient may present with allodynia, hyperesthesia, hypoesthesia, or dysesthesia, often caused by tissue adhesion, surround soft tissue damage, underlying skeletal damage, or neuroma or perineural scar formation. An increase in nociceptive nerve fibers following burn injury is associated with increased cutaneous innervation and increased density of certain neuropeptides, and may be responsible for chronic pain in patients with burns.[81-83] Excision of

TABLE 22-3 Patient and Observer Scar Assessment Scale

OBSERVER SCAR ASSESSMENT SCALE

Normal Skin	1	2	3	4	5	6	7	8	9	10	Worst Scar Imaginable
Vascularization	○	○	○	○	○	○	○	○	○	○	
Pigmentation	○	○	○	○	○	○	○	○	○	○	Hypo ☐ Mix ☐ Hyper ☐
Thickness	○	○	○	○	○	○	○	○	○	○	
Relief	○	○	○	○	○	○	○	○	○	○	
Pliability	○	○	○	○	○	○	○	○	○	○	
	→										
Total score, Observer Scar Scale											

PATIENT SCAR ASSESSMENT SCALE

No, no Complaints	1	2	3	4	5	6	7	8	9	10	Yes, Worst Imaginable
Is the scar painful?	○	○	○	○	○	○	○	○	○	○	
Is the scar itching?	○	○	○	○	○	○	○	○	○	○	
No, As Normal Skin	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**Yes, Very Different**
Is the color of the scar different?	○	○	○	○	○	○	○	○	○	○	
Is the scar more stiff?											
Is the thickness of the scar different?	○	○	○	○	○	○	○	○	○	○	
Is the scar irregular?	○	○	○	○	○	○	○	○	○	○	
	→										
Total score, Patient Scar Scale											

Adapted from Draaijers LJ, Tempelman FR, Botman YA, et al: The patient and observer scar assessment scale: A reliable and feasible tool for scar evaluation. *Plast Reconstr Surg* 113:1960–1965, 2004.

this tissue, in addition to reconstruction using local, regional, or distant flaps, may relieve pain in some patients. Scars in the midline are less likely to produce pain, but may cause more scarring because of increased tension.[84]

Elevated scars present in different patterns, depending on the thickness and integrity of the remaining dermal wound edges. Hypertrophic scars are dome-shaped and have a thin epidermis, whereas partially avulsive or oblique wounds result in contraction at the base of the wound, creating an elevated and inclined scar. As the scar matures, wound edges of different dermal thickness and elevation result in elevated scars with gradual step-offs; misaligned wound edges of the same dermal thickness produce elevated scars with abrupt step-offs. Depressed scars may be related to underlying tissue loss or to increased tension across the wound, resulting in atrophy of the underlying dermis, which results in a smooth and shiny scar.

INCISION PLACEMENT

Understanding of the natural lines of tension of the head and neck is imperative for proper placement of skin incisions and to optimize final scar appearance (Fig. 22-6). Dupuytren first observed that an oval wound was created when a round instrument punctured the skin.[85] Karl Langer, an anatomist, further studied this change in facial skin configuration in cadavers in rigor mortis and postulated that intrinsic skin tension occurred along predictable lines of cleavage.[86] Later called Langer's lines, these lines of tension became the basis for elective facial skin incisions for the next century. Langer's studies were, however, limited because they were performed in cadavers, showing the effects of rigor mortis. Kraissl et al preferred lines perpendicular to the action of the underlying muscle.[87-89] Borges has studied this concept in vivo and distinguished lines that follow furrows when the skin is relaxed from those that are produced by pinching the skin, and found that these lines are distinct from wrinkles, creases, and Langer's lines and that the number of lines and spacing varies among individuals.[90,91] Numerous other lines of tension have been described in the past by Cox, Webster, Stark, and Ogilvie, Kocher, and Kazanjian, among others.[87,92] These lines of tension would be called Borges' lines, or RSTLs. They follow furrows formed on relaxed skin. It has been demonstrated that placing incisions in the direction of RSTLs will produce more aesthetic outcomes with minimal scarring. RSTLs and wrinkles caused by aging may not be in the same location. Wrinkles are the change in overlying skin as a result of muscle pull over time. Conversely, RSTLs of the face often run perpendicular to the underlying muscle and, in the neck, they usually run perpendicular to areas of flexion (i.e., RSTLs run horizontally on the forehead, perpendicular to the vertical frontalis muscle). Examples of wrinkles that do not coincide with RSTLs include glabellar lines, crow's feet, and bunny lines. An exception to this rule is in the area of the circular orbicularis oculi underlying the eyelids, where RSTLs run horizontal and parallel to the muscle because of the rigid scaffold of the tarsal plate. In older patients, RSTLs often run parallel with the natural creases and wrinkles of the face. RSTLs are determined by having the patient go through a series of facial expressions (e.g., smile, frown, laugh, squint) and then pinching the skin. The line where the longest straightest line results is the RSTL.[93] RSTLS should be determined prior to administration of local anesthesia.

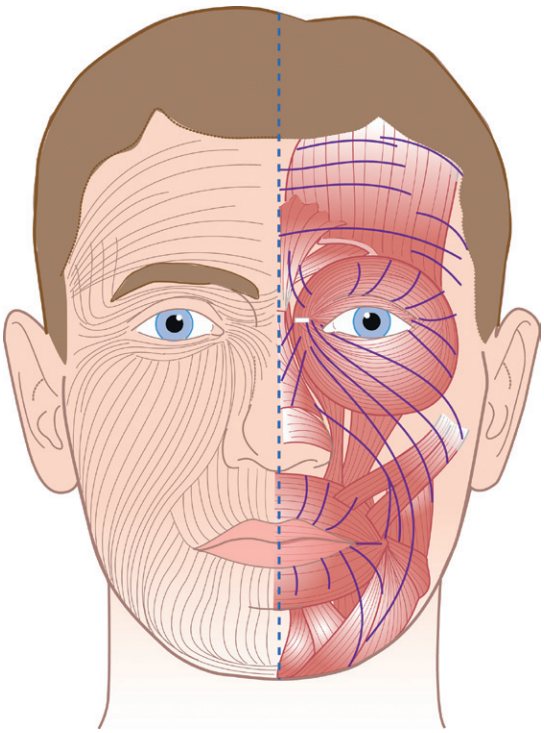


FIGURE 22-6 The relaxed skin tension lines of the face. (From Kaminer M, Arndt K, Dover J, et al: Atlas of cosmetic surgery, ed 2, St. Louis, 2009 Saunders.)

The lines of maximum extensibility (LMEs), also known as antitension lines (ATLs), run perpendicular to RSTLs. When incisions are placed perpendicular to RSTLs, an S-shaped scar will develop. Pinching the skin perpendicular to RSTLs produces humps; incisions parallel to this plane should be avoided. When the areas is too distorted for assessment, the contralateral side may be used.[94] Incisions made parallel to the RSTL will produce a narrow scar under minimal tension, as opposed to those placed parallel to the LME or ATL, which will be under maximum tension and will have a significant propensity to widen and produce broad unsightly scars. A major factor in deciding to undertake scar revision is whether scars are located perpendicular to RSTLs or in ATLs. Note that the ultimate goal of scar revision is to orient the scar parallel to RSTLs.

FACIAL AESTHETIC UNITS

In trauma and reconstructive facial surgery, the face can be divided into facial aesthetic units.[95] Gonzales-Ulloa initially described 14 aesthetic units.[96-99] These include the forehead, right and left cheeks, nose, right and left upper lids, right and left lower lids, right and left ears, upper lip, lower lip, mental region, and the neck (Fig. 22-7). These aesthetic units can be further classified into subunits based on skin thickness, color, elasticity, and

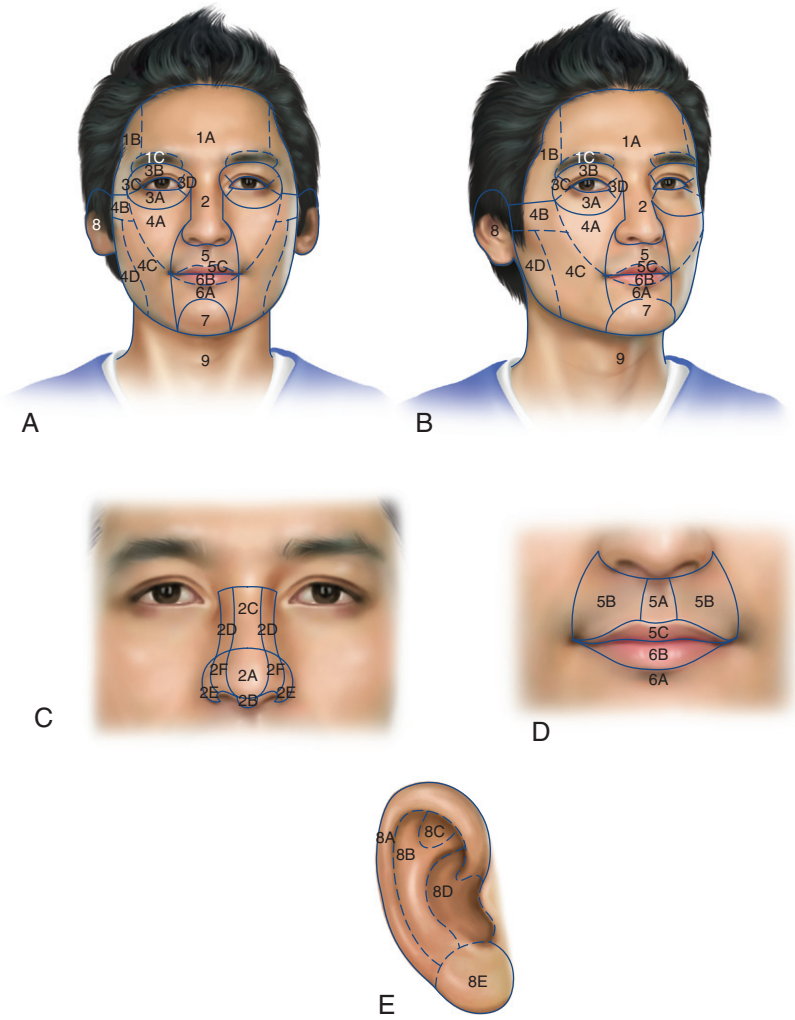


FIGURE 22-7 **A,** Frontal and profile **(B)** views of the face. **C-E**, Nasal, lip, and ear units of the face, respectively. *1,* Forehead. Subunits—A, central; B, lateral; C, eyebrow. *2,* Nasal. Subunits—A, tip; B, columellar; C, dorsal; D, right and left dorsal side wall; E, right and left lateral alar base; F, right and left alar side wall. *3,* Periorbital. Subunits—A, lower eyelid; B, upper eyelid; C, lateral canthal; D, medial canthal. *4,* Cheek units. Subunits—A, medial; B, zygomatic; C, lateral; D, buccal. *5,* Upper lip unit. Subunits—A, philtrum; B, lateral; C, mucosal. *6,* Lower lip. Subunits—A, central; B, mucosal. *7,* Mental. *8,* Auricular. Subunits—A, helical; B, antihelical; C, triangular fossa; D, conchal; E, lobe. *9,* Neck.

underlying structural support. Additional subunits within each facial unit have been described that delineate natural break lines that allow the surgeon to conceal a scar or skin graft placement[100] (Fig. 22-7). For example, the nose can be broken down into the nasal bridge, sidewall, tip, ala, soft triangle, and columella.[101] The upper lip is divided into the philtrum column, nostril sill, alar base, and nasolabial crease[102] Placement of incisions at the seams of these units or subunits, or at facial midline will produce the least perceivable scars.

The division of facial aesthetic units is based on natural breaks formed by shadows, folds, hair, and on skin thickness, color, and texture. Facial restoration should respect the boundaries of facial units and subunits. Any restoration that includes two or more units may result in aesthetic and functional deficits; superior cosmetic results are often obtained when treatment is rendered to an entire aesthetic unit rather than to an isolated scar within a unit. An example would be when planning for dermabrasion as revision treatment to a scar of the nose. In this case, the entire nasal unit should be treated to minimize the perception of residual scarring and the final scar will blend better with the adjacent units and not appear as a stand-alone patch.

When planning excisional scar revision, care should be taken to limit the revision to within one facial unit. For example, when revising a scar of the cheek, the revised procedure should not encroach on the upper lip or lower eyelid unit. Facial units are important in oncologic skin resection and in regard to tissue avulsion in trauma, when it is important to remove the entire unit or subunit, even if extension of the incision is necessary. The subsequent reconstruction, whether it be local or regional flaps or free tissue transfer, should span the entire facial unit or subunit. When the avulsed or excised area of tissue encompasses an entire unit and subunit, the final outcome blends with surrounding tissues and is less conspicuous.[1,102]

SCAR TYPE, PATTERN, AND COLOR

It is important for the surgeon to consider the type and pattern of scar when considering surgical revision, which also may give an indication about the type and extent of injury. For example, flat, linear, or curved scars often result from low-energy injuries and are more easily treated than depressed scars and scars resulting from high-energy avulsive tissue loss. Wide scars caused by increased wound tension or tissue loss are often flat and

faded and are difficult to treat because of a texture mismatch with surrounding tissues. These scars often need reexcision and closure. Some hypertrophic scars or keloids may benefit from excision or other nonsurgical treatment such as intralesional steroid injection, chemotherapy, cryotherapy, radiation, laser, or silicone sheet treatment.[80] Atrophic or volume-depleted scars often result from wounds not closed primarily, in stellate scars, with inappropriate approximation of wound edges in primary closure, or in deep abrasions. These scars often require dermal fillers, including autogenous materials such as fat transfer[103,104] and synthetic fillers.[1]

Scar camouflage is difficult in widened scars because of their smooth shiny surface, which reflects light differently than adjacent tissues. Similarly, scars that cross multiple facial units and subunits are challenging to treat because they are usually have varying skin texture, thickness, and histologic makeup. For example, nasal skin is different than skin of surrounding facial subunits containing a thicker dermis and higher density of sebaceous glands; therefore, the nearest skin is the best donor for reconstruction.[101]

Red scars are often immature and turn whiter than surrounding tissues once fully matured. Erythematous scars seen in the early stages of scar maturation are not good candidates for revision unless the redness is confined to the scar itself. The color of white scars may be the only concern of the patient; these are often treated successfully with tattooing, making them appear similar to surrounding skin.

SCAR LOCATION

The location of the scar should be considered before developing a treatment plan for scar revision. Facial units such as the forehead and nose are well supported by the underlying skeleton and tend to result in better outcomes than other facial units. Areas with multiple vectors of muscle pull, such as the medial canthus, lateral cheek, and submandibular area, often result in suboptimal outcomes. Scars with similar adjacent local skin color, thickness, and texture that are easy to mobilize and transfer without distorting the donor site area are likely to have better outcomes. A discussion of specific local and regional flaps is beyond the discussion of this chapter.

SUN-REACTIVE SKIN TYPE CLASSIFICATION

Fitzpatrick developed the sun-reactive skin type classification to describe the degree of skin reaction after sun exposure[105-107] (Table 22-4). Although developed as a guide for the treatment of skin rejuvenation procedures, this classification scheme is useful when choosing the correct management of the traumatic scar. Patients with a higher classification will often experience inflammatory changes such as dyspigmentation after soft tissue injury. Although hyperpigmentation is usually temporary, hypopigmentation is often the permanent result of a mature scar. Furthermore, patients with a very fair skin (Fitzpatrick classification type I or II) will often respond better to laser resurfacing, whereas patients with very dark skin (Fitzpatrick type IV or V) may benefit from more conservative treatment, such as superficial chemical peels and scar excision.[108,109]

TABLE 22-4 Fitzpatrick Classification of Sun-Reactive Skin Type

Skin Type	Skin Color	Sunburn	Tan
I	White	Always	Never
II	White	Usually	With difficulty
III	White	Mild	Average
IV	Moderate brown	Rarely	Easy
V	Dark brown	Very rarely	Very easily
VI	Black	Never	Always

SKIN AND AGING

A patient's age will often guide the surgeon to the timing and type of treatment chosen for scar revision. Older patients should be evaluated for the risks versus benefit in any surgical procedure in the context of complex medical comorbidities. Glogau has developed a classification for skin wrinkling related to aging as a guide to treatment for each skin type (Table 22-5). This should be taken into consideration before undertaking scar revision procedures, because skin resurfacing techniques can be chosen to match the depth of wrinkles and degree of wrinkling.[110]

It has been recommended that children have a higher tendency for scar widening than adults and should undergo scar revision after puberty because of a prolonged and exuberant inflammatory response, increased skin tension, diminished skin and dermal thickness, and higher concentration of collagen and elastin,.[111] Children past the age of puberty are more likely to cooperate with treatment and postoperative care than their younger counterparts. Severe psychological or functional impairment secondary to scar formation, however, may warrant earlier treatment.

TIMING OF SCAR REVISION

Experience has shown that in general, the appearance of most scars tends to improve spontaneously after a period of maturation during the first year after injury, after which time the appearance of the scar stabilizes and surgical revision may be considered.[112,113] However, scars oriented perpendicular to RSTLs or those associated with functional impairment may be revised earlier than 1 year on an individualized basis.[114,115]

Early scar revision may be undertaken as early as 4 weeks when there is severe functional impairment, such as salivary and lacrimal gland obstruction, lip incompetence, or lagophthalmos with corneal exposure. Early scar revision may also be advisable in well-localized scars sustained from low-energy injuries in which adjacent skin damage is minimal. Although a delay in scar revision of at least 6 months is generally recommended for most adult scars, high-energy injuries may continue to remodel and mature over a longer period of time, and delay for 12 months may be advisable.[116]

Interventions such as dermabrasion and shave excision may be performed as soon as 4 weeks from the initial

TABLE 22-5 Glogau Classification of Photoaging

Group	Classification	Typical Age (yr)	Description	Skin Characteristics
I	Mild	28-35	No wrinkles	Early photoaging, mild pigmentary changes no keratosis, minimal wrinkles, minimal or no makeup
II	Moderate	35-50	Wrinkles in motion	Early to moderate photoaging, early senile lentigines visible, keratoses palpable but not visible, parallel smile lines beginning to appear, usually wears some foundation
III	Advanced	50-60	Wrinkles at rest	Advanced photoaging, obvious dyschromia, telangiectasia visible keratosis, wrinkles even when not moving, always wears heavy foundation
IV	Severe	≥60	Only wrinkles	Severe photoaging, yellow-gray color of skin, prior skin malignancies, wrinkled throughout, no normal skin patient age, cannot wear makeup—"cakes and cracks"

BOX 22-2 Steps in Scar Revision

1. Shave excision (for elevated scars)
   - Serial scar excision
2. Tissue rearrangement
   - W-plasty
   - Z-plasty
   - Geometric broken line closure
3. Surface retreatment
   - Dermabrasion/microdermabrasion
   - Chemical peels
   - Laser ($CO_{2,2}$, erbium:YAG, 585-nm flashlamp-pumped PDL)

wound repair, based on the high level of fibroblastic activity during this period. Because dermabrasion itself causes erythema, early intervention may overlap with erythema from initial repair, leading to less total time of erythema for the patient. For hypertrophic scars and keloids, steroid injections may be considered as early as 3 weeks and may be given at the time of scar revision. Cigarette smoking, isotretinoin, and vitamin E use are injurious to wounds and scar revision should be delayed until they have been discontinued.[117]

SURGICAL OPTIONS

Scar revision can be broken down into three different treatment modalities—scar excision, tissue rearrangement, and surface treatment (Box 22-2). The selection of treatment should be based on the individual scar and on the needs and expectations of the patient. For example, conservative treatment may be warranted for a demanding patient with unrealistic expectations. Narrow scars parallel to RSTLs may have distracting features such as surface discoloration and texture irregularities. These scars will often benefit from surface treatments. Longer scars, which vary more than 35 degree from neighboring RSTLs and have a local tissue reservoir, may be improved with tissue rearrangement such as W-plasty or geometric broken line excision and closure (GBLC). Z-plasty, local or regional flaps, or free tissue transfer may be indicated if neighboring tissue reservoirs are deficient or if anatomic structures and landmarks would be distorted by closure.

GENERAL PRINCIPLES OF LINEAR SCAR REVISION

The first step in any scar revision procedure is to examine skin thickness, elasticity, texture, hair growth, and RSTLs. A fine-tipped marking pen should be used to plan the incision, which should be measured and remeasured as many times as necessary before the skin is incised. Care should be taken to avoid excess tension or forceps compression at wound edges because this might cause tissue slough because of compromised blood flow to flap margins and distort adjacent landmarks, ultimately causing hypertrophic or widened scars.

Most facial incisions may be made with a no. 15 or no. 11 blade on a no. 9 knife handle. The no. 11 blade is the most useful for straight cuts; these should be made perpendicular to the skin in a sawing-type motion, with the tip of the blade used to make initial incision. The handle of the blade is then brought down toward the skin so that the belly of the blade cuts the middle part of the incision. Finally, the blade handle is brought up again so that the tip finishes the incision. This technique ensures that skin and subcutaneous layers are incised evenly throughout the entire incision. If incising a scar, the blade should be beveled outward (Fig. 22-8*A*). This aids in creating a plane for undermining skin, allowing eversion of the wound edges and minimizing scar depression. The skin at the ends and sides of the incision should then be undermined with a no. 15 blade or curved scissors at least 2 cm in the subdermal plane to avoid motor nerves of the face (see Fig. 22-8*B*). Undermining the scalp will not aid in closure because of the tensile strength of the galea aponeurosis. Galeotomies, however, may assist with mobilization and eversion of scalp tissue.

Primary closure should be achieved, whenever possible. Healing by secondary intention may be advisable in some cases, however, such as the lateral canthi of the eyes, where skin is thin and well-contoured and is subject to constant motion because of lid closure. To avoid bunching (dog ear) at wound edges, the principle of halving should be used, whereby the first suture is placed

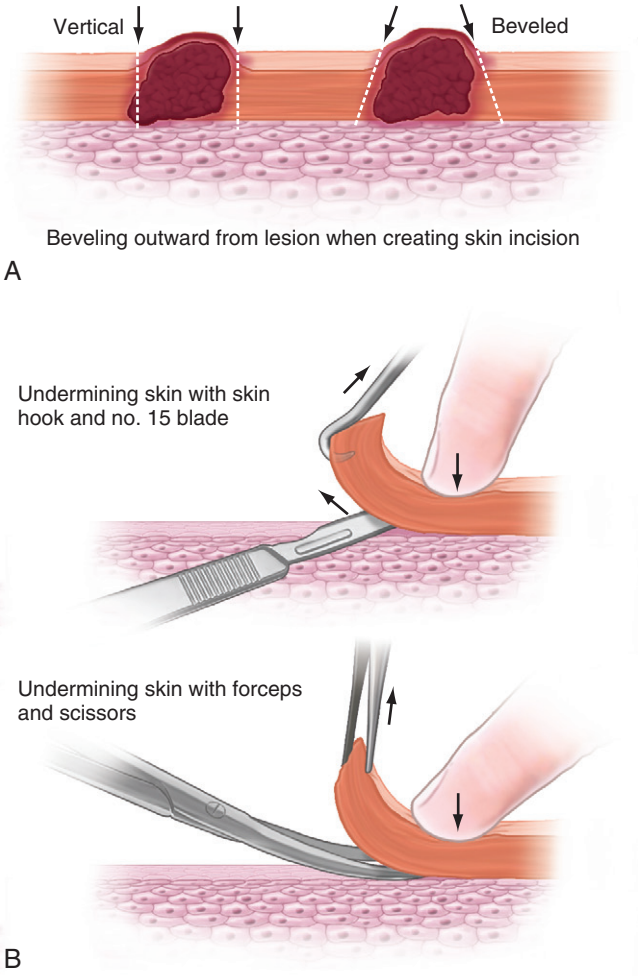


FIGURE 22-8 **A,** Beveling the blade outward when excising a scar. **B,** Undermining skin should be done with a skin hook and no. 15 blade or forceps and scissors.

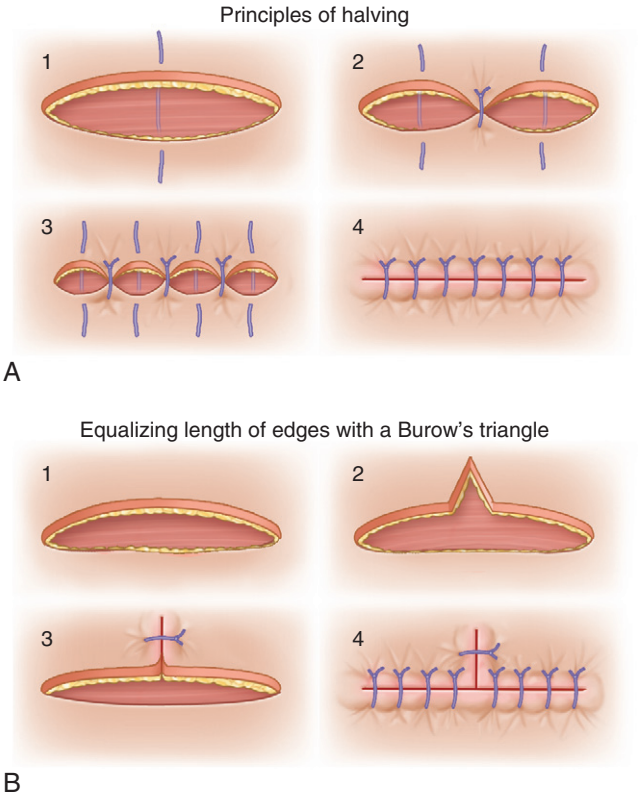


FIGURE 22-9 **A,** The principle of halving is used to avoid a standing cone deformity at the end of the incision. **B,** When an incision is not of equal skin flap proportion at both ends, a triangle is removed from the longer skin flap and closed primarily, making skin flaps of equal size.

at the center of the incision and the next two are placed at the center of the remaining halves (Fig. 22-9*A*). Continuing this down the length of the skin margins will close the wound with equal tension and eversion throughout. When incisions are not of equal length and shape, the principle of equalizing edges should be used (see Fig. 22-9*B*). In this technique, a Burow's triangle is removed from the longer skin flap and closed primarily to make the two primary skin flaps equal in length.

Meticulous attention to proper suturing technique is important for successful outcomes. A simple stitch provides minimal wound eversion and scant relief of wound tension, and should be avoided in the face. Alternatively, a mattress suturing technique is best for ensuring a tension-free wound closure, with eversion of the skin edges. The suture needle should be angled outward from the skin edge and the knot tied as far as possible from the wound (Fig. 22-10*A*). The vertical mattress suture is performed by placing the innermost suture first while holding up the wound edge, causing eversion of the skin flap. The wide suture should be thrown next. There are two types of vertical mattress sutures, the Donati and Allgower types (see Fig. 22-10*B*). Bolsters of silicone tubing or swabs may be placed under the knots of the vertical mattress to minimize tension (see Fig. 22-10*C*). A simple interrupted (see Fig. 22-10*D*) or, preferably, a Gilles half- buried corner stitch should be used (see Fig. 22-10*E*) for tacking wound corners, The Gilles half-buried stich is performed by placing a subcutaneous suture in the corner flap, finishing transcutaneously in the opposing skin edge. The most useful stitch for relieving skin tension and everting wound edges, however, is the running buried subcuticular stich. However, care must be taken to avoid tension and skin edge vascular compromise (see Fig. 22-10*F*). It should be noted that a running subcuticular suture does not relieve tension from wound edges like a simple subcuticular stich. Preferably, a monofilament absorbable suture on a small half-curved reverse cutting needle should be used. The stich should be placed while everting the skin edge with a skin hook at the middle finger of the surgeon and reaching the suture back under the dermis 2 to 3 mm from the wound edge. The knot should be buried. The skin should then be closed with fast-absorbing gut or monofilament to align the depth of the two skin flaps. Skin staples provide wound eversion, are positioned above the level of the skin when placed, and are useful for scalp and neck defect closure (see Fig. 22-10*G*).

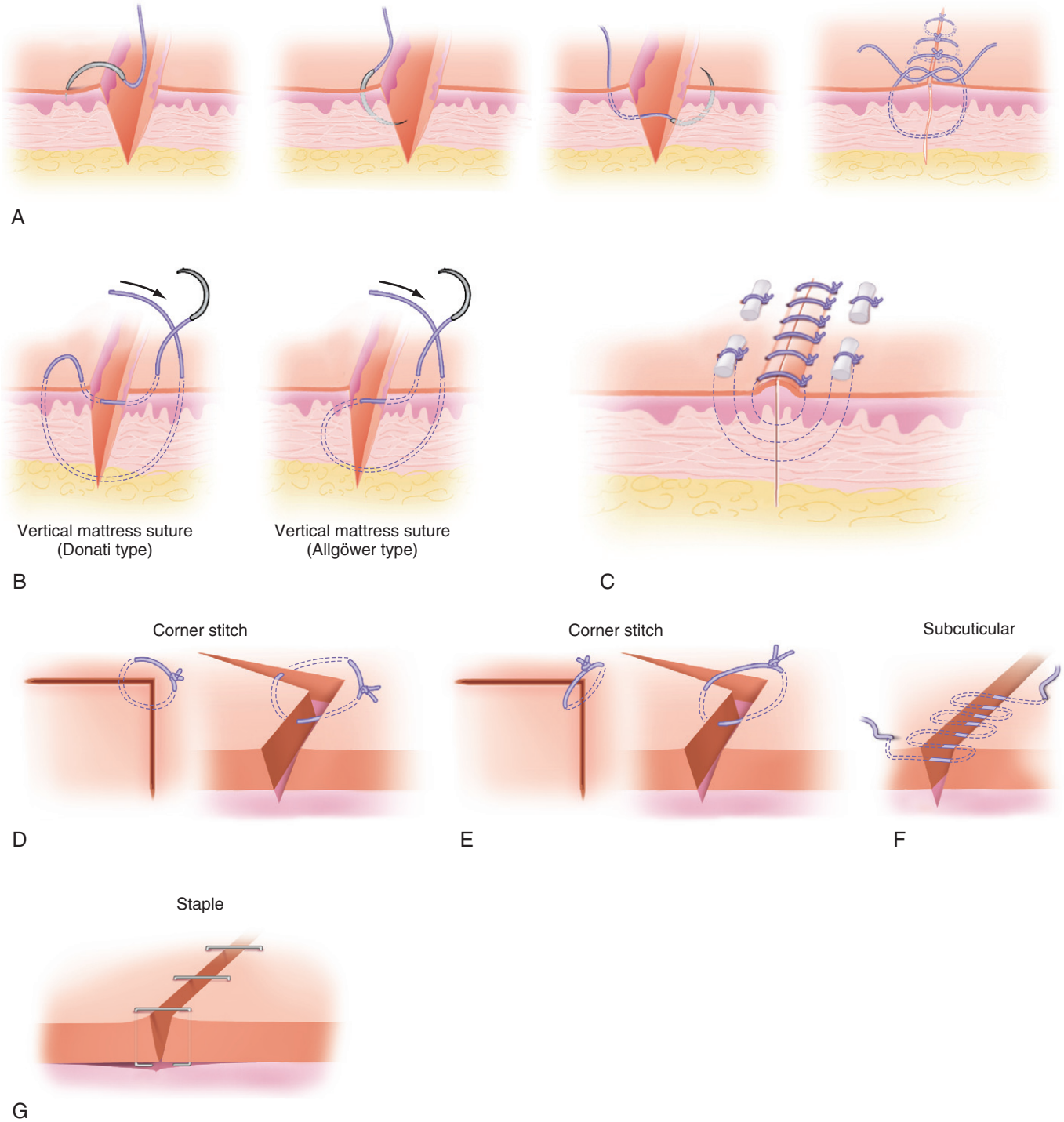


FIGURE 22-10 **A,** For simple sutures, the needle should be inserted at an outward angle from the incision, with the knot tied as far away from the incision as possible. **B,** To create more eversion of skin edges in a vertical mattress suture, a bolster may be placed between the knot and the skin **(C)**. **D,** To create maximum skin eversion, the Gilles half-buried stich is recommended. **E,** However, a simple interrupted corner stich may be used for a triangular skin flap. **F,** A subcuticular stich is useful for creating skin eversion. **G,** Staples provide good wound eversion for scalp and neck closure.

PROCEDURES

Scar Excision

The fusiform, or elliptical, incision is the most common incision used in the head and neck. It should be placed within the RSTL, with a length-to-width ratio of 2.5:1 to 3:1 to limit dog ears (Fig. 22-11). It may be straight or crescent-shaped, but should always follow a long axis parallel to RSTLs and be limited to scars shorter than 2 cm.[115,118,119] The only exception to this is the lower eyelid, where a crescent-shaped incision may leave an unsightly scar. It should be used for short anti-RSTL



FIGURE 22-11 Fusiform, or elliptical, excisions are useful for small scars that lie parallel to RSTLs.

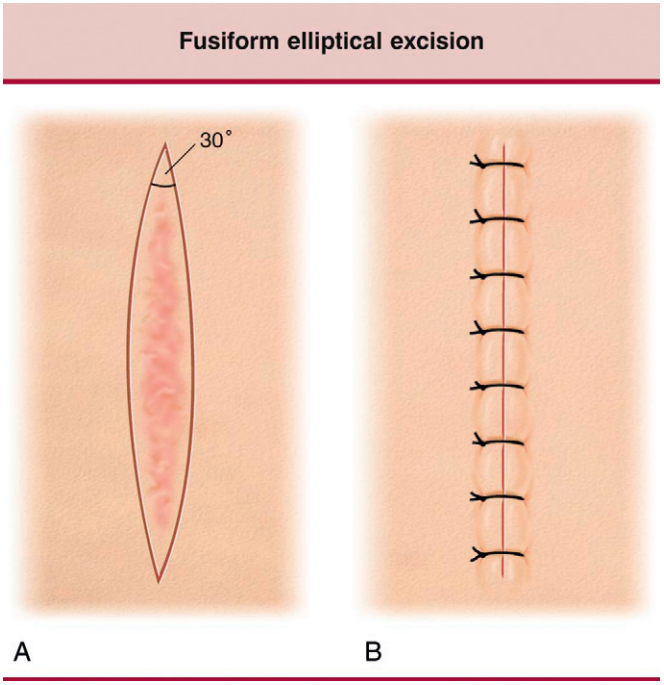


FIGURE 22-12 **A,** The skin edges at the end of the fusiform incision should be no more than 30 degrees apart. **B,** If a larger angle is used, the risk of a standing cone deformity is more likely. (From Robinson J, Hanke CW, Siegel D, et al: Surgery of the skin, ed 2, Philadelphia, 2010, Mosby.)

scars, achieving a more favorable reorientation of the lesion, or for short but widened or depressed scars. Internal angles of the planned excision may not exceed 30 degrees (Fig. 22-12).

An abundance of subcutaneous tissue in present in areas such as the ear and nose because of loose cartilage, which leads to loss of surface definition and results in unpredictable wound healing. In these areas, the surgeon may choose to debulk, inject steroids, or use local skin flaps to obtain an optimal result as an alternative to standard incisions. Furthermore, the standard elliptical excision may lead to bunching or a standing cone deformity at the end of a wound, which results from rotation of skin around a pivot point. These can be predicted and prevented by the excision of excess tissue parallel to RSTLs (Burow's triangle) that allows the cone to lie flat (see Fig. 22-9*B*). If a shorter incision is needed, an M-plasty can be created at the end of the incision to decrease the volume of healthy tissue removal, with the additional incisions functioning as Burow's triangles (Fig. 22-13). The limbs of an M-plasty should be less than 30 degrees to avoid cone deformities; closure is performed using a V-Y maneuver. This shortens the scar length along its central axis and should be used when a longer fusiform excision would encroach along a landmark, such as the lateral cantus or the vermillion. The compromise is that an M-plasty requires additional incisions.

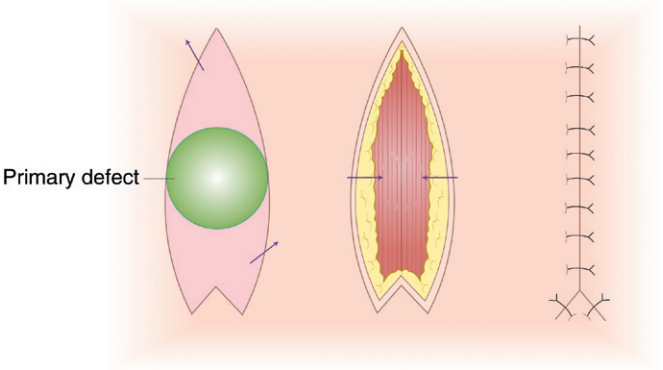


FIGURE 22-13 An M-plasty may be used to decrease the length of the incision needed, but requires additional incisions. (From Kaminer M, Arndt K, Dover J, et al: Atlas of cosmetic surgery, ed 2, St. Louis, 2009 Saunders.)

Serial Excision

Serial excision is a technique that can be used when a scar is too large to allow closure of the surrounding skin in one stage. Serial excision allows for the recruitment of adjacent skin through a process called mechanical creep, which involves elongation of the skin from constant tension over time.[120] This is distinguished from biologic creep, which involves the generation of skin from constant tension over time and only occurs with tissue expansion techniques.[121]

Initially described by Morestin,[122] the technique of serial excision is especially useful for larger scars closed primarily with a skin graft, as well as burn injuries, and uses the skin's ability to stretch and accommodate. Serial excisions can be used to move scars to or away from a more or less conspicuous facial unit or area, such as junctions of facial units, hair lines, or RSTLs. Its goal is to recruit adjacent normal tissue to cover a defect in stages.

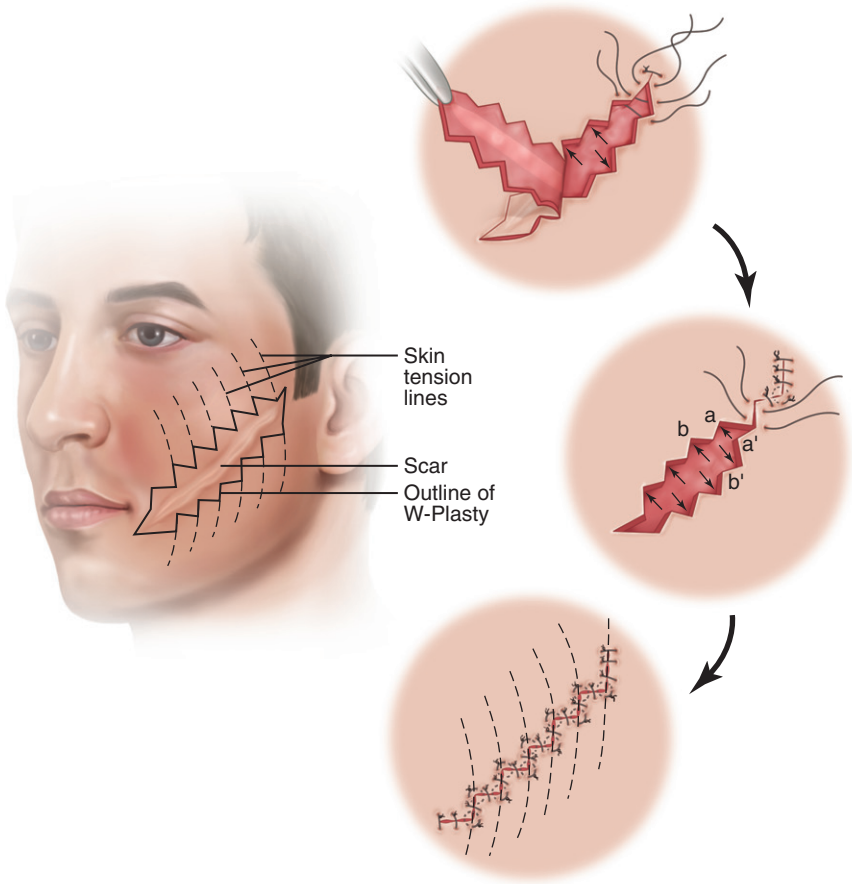


FIGURE 22-14 A W-plasty may be used for scars with a long axis more than 35 degrees from the RSTLs.

Serial excision should only be used on scars without any suspicion of malignancy and should be performed as early as possible, because scar elasticity decreases with time. All incisions should be kept within the previous scar and wounds should be undermined and closed under minimal tension.

The initial excision removes an ellipse from within the body of the scar, or from along one of its edges. Wound margins are undermined and a portion of the scar is excised after advancement and the wound is closed.[123] The skin of the wound is allowed to stretch for 8 to 12 weeks and then the procedure is repeated. For the last excision, the remaining scar is excised and native skin margins are approximated in a straight line, W-plasty, or broken line closure. One drawback of this technique is that it may lead to wide and atrophied scars caused by the tension across the wound.[124] A variety of other serial excision techniques have been described in the literature.[120,125-127] One technique divides the initial scar into four segments along RSTLs; two of the opposing segments are excised. The remaining flaps are advanced in a sigmoid-shaped ellipse and the remaining incision is closed in a Z pattern. This is repeated as needed and a fusiform excision is used to for final closure.

W-Plasty

Borges initially proposed the running W-plasty, so-named for it resemblance to the letter W[128,129] (Figs. 22-14 and 22-15). The benefit of this technique is that it will break up a long scar into smaller ones that are more aligned parallel to RSTLs, thus improving its overall appearance. This technique is indicated for long scars with a long axis more than 35 degrees from the RSTL; it is particularly useful to camouflage vertical linear scars of the forehead and temples, linear horizontal scars of the chin and cheeks, and scars located along a wrinkle. W-plasty is optimal around nonanimated facial units such as the forehead (Fig. 22-16), but is contraindicated for scars with a long axis less than 30 degrees from the RSTL, and should be avoided around the eyelids, nose, vermillion, and neck (Fig. 22-17).[130] Furthermore, the use of W-plasty is not recommended in the presence of excess tissue tension or distorted landmarks, and in scars involving two different skin types, such the cheek and lower eyelid.

Advantages of the W-plasty technique include reduced tension and simple design and execution, and it can be used for nonlinear scars. Unlike a Z-plasty, it does not lengthen a scar. For long linear scars, a GBLC is preferable to the W-plasty, because the repeating W pattern is readily noticeable by the eye.

Technique. Standard marking for a W-plasty is shown in Figure 22-18. The patient's skin should be thoroughly cleaned and degreased with alcohol or acetone to remove surface oils and should be allowed to dry completely before marking the skin to prevent smearing of the ink. Then, 1-cm segments are marked perpendicular to the



FIGURE 22-15 Reconstruction of post-traumatic scalp avulsion with secondary cicatricial alopecia, brow asymmetry, and hairline deformity. Patient was managed with tissue expansion, W-plasty excision, local rotation-advancement flaps, and brow lift. **A,** Preoperative appearance. **B,** Tissue expander in place. **C,** Outline of W-plasty. **D,** Postoperative appearance 1 year following surgery.



FIGURE 22-16 Depressed post-traumatic forehead scar in 27-year-old woman treated with W-plasty excision. **A,** Preoperative appearance. **B,** 1 year postoperatively.



FIGURE 22-17 W-plasty (W) should be used around nonanimated facial areas, whereas Z-plasty (Z) is more appropriate around mobile facial landmarks.

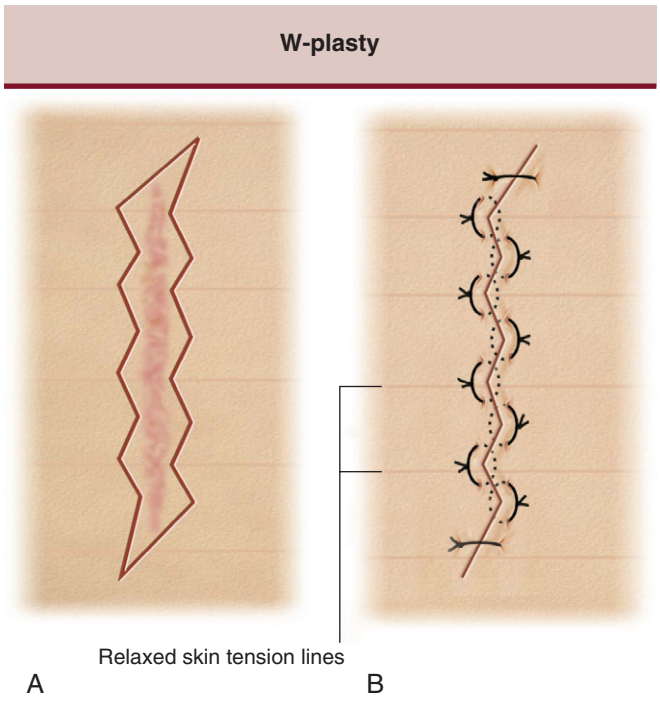


FIGURE 22-18 **A,** Markings for W-plasty should be in 1-cm segments drawn perpendicular to the long axis of the scar, with limbs drawn parallel to RSTLs **(B)**.

long axis of the scar and the limbs are drawn using these marks and the RSTLs as a guide. The ideal length of W-plasty limbs should be approximately 5 to 8 mm. If the limbs are smaller than this, they become more noticeable and the goal of camouflaging is lost. Limbs longer than 7 mm become aesthetically unsightly. After prepping and draping the area, and atraumatically injecting local anesthetic, a no. 11 blade is used to excise the limbs. The limbs are incised in a punch manner, directing the tip of the blade toward the scar to help prevent undesired damage to healthy tissue. No part of the pattern should cross the scar. A running set of triangles, alternating with their base toward and away from the scar, should be drawn out. The angle between limbs should be between 50 and 60 degrees and oriented as closely as possible to the RSTLs. Wider angles will not appreciate the elasticity throughout the excision seen with more acute angles. Narrower angles compromise blood supply to the tip of the triangle. To avoid a repeating pattern, the surgeon should slightly vary each triangle. Each proposed limb incision should be marked as close to the scar as possible to minimize tissue loss. Triangles along the scar should have their points facing the midbase of the triangles on the other side. It is easiest to begin marking each incision centrally on one side of the scar and then its counterpart on the other side of the scar in sequence. Unlike doing all of one side and then the other, this maintains wound tension and allows more efficient excision. This should continue toward the end of the incision, finishing with a small right triangle with its base perpendicular to the long axis at the end of the scar. Incision design in this manner will create a 30-degree closing angle of the wound and help minimize a standing cone deformity.

Hemostasis should be obtained before closure. In longer wounds, it may be useful to place several temporary skin sutures to align flap edges before placing dermal sutures. Often, a fusiform incision may be needed at the end of a W-plasty to reorient any anti-RSTL lines that have formed during closure or to excise any standing cone deformity.[131] It is essential to evert wound edges at the dermal layer with interrupted absorbable sutures placed at the midpoint of the tips of each point of the triangles (Fig. 22-19). Alternatively, running subcuticular monofilament absorbable sutures may be used. Skin may be closed with running absorbable or nonabsorbable sutures, with careful attention to evert wound edges. Postoperative wound care (see earlier) should be used. To prevent scar widening, Steri-Strips may be placed across the wound for 4 to 6 weeks after surgery because wound tensile strength progressively increases during this period. Dermabrasion or laser resurfacing may usually be performed 6 to 8 weeks after the W-plasty surgery to blend the scar with adjacent tissues.[132]

Z-Plasty

There is debate in the literature about who reported the first Z-plasty. Many credit Denonvilliers with describing the first Z-plasty in 1854 for a case of lower eyelid ectropion in a patient following a shrapnel injury.[133] Others argue that the first true Z-plasty was reported by Berger in 1904.[134] The goal of Z-plasty is to take a scar perpendicular to RSTLs and align the axis of the central

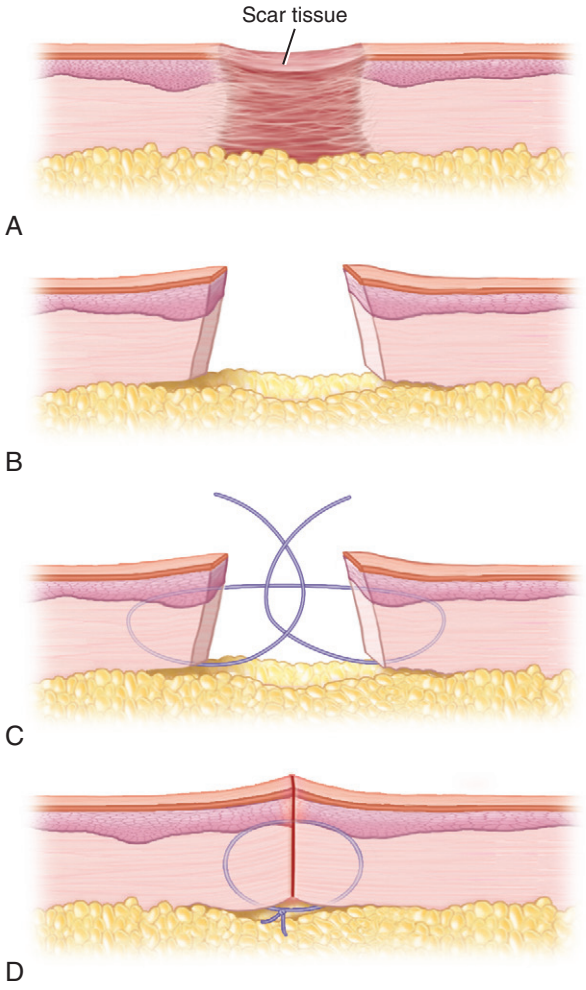


FIGURE 22-19 **A,** The scar is excised and undermined down to the level of the subcutaneous layer **(B). C, D,** The dermis is approximated, with eversion of its edges.

diagonal limb parallel to RSTLs (Fig. 22-20). Then, using triangular flaps, a contracted or webbed scar can be lengthened, a depressed scar may be leveled, or a long scar may be divided into multiple shorter segments. Its use is optimal around the mobile parts of the head and neck, such as the eye,[135] ear,[136] lip,[137,138] nose,[139] and intraoral mucosa scars caused by trauma and tissue loss (Fig. 22-21; see Fig. 22-17).

Unlike the actual letter Z, which has a diagonal longer than the other shorter limbs, the traditional Z-plasty has three limbs of equal length, with the central diagonal representing the original scar to be revised.[140] It is ideal for revision of the scar that runs parallel to ATL (see Fig. 22-20*B* and *C*). Its primary purposes are simply to rotate the long axis of the scar to a favorable position within the RSTL, lengthen a contracted scar, and align misaligned scars around important facial landmarks. The peripheral limbs are parallel and the angles of divergence from the diagonal should be equal and lie as close to RSTLs as possible. The peripheral limbs should be of equal length and at an angle of 60 degrees from the central limb (Fig. 22-22).[141] This angle will give the best balance for optimizing flap rotation and lengthening and for tension-free closure while limiting flap tip necrosis and the possibility of a standing cone deformity at final closure. The smaller the angle between the central and peripheral limbs, the less chance that the resultant scar will lengthen and a smaller amount of rotation will be achieved. Also, the chance of necrosis of the skin flap tip increases as the angle decreases. Therefore, the angle should never be less than 30 degrees. With a larger angle, more flap rotation and longer resulting scar length is achieved; however, with larger angles, more tension is placed on the wound at closure and may cause a standing cone deformity. Angles that exceed 70 degree are more likely to result in a standing cone deformity. Increase in scar length is usually less than predicted. This angle, however, can vary between 30 and 90 degrees. Generally, 30-degree Z-plasty achieves a 25% increase in length of the axis to the central limb, a 45-degree Z-plasty achieves a 50% increase, and a 60-degree Z-plasty achieves a 75% increase (Fig. 22-23).

Technique. Z-plasties should be planned and marked ahead of surgery, if possible, using a set of Castroviejo calipers.[142] If the central diagonal scar is wide, it should be preserved and transposed rather than excised to prevent recurrent scar widening. Similar to the W-plasty, a no. 11 blade should be used in a punchlike fashion to create skin flaps, moving from side to side and undermining each flap, as described earlier. Like other techniques, eversion of wound edges and management of the standing goal deformity will optimize wound esthetics. Closure should be started with tension-free placement of interrupted, subcuticular, dermal absorbable monofilament sutures, with eversion of the dermis. Skin is then closed using horizontal mattress, or running locked absorbable gut, or nonabsorbable monofilament sutures. Like previous techniques, wound edges should be held with skin hooks with only minimal tension, not forceps. Hemostasis must be achieved before closure and appropriate postoperative care should be used (see earlier).[143]

Many variations of the traditional Z-plasty have been described. Standard Z-plasty is often referred to as the stereometric Z-plasty because it will often alter tissue depths, producing elevations and depressions. Roggendorf[144] has described a planimetric Z-plasty that produces scar lengthening without the surface irregularities accompanying the traditional Z-plasty.[145] The planimetric Z-plasty include 75-degree transposition angles, a central limb half the length of the lateral limbs, and a geometric pattern area of excision to eliminate excess scar tissue. Using this technique, the peripheral arms are longer than the central limb, which is compensated for with two excisions on either end of those arms. making the angles more obtuse than with a standard Z-plasty (Fig. 22-24). The technique can be adapted to local skin conditions, deepithelialized excess skin can be buried to provide underlying wound support, and scar line can be varied more easily, as needed, than with a traditional Z-plasty, giving the surgeon more variety.

A multiple Z-plasty is a useful scar revision for the trapdoor or gouge defect, curved depressed scars, when there is inadequate tissue for a large Z-plasty or when the arms of a single Z-plasty would encroach on another

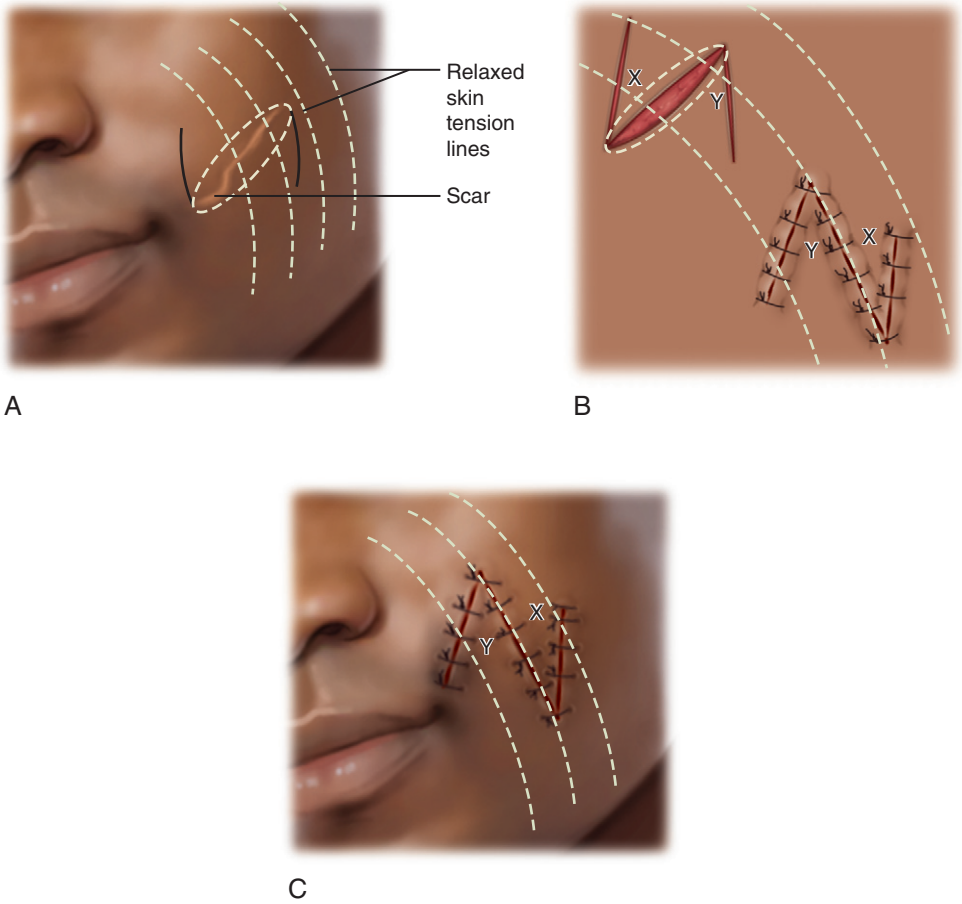


FIGURE 22-20 **A,** A Z-plasty is designed so that the central limb (the scar) of the Z-plasty **(B)** is reoriented to a plane parallel with RSTLs **(C)**.



FIGURE 22-21 Hypertrophic scar with lower lip retraction. A Z-plasty is used to excise and reorient the scar to elevate the lower lip and improve lip competence. **A,** Preoperative appearance. **B,** 8 weeks postoperative appearance.

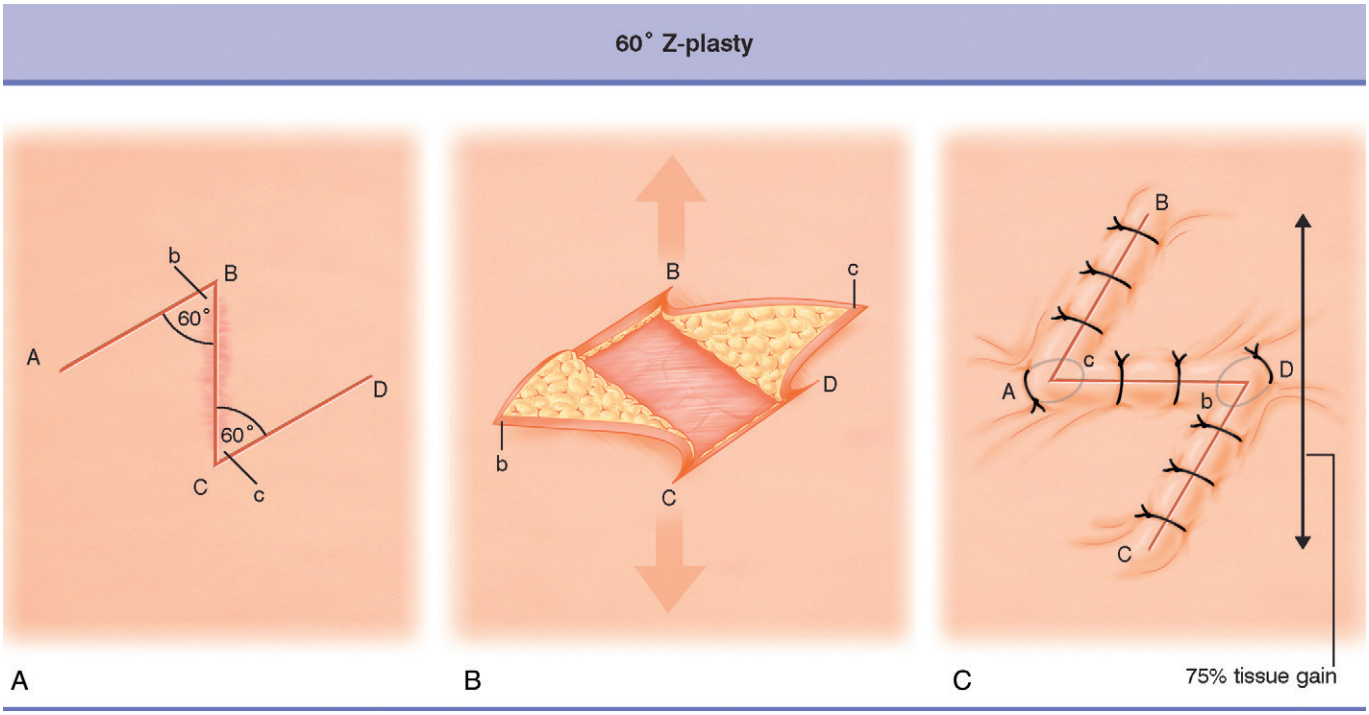


FIGURE 22-22 **A,** Peripheral arms designed at an angle of 60 degrees from the central limb will give the best balance for optimizing flap rotation and lengthening **(B)** and tension-free closure while limiting flap tip necrosis and the possibility of a standing cone deformity at final closure **(C)**. (From Robinson J: Surgery of the skin, St. Louis, 2006, Mosby)

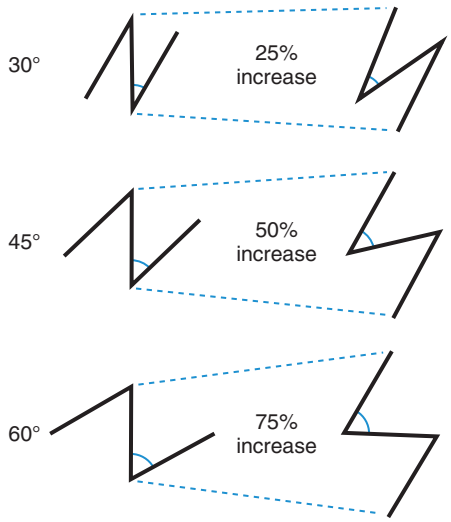


FIGURE 22-23 In general, as the angle between the peripheral and central limb increases, so does the length of the resulting scar.

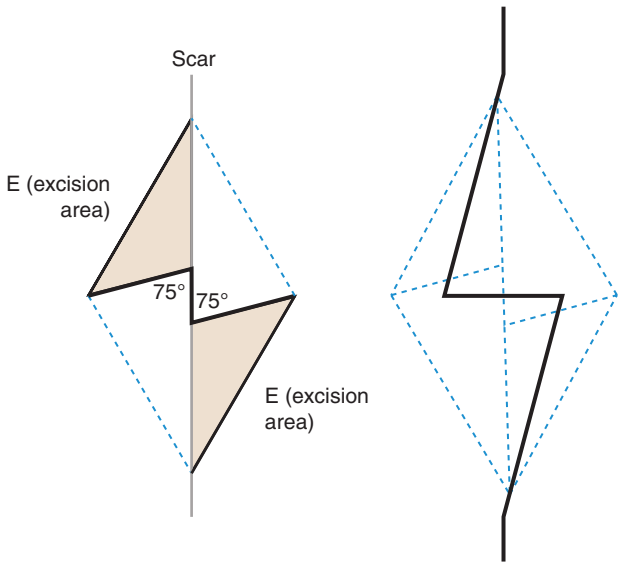


FIGURE 22-24 Planimetric Z-plasty. This includes 75-degree transposition angles, a central limb half the length of the lateral limbs, and a geometric pattern area of excision to eliminate excess scar tissue.

facial subunit (Figs. 22-25 and 22-26). This technique has less tension on closure and less surface depression, elevation, and lateral distortion. It may provide even more scar lengthening and camouflage than the traditional Z-plasty because of the smaller peripheral limbs used. The surface depression, elevation, and lateral distortion seen with a large, single-unit Z-plasty are lessened when multiple units are used. Multiple small Z-plasty units may achieve additional scar lengthening and result in improved scar camouflage as compared with a single Z-plasty. A drawback of multiple Z-plasties is the creation of more scars. This may be avoided by using serial Z-plasties for oblique scars of equal dimensions. Serial Z-plasties can be arranged in continuity or can also be interrupted. These are particularly useful in situations in which there is not enough surrounding tissue for transposition if one large Z-plasty were to be designed.[143,146] For final closure, an elliptical or fusiform excision may be preferred to a Z-plasty if the original scar is close to

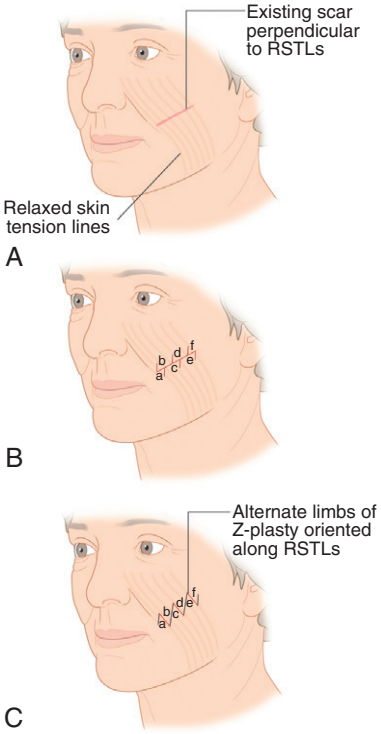


FIGURE 22-25 **A, B,** Multiple Z-plasties may be designed with the peripheral limbs at acute angles to the central limb, reorienting multiple segments of the central limb to RSTLs of the face **(C)**. (From Arndt K: Procedures in cosmetic dermatology series: Scar revision, Philadelphia, 2006, WB Saunders.)

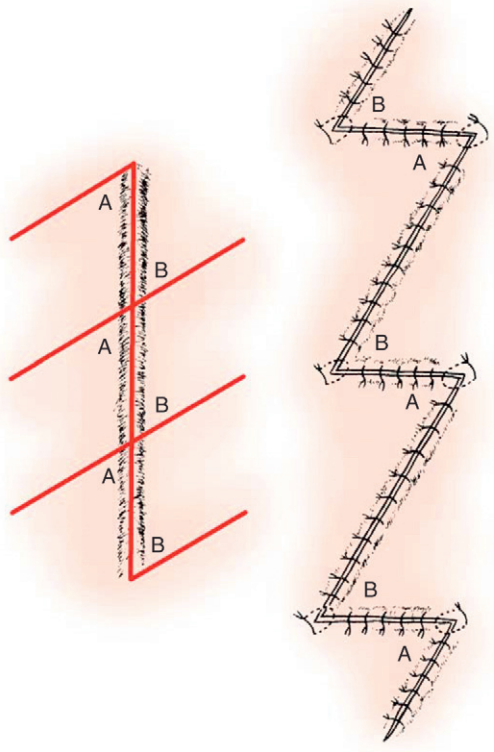


FIGURE 22-26 Final closure of multiple Z-plasties will reorient the divided central limb to the RSTLs. (From Canale ST, Beaty JH, editors: Campbell's operative orthopaedics, ed 11, Philadelphia, 2008, Mosby.)

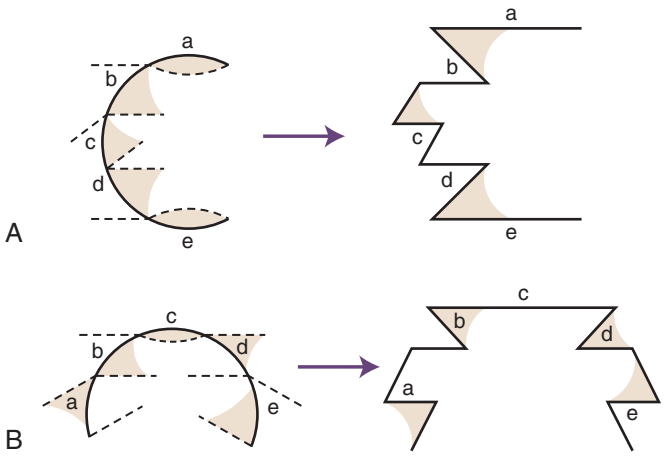


FIGURE 22-27 Various combinations of Z-plasties and fusiform excisions may be used, depending on the relationship of the defect to the orientation of the RSTLs. **A,** Two fusiform excisions and three Z-plasties are used for defects perpendicular to RSTLs. **B,** One fusiform excision and four Z-plasties are used for defects parallel to RSTLs.

being parallel to an RSTL; this technique that will avoid triangular flaps with very acute angles and probable skin necrosis. Borges has described a combination Z-plasty and fusiform excision for U-shaped or semicircular trapdoor deformities.[94] Segments of the scar parallel to RSTLs will benefit from fusiform excisions, whereas segments oblique or perpendicular to RSTLs benefit from Z-plasty. An example of this technique would include two fusiform excisions and three Z-plasties for defects perpendicular to RSTLs (Fig. 22-27*A*), or one fusiform excision and four Z-plasties for defects parallel to RSTLs (see Fig. 22-27*B*).

A variation of this technique has been described in which the distortion of small angled flaps is overcome by converting a two-flap Z-plasty to a four-flap Z-plasty.[147] It is especially useful in the webbed and functionally contracted neck. This technique involves marking a 120-degree angle Z-plasty and dividing each flap into two flaps, creating four equal 60-degree triangle flaps. This then allows for maximum lengthening of the scar without sacrificing ease of closure (Fig. 22-28). Before dividing a two-flap Z-plasty, the surgeon must make sure that the initial flaps are of large enough angles. Dividing flaps of questionable size may compromise blood supply and can result in necrosis to the flap tip. In general, 60 degrees should be the smallest angle used.

To gain maximum length of the final scar, another set of modifications has been described. This includes a 90-degree four-flap Z-plasty, lengthening the scar by up to 111%, and a 120-degree four-flap Z-plasty, lengthening the scar by 164%.[148] Alternatively, a 90-degree six-flap Z-plasty achieves 180% of flap lengthening (Fig. 22-29). The surgeon should begin with a simple 90-degree Z-plasty. If excess tension is encountered on transposition, converting to a four-flap Z-plasty is advisable. The six-flap Z-plasty may be considered when more length is desired and performed after a four-flap Z-plasty is marked and incised, or even after a 90-degree Z-plasty is designed, regardless of tension encountered.

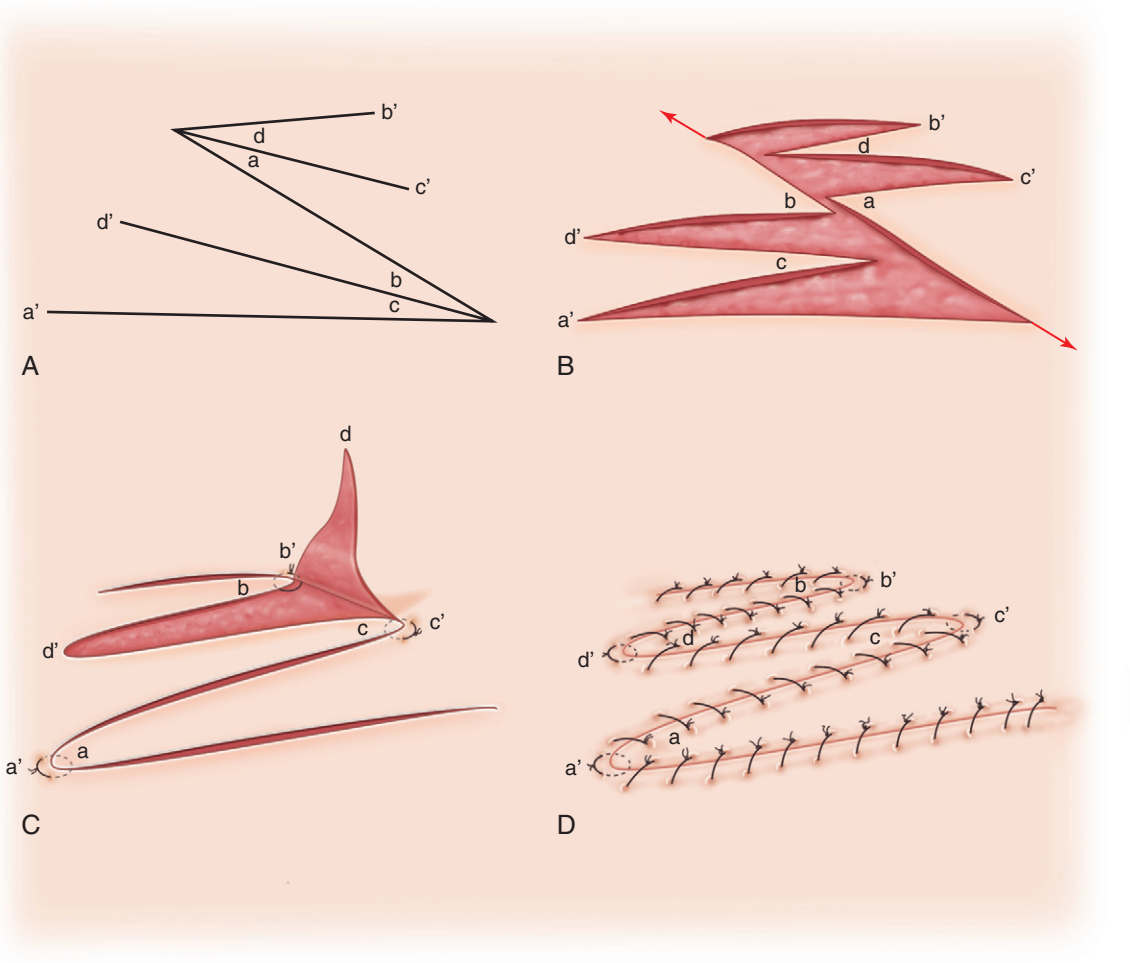


FIGURE 22-28 Two 120-degree angle Z-plasties are divided into two flaps, creating four equal 60-degree triangle flaps, which maximizes the lengthening of the scar without sacrificing ease of closure.

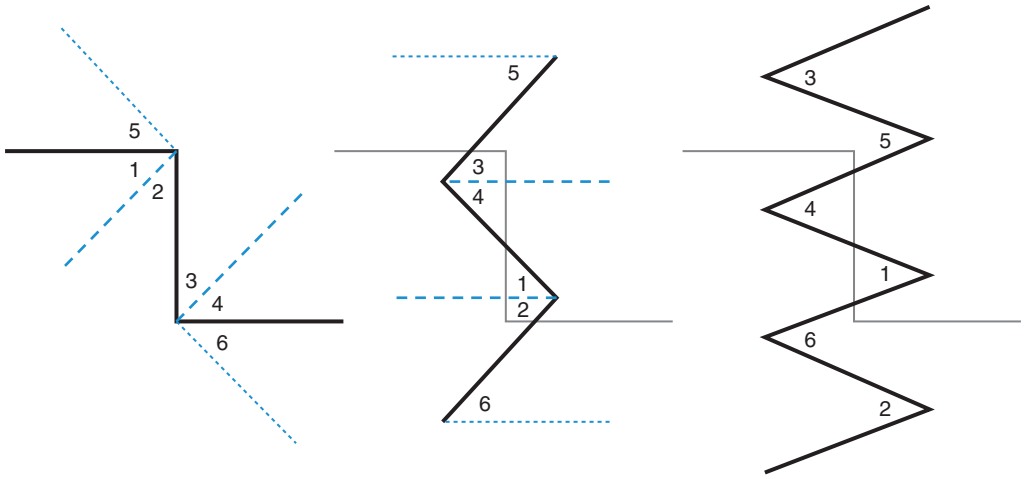


FIGURE 22-29 The peripheral arms of the six-flap Z-plasty may be up to 90 degrees from the central limb and may achieve up to 180% of scar lengthening.



FIGURE 22-30 Vertical scar band of the neck following neck dissection and pectoralis major muscle flap reconstruction treated with Z-plasty to lengthen tissue and improve mobility. **A,** Preoperative appearance. **B,** Postoperative appearance.

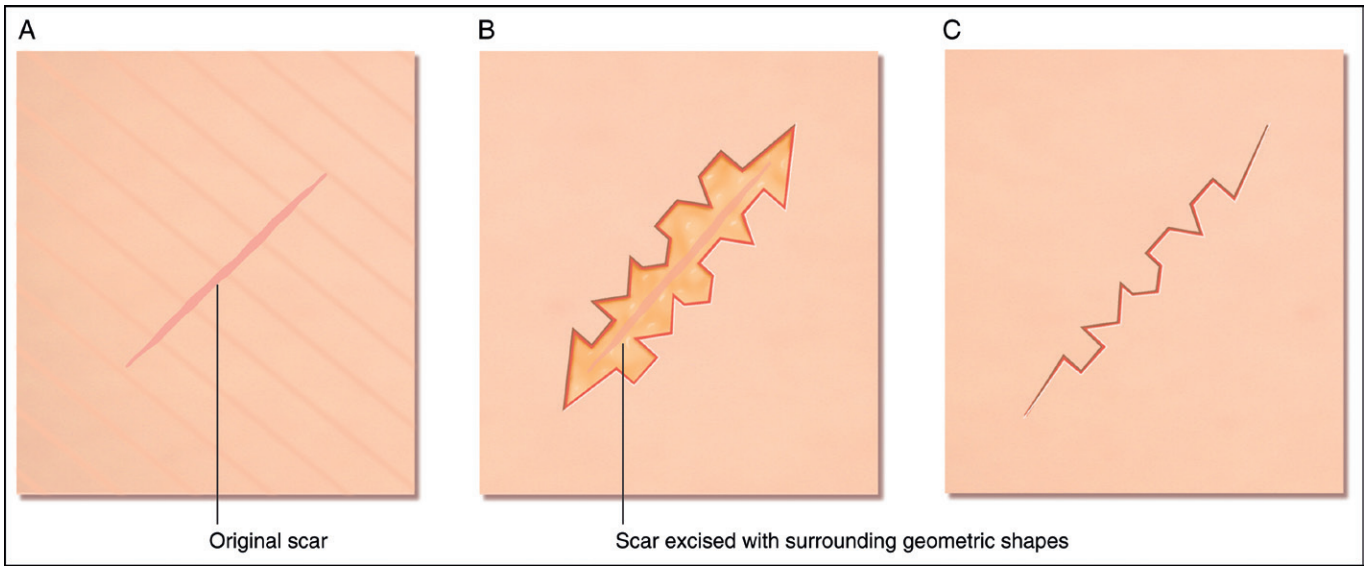


FIGURE 22-31 The GBLC technique is used for scars parallel to ATLs. The random pattern renders the scar less conspicuous. (From Arndt K: Procedures in cosmetic dermatology series: Scar revision, Philadelphia, 2006, WB Saunders.)

As an alternative to standard two-dimensional Z-plasties, a three-dimensional technique may be used. These types of Z-plasties are especially useful in the webbed neck (Fig. 22-30).[149] They can turn a cleft into a peak or depress hypertrophic tissue by incorporating tissue wedges into the transposed flaps. Several three-dimensional Z-plasties have been described, including a five-flap technique-s such as Converse's double-opposing Z-plasty, Numanoglu's paired five-flap Z-plasty, or Mustarde's jumping man flap. Building on these flaps is a modification by Hirschowitz that combines a five-flap Z-plasty with a V-Y advancement.[150-152]

Geometric Broken Line Closure

The regular repeating pattern of the W-plasty can detract from its value in scar camouflage, especially in longer scars. Initially described by Webster, the GBLC is similar to a W-plasty but relies on creating a combination of various geometric shapes (e.g., crescents, squares, triangles, rectangles, trapezoids) along a scar, with corresponding shapes on the opposite side to allow for precise closure, orienting most of the scar more parallel with RSTLs (Fig. 22-31).[153,154] In general, W-plasty and GBLC techniques are identical, with similar indications and contraindications. The resulting pattern from GBLC is

less predictable to the observer and thus is less noticeable than that of a W-plasty. In general, rectangles and squares produce superior results when kept small and as parallel to RSTLs as possible. Crescent shapes may result in small trapdoor defects and should be used minimally in the incision design. Each shaped segment should be between 5 to 7 mm in length. Smaller segments will resemble a straight line, whereas larger ones will result in a more unsightly scar.

There are several drawbacks to the GBLC technique. As compared with the W-plasty, it is conceptually more difficult to design, is significantly more time-consuming, and it lacks usefulness for curved scars. In addition, by nature of the design, some segments of the resulting scar will be at 90 degree to the RSTLs. Some investigators have questioned whether the outcome from GLBC is actually more aesthetically pleasing than from W-plasty.[155] Like the W-plasty, this technique should not be used for scars less than 30 degrees to RSTLs; these scars are better treated with a simple fusiform excision.

Technique. Preparation for GBLC is the same as for previously described techniques; incision design is the same as for a W-plasty, except geometric shapes instead of running W are marked out. A random pattern of geometric shapes, with most being rectangles and squares, are marked on one side of the incision and then the other to allow the original side to key into the opposing side on closure. The shapes should not exceed 7 mm in length in the case of rectangles, triangles, or squares or diameter in the case of crescents or circles. The design is finalized by placement of a right triangle at each end of the scar to produce a 30-degree closing angle of the wound, minimizing a standing cone deformity. Wounds should be closed with technique identical to that described for a W-plasty. Wound care after surgery and planned dermabrasion are the same as for a W-plasty. Similar to patients undergoing W-plasty, patients undergoing GBLC should be warned that they will experience moderate to severe erythema for several months before the scar will turn pale. Dermabrasion should be planned as early as 6 weeks postoperatively, just after the proliferative phase of wound healing and just before scar contracture begins.[115,117,119]

COMPLICATIONS

Scar revision requires meticulous planning, precision, patient cooperation, and surgical skill. Even when all these factors are present, errors may occur; therefore, it is imperative that the surgeon optimize the patient's chance for success. Diagnostic, planning, and execution errors can result in catastrophic results following scar revision. The most common complications include hematoma, infection, necrosis, hyperpigmentation, distortion of facial landmarks and their function, and recurrent scar widening or contracture. Because of not only the functional and aesthetic results, but also the psychological repercussions of these adverse outcomes, the surgeon is encouraged to try to prevent any unforeseen complications from occurring. Inevitably, it is not the complication, but its management that will predict the final outcome.

HEMATOMA

The prevention of hematoma begins with the initial preoperative visit, obtaining a comprehensive medical history, history of medication, vital signs, social history, psychological status, and normal activity schedule.[156] Patients on medications affecting the coagulation cascade or platelet function, such as acetylsalicylic acid or warfarin (Coumadin), are at high risk for hematoma formation within the first 14 days after surgery.[157,158] Patients with cardiovascular comorbidities may forget that they are taking these medications. A thorough history of bruising and bleeding should be obtained.[159] Other risk factors for hematoma include excessive alcohol intake and poorly controlled hypertension. These factors should be identified and the risks and benefits of surgery discussed with patient. Intraoperative factors associated with hematoma formation include hemodynamic instability and poor wound hemostasis. Postoperative factors associated with hematoma formation include increased postoperative activity, nausea, and vomiting and retching.

Prior to wound closure, the wound bed and skin flaps should be examined for any bleeding and meticulous hemostasis should be obtained using local techniques, such as ligation of vessels and electrocautery. Surgical clip placement should be avoided in the face because of the risk of their expulsion. All dead space should be eliminated by compressive closure of all wound layers. Drains should be used whenever dead space is suspected. Finally, pressure dressings should be used to minimize dead space formation edema from fluid accumulation. Small hematomas may be treated with needle aspiration,[156] whereas large hematoma should undergo formal wound exploration and all sources of bleeding should be identified and stopped. Failure to treat hematomas in promptly may result in infection, skin flap necrosis, and increased scarring.

INFECTION

Like hematoma, prevention is the best form of treatment for infection. A thorough medical history that includes questions about immunologic disease and immunosuppressive medications should be performed in the preoperative period. Infection following scar revision is rare and follows the rate seen with other cosmetic head and neck procedures.[160,161] Redundant tissue, electrocautery char, and excess suture may act as a foreign body and become a nidus for infection in patients.[162] When sterility is compromised, such as in procedures in which the oral mucosa is breached, lip and ear procedures, skin flaps on the nose, skin grafts, patients with extensive inflammatory skin disease, or a surgical site infection is present, administration of antibiotics should be considered.[163,164] Hematoma and seroma are also risk factors for infection, so dead space should be eliminated using the techniques described earlier. The type of prep used for surgery has been questioned with conflicting opinions about the superiority of chlorhexidine- based preps[165] versus povidone-iodine.[166] Swenson and Sawyer have concluded that the use of alcohol is key for preventing surgical site infections, regardless of the product used.[167]

NECROSIS

Fortunately, gross necrosis of skin following scar revision procedures is rare. Minor necrosis of skin flaps edges is not uncommon. This can be avoided using the techniques described earlier. It results from skin flaps with vascular compromise, including poorly designed skin flaps such as small skin flaps or flaps designed without consideration for an adequate blood supply, skin flaps without a broad enough base, excessive suturing of skin flaps, and placing too much tension on a wound at final closure.[168] In addition, complications such as hematoma formation from inadequate hemostasis or inadequate drain place and infection can also initiate skin flap necrosis.[169,170] Inadequate wound undermining and closure at the dermal level will help decrease wound dehiscence and necrosis. Medical problems such as Raynaud's disease may contribute to wound-healing difficulties and skin necrosis.[171] In a landmark study by Saran et al, it was concluded that a single cigarette reduces mean blood flow velocity to the fingers by 42%.[172] Smokers have a higher incidence of skin flap necrosis,[173-178] which has been demonstrated to be caused by the effects of nicotine, a vasoconstrictor, on local capillary blood flow.[179] Local exposure to noradrenaline does not seem to affect the incidence of skin flap necrosis.[180] In the trauma patient, previous embolization of major vessels caused by uncontrollable bleeding may compromise skin flaps in scar revision.[181]

Topical agents such as nifedipine, trolamine, and salicylate have been found to help salvage failing necrotic skin flaps. However, the efficacy of these agents needs to be explored further.[182] Other agents have been described for skin flap salvage.[183] Leech therapy has been shown to be beneficial in the treatment of skin necrosis secondary to venous congestion,[184-193] but its use is also associated with complications such as *Aeromonas* spp. infections and anemia that might require a blood transfusion.[194] The surgical treatment of gross skin necrosis involves débridement of all compromised tissue, allowing the remaining wound to heal by secondary intention. To minimize contracture, earlier skin graft placement, a local or regional flap, or even free tissue transfer may be performed to limit long-term aesthetic and functional impairment.

HYPERPIGMENTATION

Sun exposure is the most common cause of hyperpigmentation following scar revision. Hyperpigmentation caused by sun exposure is readily preventable; all patients should be educated to wear sunblock and protective clothing for the first 8 weeks following surgery. First- line treatment for hyperpigmentation includes the tyrosinase inhibitor 4% hydroquinone, twice daily.[195] Hydroquinone acts by blocking tyrosinase from developing melanin precursors for the production of new melanin. Essentially, it blocks new pigment formation as the new epidermis heals following a procedure. Secondary topical treatments include topical retinoids such as tretinoin, adapalene, tazarotene, mequinol, azelaic acid, kojic acid, and arbutin.[195] Over-the-counter remedies such as soy, licorice extracts, niacinamide, *N*-acetylglucosamine, and green tea have been shown to be beneficial in the treatment of hyperpigmentation.[196,197] Ablative nonsurgical treatments include chemical peels such as glycolic acid, salicylic acid, trichloroacetic acid, and Jessner's solution; laser therapy such as blue light photodynamic therapy, fractional photothermolysis, and neodymium-doped (Nd):YAG (yttrium- aluminum-garnet) laser have also been shown to be effective in treating hyperpigmentation.

DISTORTION OF MOBILE LANDMARKS

Trauma to mobile landmarks such as the lips, eyelids, or nose is not uncommon; scar revision is often difficult because of the constant muscle pull, tension, and motion present. Unless there is a preexisting vertical asymmetry, revision in a vertical vector should be avoided. Treatment must be tailored to the patient. In general, W-plasty should be avoided. Z-plasty is the preferred treatment around the eyelids, nose, vermilion, and neck because of excess tissue tension in these areas.[135,137-139]

RECURRENT SCAR WIDENING

Scar widening will often take place within several months after injury and may occur in a scar that appears to be healing without difficulty.[198] It occurs when opposing forces such as muscle pull, elastic forces of skin, and external pressure are applied to newly formed collagen before it reaches final maturity. It may take several months before completion of collagen maturation.[199]

Demographic factors associated with scar widening include increased age and race. Location of the scar and amount of volume loss have severe implications on scar widening. For example, scars around facial landmarks in constant motion or those under constant tension because of loss of local tissues are at risk for scar widening. Patients should be screened for heritable collagen diseases such as Ehlers-Danlos syndrome, osteogenesis imperfect, cutis laxa, Marfan's syndrome, homocystinuria, Menkes syndrome, focal dermal hypoplasia, tuberous sclerosis, familial cutaneous collagenoma, and epidermolysis bullosa, because these will contribute to postoperative scar widening. When it occurs, the cause of scar widening must be determined to manage the deformity correctly.[200]

Although there are many therapies available for hypertrophic scarring,[201-203] there are no time- proven interventions available to correct scar widening. Wide and hypertrophic scars resulting from straight linear incisions are difficult to treat surgically. The most common cause of scar widening is from excess skin tension caused by tissue volume loss. These defects are best treated with tissue transfer in the form of local, regional, or free tissue flaps, because excision and manipulation techniques such as fusiform scar excision, Z-plasty, or W-plasty often result in recurrent scar widening and increased scar length. In an attempt to provide support to skin edges in the widened scar, Millard has advocated leaving a base of scar tissue and suturing wound edges to the base to diminish opposing skin flap forces on each other.[204] In this technique, the area of scar is deepithelialized and then incised on one side down to subcutaneous tissue (Fig. 22-32*A*). Using a curved scissors, the healthy wound edge away from scar is undermined to at least double the

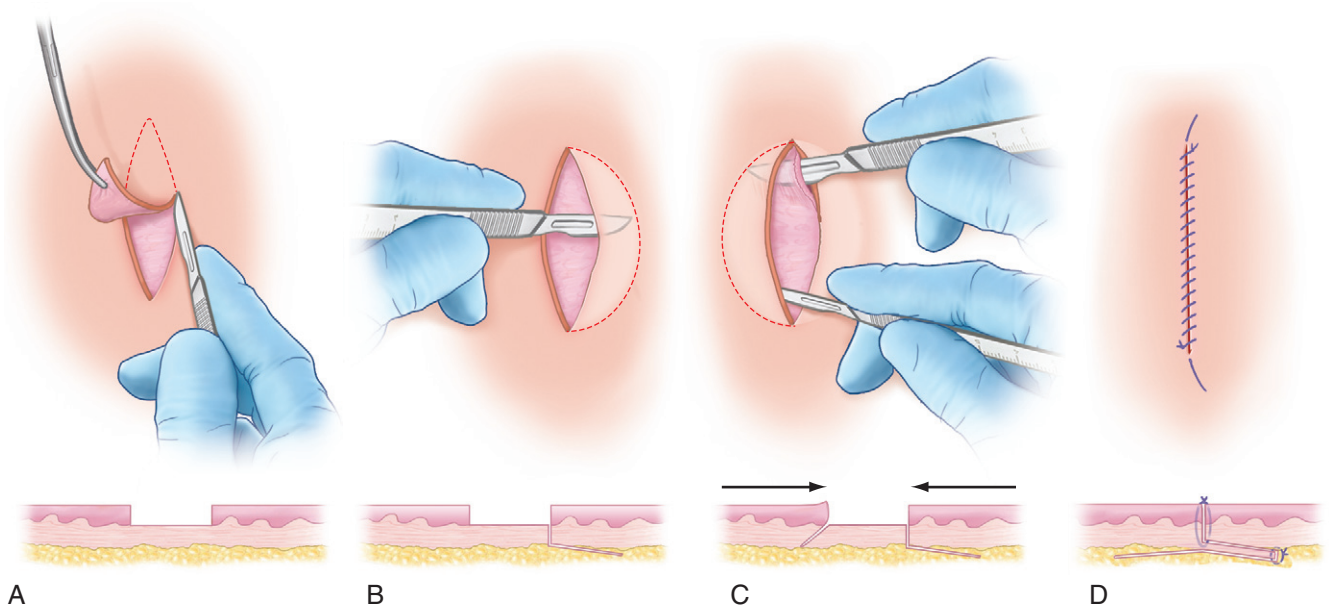


FIGURE 22-32 **A,** The epithelium of the scar is excised, leaving an exposed dermal sheet. **B,** A vertical incision is made through one edge of the dermal sheet, undermining that skin edge slightly more than the width of the scar. **C,** The dermal scar sheet is now undermined for a greater distance in the opposite direction (see *broken line* in cross-sectional view). **D,** An incision on the bias (diagonal) is made at the opposite edge and carried partially through the dermis to provide an edge equal to the opposite side.

width of the scar (see Fig. 22-32*B*). The dermal scar sheet is then undermined in the opposite direction (see Fig. 22-32*C*). An incision at a diagonal is made at the opposite skin edge and carried partially through dermis to provide an edge equal to the opposite side (see Fig. 22-32*D*). The dermal scar sheet is then advanced and sutured subcutaneously, taking tension off the new surface closure that is now sutured in a staggered location away from the deep closure. In the case of a depressed scar, the scarred dermal sheet may be advanced to fill the defect. Wilson has shown good success with this technique in a series of patients undergoing revision surgery for widened scars.[199]

RECURRENT SCAR CONTRACTURE

Scar contracture of the head and neck is an especially difficult problem because of the aesthetic changes and functional disability that it may cause in the patient. These types of scars are especially common in post-traumatic and burn patients. Scar release incisions should be placed in facial unit or subunit borders to aid in the aesthetic placement of skin grafts. Scar release may be performed by fusiform excision, Z-plasty, W-plasty, or straight line incision.

Smaller areas of contracted scar may be treated by the Y-V skin flap, avoiding the need for grafting.[205-213] A single or running Y-V flap will result in scar lengthening without flap transposition. The stem of the Y must be placed perpendicular to RSTLs. The scar release occurs at the stem of the Y and skin is then advanced, creating a V-shaped defect. The triangular flap of healthy skin is advanced to fill the defects (Fig. 22-33). Alternatively, a V-Y release may be performed, in which a V is incised and the tissue inside the V is advanced and used to bulk up an area and lengthen the long axis of the scar (Fig. 22-34).[214-216] Other simple local skin flaps may be used, including a single advancement, bilateral advancement, and rotational, sliding, bilobed, and rhomboid flaps.[217]

Scar release will often result in tissue loss and may cause tethering of skin to deep tissues, such as bone or muscle, severely limiting function. These scars are often caused by trauma, burns, or infection and will ultimately need tissue transfer in the form of a graft or flap. Small areas of scar contracture may be released with minimally invasive procedures such as subcision (subcutaneous incisionless surgery) and subsequent injection with autogenous or nonautogenous fillers. Scar subcision is used to treat contracted, atrophic, or depressed scars or deep wrinkles.[218-221] It should be attempted before using any filler material. It is most commonly done with a needle that acts as a scalpel and releases fibrotic strands within a scar, elevating the surface of the scar. Briefly, the needle is introduced at an angle into the skin and then manipulated back and forth in a lateral motion to break any fibrous bands. The placement of the needle should be meticulously planned in an orientation parallel to the underside of the dermis, because the sharp cutting edge is a threat to deeper vital head and neck structures. Care must be taken by the surgeon not to rotate the needle, so that its triangular tip stays horizontal to the skin surface. This procedure should be avoided in the preauricular, temporal, and mandibular areas to avoid injuries to the facial nerve and major vessels. Some standard principles include the following: individual scars should be treated using separate, multiple puncture sites and, when multiple scars are treated, the most dependent one should be treated first. This procedure may be repeated multiple times, with 3 weeks between sessions, and the procedure should be ideally performed before a weekend or holiday for working patients.[222] Following subcision, there is an organization of blood in the induced dermal

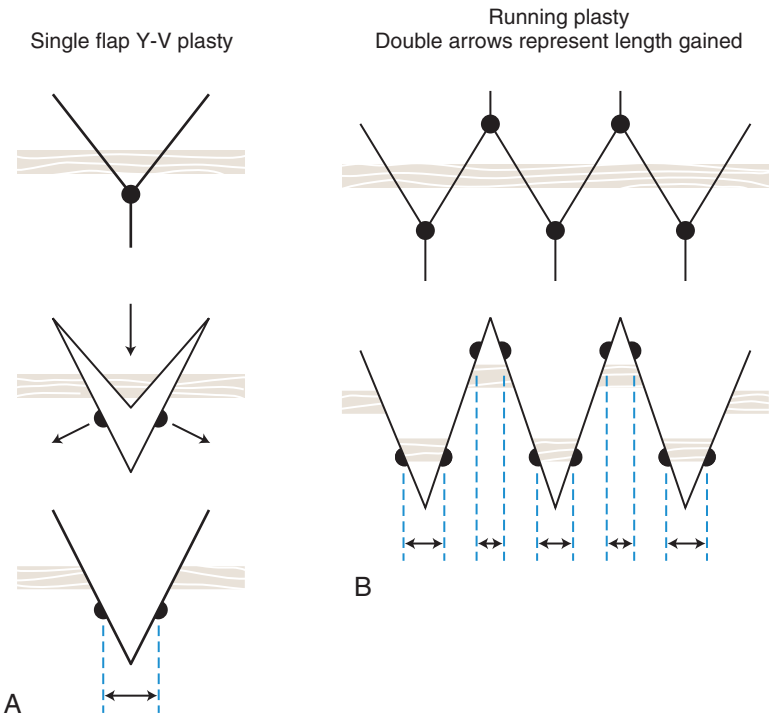


FIGURE 22-33 The V-Y flap can be used in single **(A)** or multiple **(B)** units.

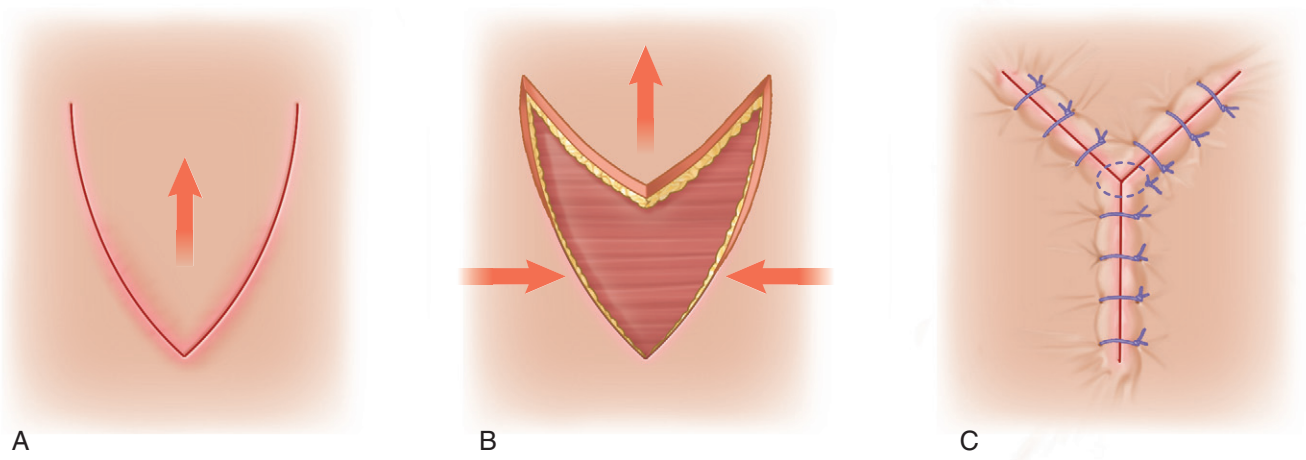


FIGURE 22-34 **A,** A scar is lengthened by making a V-shaped incision in the area of contraction. **B,** The flap is then advanced in the direction of the arrow. **C,** The final closure resembles the letter Y.

pocket, followed by connective tissue formation and collagenization. Patients should be counseled about temporary hematoma formation and bluish discoloration that will occur. This procedure has the advantage that it can be done in the outpatient setting under local anesthesia.

SPECIFIC TYPES OF SCARS

DEPRESSED SCAR

Scar depression usually occurs secondary to loss or atrophy of subcutaneous or dermal layers, and therefore is not amenable to traditional surgical techniques (see earlier). Management of the depressed scar involves restoring bulk to the subcutaneous and dermal soft tissue structures. Dermal restoration may be achieved by collagen injection, including bovine (Zyderm, Zyplast, Inamed, Santa Barbara, Calif) and autologous dermal collagen (Autologen, Collagenesis Corporation, Beverley, Mass) or, less commonly, by steroid injection.[223] Subcutaneous restoration of facial contour defect may be done with autologous fat transplantation,[103,104,224-226] although some have reported conflicting long-term results using these techniques.[227] Other options for soft tissue augmentation include autogenous dermal fat grafts and acellular dermal grafts (AlloDerm, LifeCell, Branchburg, NJ). These materials may result in a longer lasting improvement of depressed facial scars but they are implantable rather than injectable and may be

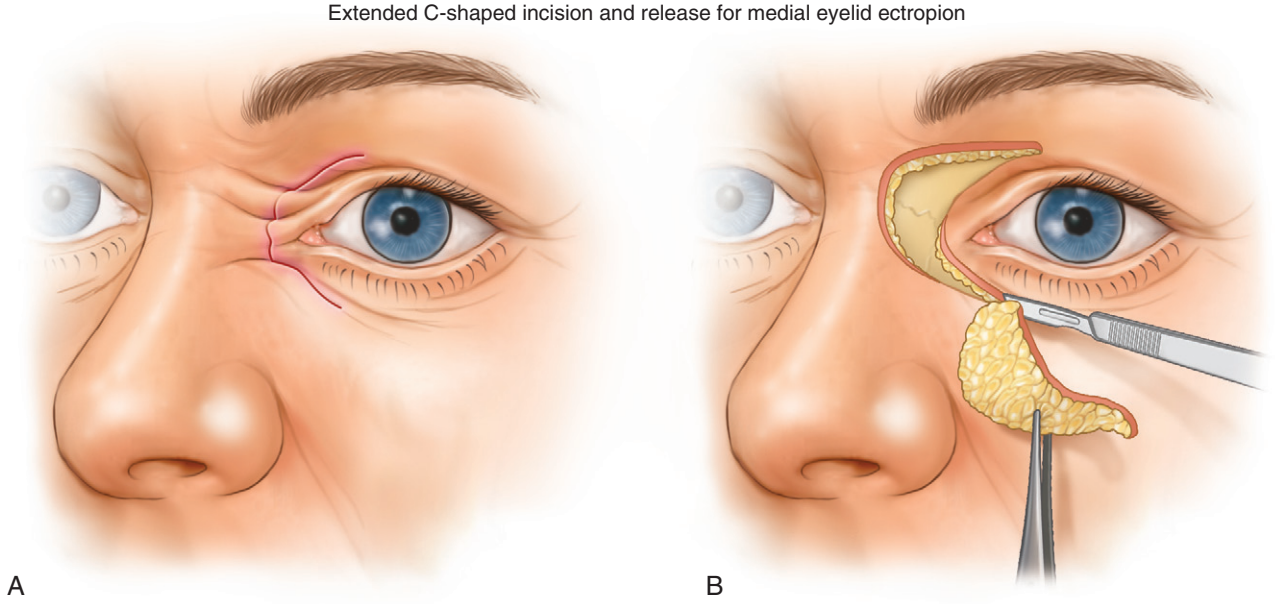


FIGURE 22-35 **A,** Extended C-shaped incision and release for medial eyelid ectropion **(B)**.

technically more difficult to use.[228] The use of these materials will be discussed later in the chapter. Previously described minimally invasive techniques, such as subcision, are also useful for the management of depressed scars.[218]

ELECTRIC BURNS

The treatment of electrical burns is challenging because the resulting scar often spreads from the skin at the point of contact to the periphery and generally has an indistinct end point of collateral damage.[229] In the head and neck region, these scars often occur when a child sucks on the female end or junction of an extension cord, or when a child chews on the end of a poorly insulated wire. Generated by a temperature of up to 2500 to 3000° C (≈4500° to 5400° F), electrical burns initially appear as a grayish-white coagulated lesion with an erythematous rim. After 2 to 4 weeks, an eschar will slough off, leaving an ulcerated area. This area will slowly be replaced by fibrous connective tissue and undergo maturation. In addition to unsightly scarring, fibrosis and scar contracture may occur with head and neck burns, which can be debilitating to the patient by causing trismus, dysphagia, dysarthria, or changes in facial expression.

Early splint therapy is important to relieve and prevent microstomia for patients with perioral burns. Patients will often need multiple débridements of scar tissue.[230-236] The initial surgical goal is to line up the vermilion. Then, if possible, fusiform scar excision or Z-plasty may be performed. Z-plasties may be used extraorally and intraorally to relive contracture and lengthen scars. Dermabrasion of skin may follow to relieve any skin surface irregularities.

EYELID ECTROPION

There are several types of eyelid ectropion, including congenital, senile, neurogenic, and cicatricial.[237-239] Cicatricial ectropion results from scarring of the anterior lamella, which consists of the skin of the eyelid and orbicularis muscle. It usually occurs as a result of facial trauma and may be associated with orbital fractures, facial burns, or chronic dermatitis, or as an iatrogenic result of excessive skin excision during cosmetic blepharoplasty. It may involve the upper or lower eye lids, or both. It may be medial or lateral and may shorten the eyelid vertically or horizontally, or both. In general, the goal of ectropion repair is to restore the original size and position of distorted tissues and to replace lost tissue with similar tissue.[240] Anterior lamellar reconstruction is best achieved using full-thickness skin grafts or local skin flaps.

Extrinsic medial ectropion may be caused by scarring of the nasal dorsum, resulting in anterior and medial pull of the medial canthus. The result is epicanthal folding, exposure of the cornea, and epiphora. Scar release in this area involves a C-shaped excision of scar tissue from the upper and lower eyelids and lateral nose (Fig. 22-35). The resulting defect is then grafted with a full- thickness skin graft. Skin over the nose adjacent to the release incision should be undermined to allow maximal release; the periosteum overlying the frontal process of the maxilla is excised to allow for graft adherence to bone. The use of a full-thickness skin graft is preferred here to minimize contracture. In addition, medial canthopexy may be performed, if needed.[241]

As with other mobile facial landmarks, the Z-plasty is optimal for treating scarring around the eye and should be considered for the treatment of lateral ectropion (Fig. 22-36). The advantage of the Z-plasty is that it transposes retracted lower eyelid tissue superiorly and posteriorly, resulting in better adaptation of the lid margin to the globe (Fig. 22-37).[133,242]

Intrinsic contracture of either eyelid and associated vertical shortening may be approached with a supraciliary or subciliary incision.[243] The incision should extend just short of the lateral and medial canthi. Skin flaps are raised and the upper or lower eyelid is approximated.

The skin flap is then undermined and the underlying stable pretarsal orbicularis oculi is used as a graft bed. A full-thickness skin graft is applied in the area of tissue loss. The advantage of this technique is that the tarsus acts as a splint, limiting contraction of the graft, and the position of the lid crease may be limited by the position of the incision. The eyelids may be separated immediately or in a delayed fashion. If indicated, a C-shaped release of skin can be combined with the approach.[241]

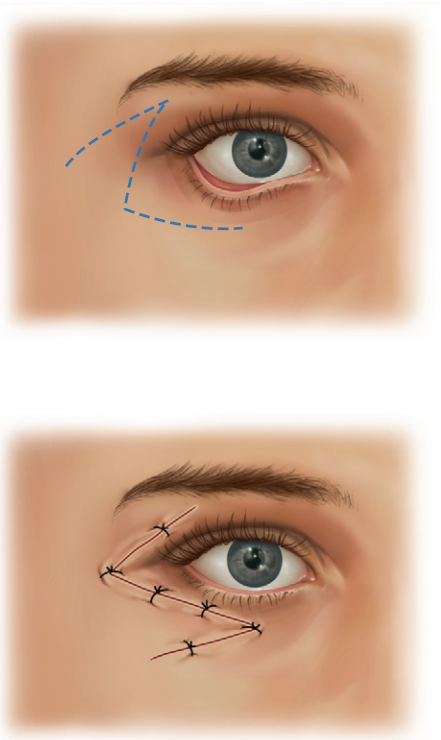


FIGURE 22-36 Z-plasty for lateral lower eyelid ectropion, transposing the retracted lower eyelid tissue superiorly and posteriorly, and acquiring better adaptation of the lid margin to the globe.

Unlike upper eye lid ectropion, which usually is caused by scarring in the forehead and temples and is generally repaired with scar release and grafting, lower eye lid ectropion repair is more complex. Restoration of its underlying tissues is crucial to prevent recurrence. The lower eyelid has almost no excess tissue, so only a small degree of scarring may cause retraction of the lower eyelid.[244] A number of techniques have been described to correct lower eyelid ectropion. Management should be tailored to the specific injury (Fig. 22-38). One approach involves subperiosteal release of all extrinsic and intrinsic components, with mobilization of the lower eyelid and grafting as necessary. A subciliary incision with a lateral canthotomy is performed, occasionally combined with a transoral vestibular approach, which facilitates subperiosteal dissection from the zygomatic arch to the pyriform rim, down to the alveolus. After release of the lower eyelid, a middle lamellar graft harvested from the hard palate or conchal bowel is secured superficially and inferiorly to the tarsal plate, if necessary. The orbicularis oris is then dissected, redraped over the lower eyelid, and sutured to the lateral orbital rim and lateral nasal wall. Medial and lateral canthopexy is completed, and a skin graft is placed.[245,246] If skin is missing, a variety of local skin flaps may be used in lieu of skin grafts .[242,247,248] In addition, autologous or allogenic dermis–only grafts may be used for the correction of lower lid retraction in which a spacer is needed but stiffness is not a concern. These grafts are associated with low donor site morbidity, but may undergo more contracture than split-thickness skin grafts. The drawback of acellular allogenic dermis grafts is that they are associated with significantly more



FIGURE 22-37 Post-traumatic cicatricial ectropion, managed with scar excision and primary skin grafting with Z-plasty and lateral canthopexy. **A,** Initial post-traumatic appearance, age 8 years. **B,** Preoperative appearance, age 13 years. **C,** 1 year postsurgery, age 14 years.



FIGURE 22-38 Lower eyelid ectropion and retraction following repair of comminuted orbital fracture and associated periorbital lacerations. Ectropion repair consisted of transconjunctival cantholysis and orbitotomy, removal of previously placed hardware, subperiosteal midface lift, and lateral canthopexy. **A,** Preoperative appearance; **B,** 6 months postoperatively.

contracture than full-thickness skin grafts and are show significant degration by 4 months postoperatively.[249,250] For conjunctival reconstruction, the use of amniotic membrane transplantation has been described, with some success.[251,252] The advantages of these grafts over oral mucosa grafts include less donor site morbidity and the structural arrangement of collagen and lamina is similar to that of the conjunctiva, providing an excellent scaffold for proliferating conjunctival cells.

HYPERTROPHIC SCARS AND KELOIDS

Keloids are defined as scars that extend beyond the edges of the original wound or incision, invading surrounding normal tissue,[253,254] and affect an estimated 1.5% to 4.5% of the population.[255] Hypertrophic scars, on the other hand, are elevated scars that remain within the original tissue injury site.[256,257] Both hypertrophic scars and keloids result from alterations in normal cutaneous wound healing that include the proliferation of the dermal tissue and excess deposition of fibroblast-derived extracellular matrix (ECM).[258-260] In normal wounds, there is a decrease in cellularity mediated by apoptosis during the transition from granulation to scar tissue. In hypertrophic scars, however, granulation tissue does not regress and alpha smooth muscle actin-expressing myofibroblasts produce excess ECM, resulting in a red, raised rigid scar.[261,262] These scars may develop only weeks after injury. Dark-skinned individuals are at increased risk for keloid formation, with the earlobe and cheek being the areas usually affected.[263] Keloids may be symptomatic and cause pain, but hypertrophic scars may be painless and fade over time.

Surgical excision of keloids is characterized by recurrence and is generally avoided in the absence of pain or dysfunction.[80] If excision is performed, care should be taken so that the tissue is handled in an atraumatic manner, using skin hooks to minimize trauma to skin flaps, and results in a layered, tension-free closure.

Radiation therapy with or without surgical excision has been advocated for the treatment of keloids. One reported protocol has called for keloids of the earlobe to receive 10 Gy in two fractions over 2 days, and other sites of the head and neck to receive 15 Gy in three fractions over 3 days. Surgical excision is performed and adjuvant radiation therapy is initiated on postoperative day 1 or 2. The reported recurrence rate with this protocol was 14% at 18 months of follow-up. A side effect of this procedure is hyperpigmentation of the irradiated area. To prevent this complication, investigators have recommended that steroid ointments be applied to the surgical site, reducing the radiation dose, or increasing the time between radiation sessions.[264] Malignant transformation is rare but has been reported.

The use of lasers has been advocated for the treatment of keloids and hypertrophic scars. Although the argon, CO_2,[265,266] and Nd:YAG lasers[267,268] have been found to be of little benefit, the flashlamp-pumped pulsed dye laser (PDL) has shown good results, with significant improvements in contour, texture, color, pliability, and pain relief and minimal side effects.[57,269-271] Early PDL treatment may fundamentally change the physiology of wound healing by reducing scare microcirculation and preventing excess scar formation.[272,273]

Intralesional corticosteroid injection is a common treatment modality for keloids and hypertrophic scars.[274] It has been shown to reduce scar volume significantly and increase scar pliability and height while reducing symptoms such as pruritus and pain in the patient with keloids.[57,275] It works by three primary mechanisms: (1) by suppressing inflammation by inhibiting leukocyte and monocyte migration and phagocytosis; (2) by causing vasoconstriction, therefore reducing the delivery of oxygen and nutrients to the wound bed; and (3) by exhibiting an antibiotic effect on keratinocytes and fibroblasts, slowing reepithelialization and new collagen formation.[276] Insoluble triamcinolone acetonide (10 to 40 mg/

mL) is the most common steroid used in scar treatment.[268] The use of a topical and/or local anesthetic is recommended prior to injection. The steroid is ideally injected into the superior dermal layer, the papillary dermis, which is where steroids promote collagenase activity. Injections may be repeated twice monthly and may be combined with surgical excision. Following surgical excision and primary closure, steroid may be injected into the wound and its edges.[277] Side effects include short-term hypopigmentation and injection pain.[57,278] Long-term side effects include skin and subcutaneous fat atrophy, telangiectasias, scar widening, and rebound effects.[279] Topical steroids have not been shown to be effective for the treatment of hypertrophic scars or keloids.[280,281]

Silicone gel has shown some success in the treatment of hypertrophic scars and keloids. First used in the 1960s,[282] and rediscovered in the 1980s[253,283-285] for the management of burn scars, silicone materials, including creams, gel sheets, Silastic sheets, and garments, have become popular for the prevention and treatment of hypertrophic scarring and burns. Their use has been shown to reduce scar height and hardness, and increase scar pliability; they may be used for several years after injury.[47,49,286-288] The usefulness of silicone sheeting may be greatest in children because it is noninvasive and painless. Better effects have been shown with early treatment and their use should be started 2 weeks after wound healing. Treatment usually lasts 6 to 12 months; patients are advised to wear the silicone material for the entire day and only remove it for cleaning. Each sheet lasts 2 to 3 weeks and the adherent types seem to work better than others.[279]

A review and meta-analysis has assessed the effects of silicone sheeting in preventing and treating hypertrophic scars and keloids following surgery, using the pooled results of randomized controlled trials (RCTs).[289] The investigators found that silicone sheeting reduces the incidence of keloid and scar formation by approximately 55% in patients prone to scarring following surgery when compared with no treatment, and that silicone sheeting produces a significant reduction in scar thickness while significantly improving scar color. However, it was noted that most of the RCTs studied were of poor quality and possibly biased. Although most research has focused on the efficacy of gel sheets, semiliquid gels have also been evaluated. Their advantage is that they are composed of a clear material that adapts to the underlying skin, producing a more inconspicuous appearance than the sheets.[253] Silicone gel may be used for 12 to 24 hours, washed off, and reapplied.[290] Because silicone sheets have a water vaporization rate lower than that of skin, water accumulation may cause skin maceration.[291] Other complications include pruritus, skin breakdown, skin rash, a foul smell from the gel pad, and poor durability of the sheet.[282,292]

Other nonsurgical options for keloids and hypertrophic scars include dermatography,[268,274] intradermal epicatechin gallate injections,[293] the ThermaCool TC system, which uses radiofrequency waves (Thermage, Hayward, Calif),[294] adhesive tape support, subdermal injection of interferon-α2b (IFN-α2b),[295,296] verapamil,[296,297] hyaluronic acid,[298-300] 5-fluorouracil,[301,302] collagenase,[303] bleomycin,[304] madecassol and alpha Centella cream,[305] imiquimod, tacrolimus, onion, adhesive tape support, vitamin E, and massage.[256,264,269] Pressure garments have been popular in the past, but studies have shown these treatments to be minimally effective.[306] Furthermore, their use is associated with problems such as nonadherence, patient discomfort, eczema, rashes, pruritus, and ulceration from excessive pressure and friction.[307]

Massage of the scar has also been advocated as a method of minimizing scar prominence. Keloids and hypertrophic scars contain a considerable amount of ground substance, which is composed of glycoproteins and glycosaminoglycans. This ground substance is fluid and can be displaced by pressure; therefore, massage of these scars can reduce swelling from a reduction in ground substance. The reported benefits of massage include increased scar pliability and decreased scar banding[308]; however, there is little evidence to support these findings.[309] Scar massage appears to have little to no effect on the vascularity, pliability, and height of the hypertrophic scar but it has been shown to reduce pain and itching.[310,311]

Emerging nonsurgical therapies are being developed that exploit an association of TGF-β with hypertrophic scar formation[57,312,313] in an effort to alter the scar formation cascade. Cryotherapy with liquid nitrogen causes ischemic necrosis and has shown to improve or completely regress keloids in significantly 51% to 74% of patients after two or more sessions.[274,314-316] To avoid potential drawbacks of classic cryotherapy, such as skin atrophy, pain, and hyperpigmentation, intralesional needle therapy has been developed and shown to be effective in the alleviation of symptoms.

Shave excision has been shown to be an effective treatment for keloids[317,318] and may be considered in elevated scars.[319] The purpose of shave excision is to flatten the scar so that it is level with surrounding tissues, making it less noticeable. It may be performed with a scalpel or a razor blade (Fig. 22-39). The wound is allowed to heal by secondary intention. It can be combined with dermabrasion or laser resurfacing. It has been found to be an exceptionally effective treatment when combined with 5% imiquimod cream for keloids of the ear. Following shave excision, patients are treated with imiquimod 5% cream nightly for 2 weeks and then three times weekly under occlusion for 1 month. Shave excision is associated with complications such as a high rate of recurrence, scar depression, and hyperpigmentation.[320] Mustoe et al have provided an algorithm for the management of various types of hypertrophic scars[57] (Fig. 22-40).

Despite much interest and a century of work on the problem of keloids and hypertrophic scarring, results of treatment remain generally unsatisfying. A meta-analysis by Leventhal et al has concluded that all currently available treatments for keloids and hypertrophic scars demonstrate only minimal improvement when compared with no treatment.[80] In addition, they found no difference in scar improvement among any of the treatment modalities assessed.

RESURFACING PROCEDURES

Facial skin resurfacing is performed to treat scarring above the skin level, scars with an irregular surface area,

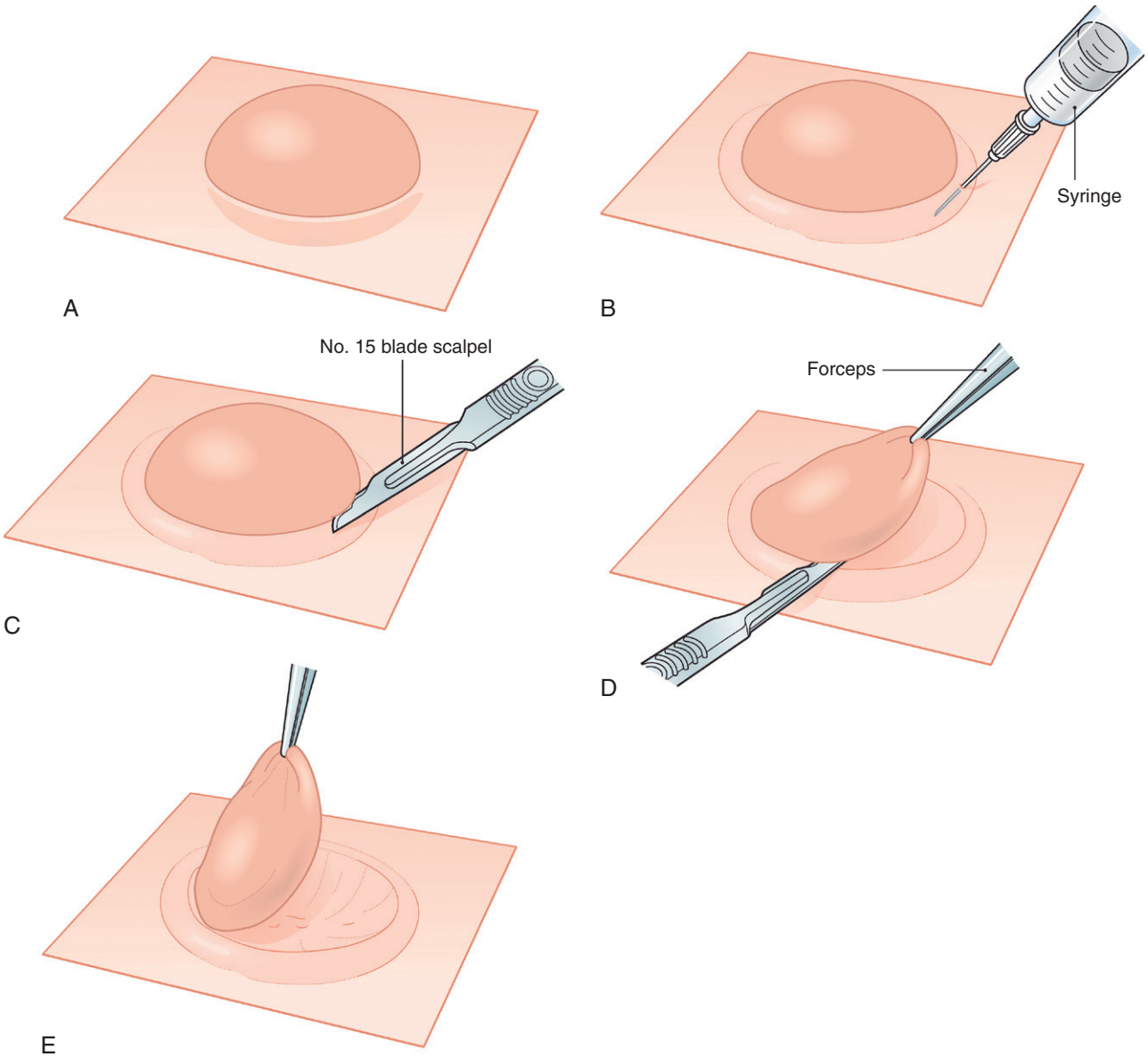


FIGURE 22-39 **A,** An elevated scar should be anesthetized with local anesthesia at the periphery of the lesion and, using a scalpel or razor blade **(C)** and forceps **(D),** the lesions is excised **(E).** (From Nouri K, Leal-Khouri S: Techniques in dermatologic surgery, St. Louis, 2003, Mosby,)

acne scars, and scars resulting from abrasions and tattoos caused by debris remaining in tissues following primary repair. However, they may also be used to blend depressed scars with surrounding normal tissue by reducing the height of adjacent normal tissue. Skin resurfacing procedures treat lesions no deeper than the boundary of the epidermis and dermis and aim to produce a smoother scar that blends in with surrounding tissues.

The concept of facial skin resurfacing was used by the ancient Egyptians as a procedure whereby they used lactic and alpha-hydroxy acids to produce from sour milk, which was applied to the skin as a way to restore its youthful appearance[321]; the same concept was used later in the French revolution, when aged wine was used for the same purpose.[322] The most significant innovation in the modern era of facial resurfacing was developed by Max Jessner, who reported the application of a combination of 14% salicylic acid, 14% lactic acid, and 14% resorcinol in an alcohol solution, known today as Jessner's solution.[321]

In general, for larger treatment areas, the entire facial subunit, unit, or face should be treated to blend final color and texture. These procedures are best performed early in the healing process, from 6 to 9 weeks after tissue injury, because during this time there is a high degree of intrinsic fibroblastic activity. When treating nonfacial skin, the surgeon should always be cautious and conservative with all ablative techniques because the lack of pilosebaceous units outside the face and upper neck reduce the regenerative capabilities of skin throughout other areas of the body. There are a variety of resurfacing procedures currently in use (see Box 22-2), generally classified according to the depth of tissue affected—superficial, medium, or deep. Superficial resurfacing

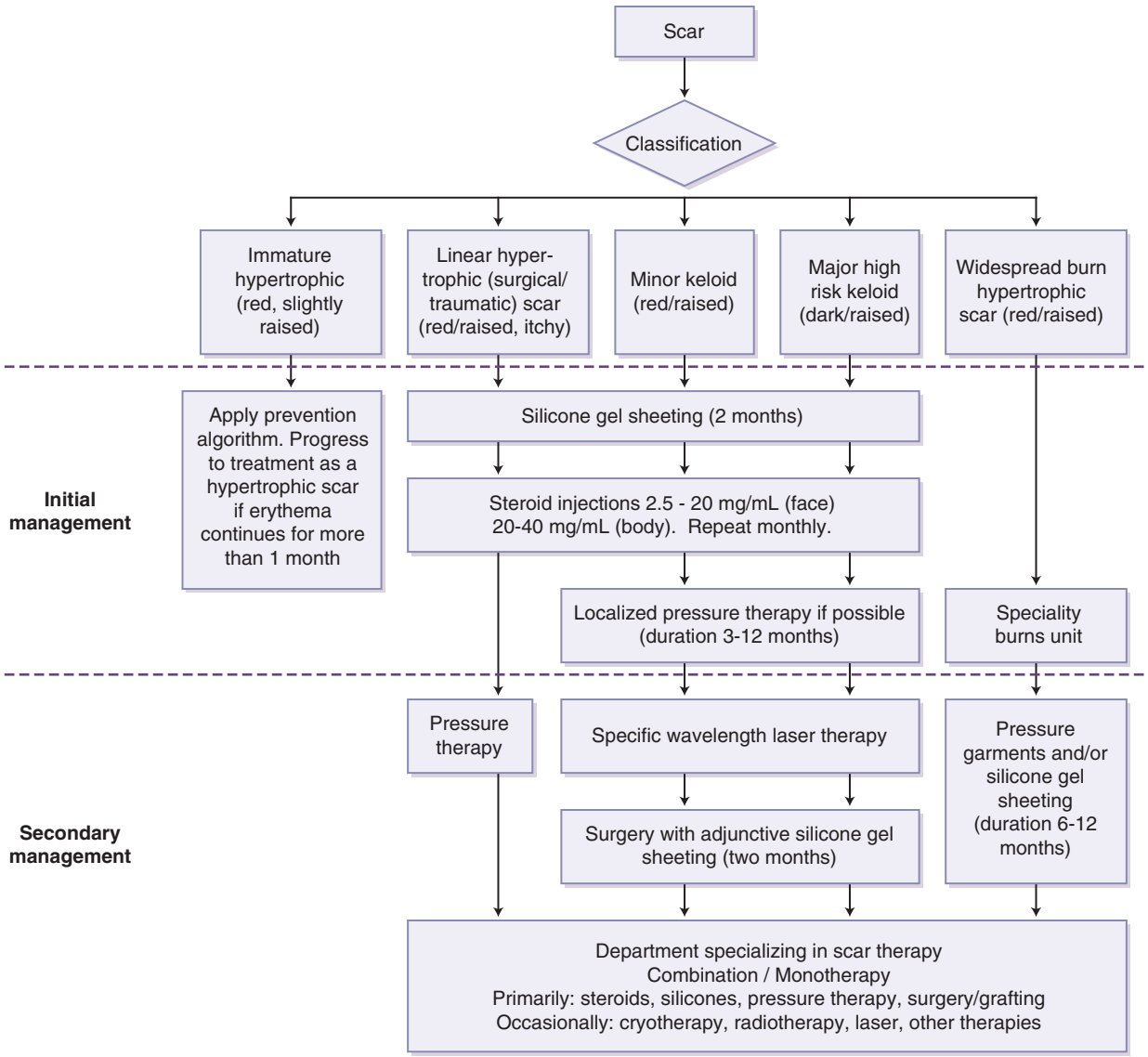


FIGURE 22-40 Algorithm for the treatment of scars. (Adapted from Mustoe TA, Cooter RD, Gold MH, et al: International clinical recommendations on scar management. Plast Reconstr Surg 110:560–571, 2002.)

affects the papillary dermis, medium-depth resurfacing affects the upper reticular dermis, and deep tissue resurfacing affects the midreticular dermis.

PREOPERATIVE CONSIDERATIONS

The patient's Fitzpatrick skin type and response to sun exposure should be considered prior to facial resurfacing.[105-107,276,323] Hypo- and hyperpigmentation changes are rarely associated with skin types I and II; therefore, all types of facial resurfacing procedures are considered safe in these patients. However, patients with type I or II skin are usually best treated with lasers. Some type III or IV patents may benefit from a medium-depth chemical peel. Before treating types IV to VI patients, the selected procedure should be performed as a test spot at the hairline to assess pigment changes prior to a definitive procedure.[324] The data regarding the appropriate treatment for type V or VI patients is conflicting because they are always at risk for hyperpigmentation and/or hypopigmentation regardless of the procedure. An erbium (Er):YAG laser may be better suited for types V and VI skin types because it is associated with less thermal damage.[325] The degree of photoaging should be considered. The Glogau classification (see earlier, Table 22-5) provides a measure of photoaging that occurs as a result of exposure to ultraviolet light (UV) radiation, specifically UVB (290 to 320 nm) and UVA (320 to 400 nm), with wavelengths that penetrate the dermis and cause photodamage.[110,326] Resorption of elastin and collagen leads to a prominent epidermis that rests on a thin damaged epidermis, resulting in wrinkle formation.

A complete medical history should be obtained and a physical examination performed on any patient considered for facial resurfacing. Patients with medical comorbidities, such as systemic lupus erythematosus, active herpetic infections, Ehlers-Danlos syndrome, scleroderma, vitiligo, discoid lupus, and ectodermal dysplasia, are at an increased risk of scarring following facial

resurfacing.[325] Patients with lupus in particular are at increased risk for developing Koebner reactions following a resurfacing procedure.[327,328] Immunocompromised patients such as those with human immunodeficiency virus (HIV) infection or those undergoing chemotherapy are at increased risk for developing postoperative infections.[329] Patients who have received chemotherapy or radiation to skin in the past should not have surgery until 6 months after therapy to ensure that dermal collagen remodeling is complete. In addition, patients with previous radiation therapy may have chronic dermatitis, which decreases the body's ability to heal.[330] Previous areas of radiation should be examined for intact hair growth. The presence of hair usually correlates with enough pilosebaceous glands for adequate healing after medium or deep chemical peels.

A complete medication history is also crucial to determine in the patient undergoing resurfacing. Women taking birth control pills or supplemental hormones are at increased risk for hyperpigmentation. Patients with a history of isotretinoin are at increased risk for postprocedure scarring following ablative procedures because the drug is thought to shut down the pilosebaceous unit (sebostatic), which is necessary for reepithelialization, and may contribute to delayed wound healing. It is also thought to cause loss of collagenase function, which will cause increased fibrosis and scarring.[331] If patients taking isotretinoin complain of dry skin or anhidrosis, treatment should be avoided for at least 6 months to 2 years.[325,332-335] The use of vitamin E should be noted and its use should be discontinued prior to resurfacing. Medications that affect the coagulation cascade such as aspirin or warfarin should also be discontinued if not otherwise medically contraindicated.

Patients undergoing perioral resurfacing treatment or those with a history of herpes infection should receive antiviral chemoprophylaxis. This is especially important in trauma patients because they are at an increased stress level and increased risk for viral activation. Patients undergoing CO_2 laser resurfacing are at increased risk for herpes infection. Patients may be prescribed valacyclovir, 500 mg twice daily, acyclovir, 400 mg three times daily, or famciclovir, 250 mg twice daily.[336] Prophylaxis should be initiated at least 24 hours before the procedure and continued for 2 weeks, or until reepithelialization is complete. Because the virus only creates lesions in the epidermis in the early stages of wound healing, herpetic lesions will manifest as superficial erosive ulcers.[337-339] It is best to avoid ablative procedures during the summer because there is an increased risk of postinflammatory hyperpigmentation caused by UV damage.[340]

PATIENT PREPARATION

Topical tretinoin has been shown to be efficacious in the treatment of photodamaged skin by increasing type I collagen formation and inhibiting collagenase,[341-346] and it potentiates the effects of superficial chemical peels and hydroquinone in melasma treatment.[347] Pretreatment with topical tretinoin in the form of 0.1% tretinoin cream for 2 weeks prior to 35% trichloroacetic acid (TCA) peels has been shown to enhance the healing time as well.[348] Patients with Fitzpatrick type III or IV skin may also be pretreated with a modified Kligman formula (0.1% tretinoin, 4% hydroquinone, and 0.1% triamcinolone) twice daily for 6 weeks in an effort to minimize the risk of pigment change.[349,350] This regimen is usually resumed in 2 to 3 weeks postoperatively. If the formula is too irritating, kojic acid can be substituted for hydroquinone.[332,333]

The patient should be advised to wear comfortable clothing on the day of the procedure. The patient should remove all makeup and then wash the face and neck with an antibacterial soap such as chlorhexidine gluconate (Hibiclens). Preoperative photographs should be taken. If lasers are being used, the appropriate eyewear should be applied. For chemical peels, moist gauze should be applied to both eyes and the skin should be scrubbed with acetone or other cleanser for 2 to 3 minutes to remove the stratum corneum and allow deep penetration. Petrolatum or other ointment is then applied with a cotton-tipped applicator to the deep grooves or rhytides (wrinkles) of the face to prevent chemical buildup in these areas. A fan blowing on the patient will decrease patient discomfort throughout this process.

SPECIFIC TECHNIQUES

Chemical Peels

Of all resurfacing procedures, chemical peels may offer the least benefit for post-traumatic scarring. Because of the irregularities of the scar, it is hard to control the depth of penetration using chemical peels. This technique, however, may be a useful adjunct for blending the margins of a scar previously treated by surgical revision, dermabrasion, or laser therapy.

Chemical peeling is the application of a chemical exfoliant to the epidermis and dermis for removal of superficial lesions. Its goal is to improve the texture of skin and remove irregularities. New epidermal growth is stimulated by removal of the stratum corneum with more superficial chemical peels. Medium chemical peels stimulate an inflammatory response, leading to new collagen formation in the deep reticular dermis; deep chemical peels stimulate ground substance formation.[351] Fitzpatrick skin types I and II patients have little risk for dyspigmentation, whereas type III or VI skin has an increased risk for hyper- or hypopigmentation following a chemical peel.[352,353] Pigment changes are usually not of concern for superficial chemical peels, but have a higher incidence in medium and deep chemical peels. Specific areas at risk include the lips and eyelids. Patients with extensive photodamage may require stronger chemical peels with greater frequency.

After skin preparation, the chemical to be used is applied with one or two cotton-tipped applicators, gauze, large swabs, or a fan-shaped sable hair brush in a rapid and uniform manner for approximately 1 minute. It can be done superior to inferior, or medial to lateral, and should be applied to the following facial units—the mandible, chin, temples, upper lip, cheeks, glabella, nose, and forehead—taking care to feather the chemical into the hair and jawline. Increasing pressure and number of applications increase the depth of penetration. Frosting of the skin often occurs and is an indication of the

evenness of the peel. Duration of chemical contact is determined by the condition being treated, patient tolerance, desired depth, and/or predetermined end point. Skin pretreated with tretinoin will frost faster, whereas photodamaged and highly sebaceous skin frost slower.

Types of Chemical Peels. Chemical peeling agents can be categorized based on their depth of penetration. They are classified as superficial, medium, or deep.

Superficial Chemical Peels. Superficial chemical peels work by exfoliating down to the level of the stratum corneum (Table 22-6). They are of little use in the posttraumatic setting and usually need to be applied more than once for desired effects. Alpha-hydroxy acids (AHAs; e.g., glycolic [2-hydroxyethanoic] acid and lactic [2-hydroxypropanoic] acid) are naturally occurring compounds that cause epidermolysis with discohesion of the keratinocytes of the stratum corneum; they are commonly used for superficial chemical peels.[354-356] Strength and depth are dependent on the amount of buffering or neutralization of AHA. Therefore, a solution of 30% glycolic acid in a buffered solution may be equivalent to 15% glycolic acid in a more acidic pH preparation. Immediate neutralization of AHA should take place after frosting, followed by a water rinse. This may be repeated weekly or monthly until desired results are obtained. Typical AHA regimens include applying glycolic acid at a concentration of 30% to 50% for 1 to 2 minutes. For a deeper superficial peel, glycolic acid at a concentration of 50% to 70% is applied for a duration of 2 to 20 minutes may be used.

TCA is a useful treatment for superficial problems such as melasma, superficial acne scarring, actinic keratosis, rhytides, and postinflammatory hyperpigmentation associated with trauma.[357-361] It works by causing coagulation necrosis of epidermal and dermal proteins. Depth of penetration varies with concentration. The desired concentration is obtained by mixing TCA crystals with 100 mL of distilled water. For example, 15 g of TCA crystals mixed with 10 mL of distilled water makes A 15% TCA solution. The solution may be stored in a dark glass bottle for up to 6 months. After standard application, neutralization with a cool water mist should take place after a frost appears. Reapplication may take place, with 35 minutes between coats. For a very superficial peel, TCA at a concentration of 10% applied for 1 to 2 minutes may be used. For a deeper superficial peel, TCA at a concentration of 10% to 30% for 1 to 2 minutes may be used. Concentrations may be increased by 5% with each application and may be used weekly to monthly for desired effect.

Jessner's solution, which is also known as the Combes formula consists of 14 g resorcinol, 14 g lactic acid, and 14 g salicylic acid in 95% ethanol to make a total of 100 mL.[321,362,363] It is useful for the treatment of dyspigmentations of the head and neck. In the immediate 2 to 3 days after application, pigmented tissue may appear hyperpigmented, with peeling and flaking up to 7 days after treatment. After standard application, Jessner's solution is neutralized by its chemical reaction with epidermal proteins. Superficial chemical peels can be performed with one to ten coats of Jessner's solution, applied as three coats per session in 5- to 15-minute intervals.

Other superficial chemical peels include beta-hydroxy acid, salicylic acid (*o*-hydroxybenzoic acid), and resorcinol (*m*-dihydroxybenzene); however, their use in posttraumatic scarring is limited.

Medium Chemical Peels. Medium-depth chemical peeling agents penetrate and destroy the epidermis, papillary dermis and, in some cases, the reticular dermis. Medium-depth peeling agents are most useful for treating fine rhytides, hyperpigmentation, actinic keratosis, dyschromias, Fitzpatrick type 2 photoaging, acne scars, and depressed scars. Medium peels are optimal for blending photoaged skin after deep chemical peels and laser resurfacing. Unlike superficial chemical peels, only one treatment session is often necessary. Recovery time is usually 7 to 10 days. In addition to the agents described earlier, pyruvic acid, an alpha-keto acid that converts to lactic acid, is useful for medium-depth chemical peels.[364] It works by epidermal lysis and penetrates the dermis in 1 to 2 minutes. Water may add comfort but does not neutralize the pyruvic acid. Edema and erythema will be present following its application, with crusting that will last approximately 10 days. TCA at a concentration of 50% is associated with complications such as scarring. Therefore, combinations of agents are commonly used for medium chemical peels, such as the following: 70% glycolic acid for 3 to 30 minutes, followed by 35% TCA[365]; Jessner's solution followed by 35% TCA[366,367]; solid CO_2 followed by 35% to 50% TCA or pyruvic acid[363]; and Jessner's solution and glycolic acid (Box 22-3).[368] Medium-depth chemical peels may be combined with CO_2 laser therapy, which reduces healing time and minimizes residual scarring.[369] Within approximately 3 months after a medium-depth chemical peel, thick bands of elastic fibers are evident in the mid to upper dermis.[370] In addition, there is an increase in glycosaminoglycans and ground substance in the dermal matrix, which hydrates and thickens the matrix-inhibiting wrinkling. Dermal

TABLE 22-6 Agents Used for Superficial Chemical Peels

Agent	Concentration	Mechanism of Action
TCA	10%-30%	Protein precipitation
Jessner's solution	Standard formula	Keratolysis
Glycolic acid	30%-70%	Epidermolysis
Salicylic acid	5%-15%	Keratolysis

BOX 22-3 Agents Used for Medium Chemical Peels

Combination of Either:
- 35% TCA and Jessner's solution
- 35% TCA and solid CO_2
- 35% TCA and 70% glycolic acid

89% phenol*
TCA, 50% concentration†

*Mainly used for deep chemical peels.
†Rarely used because of scarring.

collagen is not finished reorganizing for 60 to 90 days; therefore, repeat medium-depth chemical peels should not be done for at least 3 months after the initial session.

Deep Chemical Peels. Deep peeling agents cause destruction of epidermis, papillary dermis, and extend into the reticular dermis. The primary agents used in deep peels are TCA at a concentration higher than 50% and phenol (carbolic acid). The latter agent penetrates to the midreticular dermis. Baker et al have used a combination of 3 mL of 88% phenol, 2 mL of water, 8 drops of hexachlorophene (Septisol) liquid soap, and 3 drops of croton oil for deep chemical peels.[371,372] Histologically, phenol-treated skin shows a thicker, more organized connective tissue in the dermis, and a finer network of elastic fibers than normal skin, which contributes to less wrinkles.[373-375] Phenol is cardiotoxic, hepatotoxic, and nephrotoxic and is contraindicated in patients with cardiac arrhythmias.[376] Preoperative electrocardiography, complete blood count, and electrolyte panel should be performed before planning a phenol peel.

To reduce toxicity, perioperative IV fluids are administered to the patient undergoing a phenol peel. The phenol is applied until a frost appears. Each facial unit should be treated 10 to 15 minutes apart, for a total treatment time of 1 hour. Immediately following the procedure, a dusky erythema will develop over the initial 12 hours, with an accentuation of pigmented lesions and exudative crusting forms. This crusting should be débrided with soaks, compresses, or occlusive salves. The goal is to remove the crust and prevent it from hardening to form a scab. Reepithelialization after a phenol chemical peel begins after 3 days and continues until 14 days postoperative. Erythema may be present for 2 to 4 months. Neoangiogenesis and new collagen formation may take place for up to 4 months during the final stages of fibroplasia. Patients should wait at least 1 year before undergoing another session. Common risks of the procedure include scarring, and hypopigmentation.

Postoperative Care. Following epithelialization, patients should use a moisturizer twice daily to prevent scarring. Patients should be advised not to rupture any blisters or pick any crusts. For major crusting, topical bacitracin or bacitracin–polymyxin B (Polysporin) should be applied twice daily. Aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided for up to 1 week after surgery. Erythema may last up to 4 weeks. For patients with significant edema, 2.5% cortisone cream may assist with recovery. Tretinoin should be resumed 1 week after a chemical peel. Sun block that prevents exposure to UVB and UVA should be used to prevent pigmentation abnormalities and further photoaging for at least the first month following a chemical peel. After 1 month, the daily use of a sunblock, SPF 15 to 30, is recommended.

Complications. Complications following chemical peel usually stem from injury to the epidermis and dermis. Hyperpigmentation may last several weeks, but treatment with tretinoin may reduce the duration. Patients with Fitzpatrick type IV or V skin and those exposed to sun are at increased risk for hyperpigmentation. They are best treated with hydroquinone, which is especially useful in the treatment of dyschromias in Fitzpatrick types III and IV patients.

Infection, which may be bacterial, viral, or fungal, may result in prolonged formation of granulation tissue. Symptoms of a postpeeling infection include fever, pain, and discharge. Infections may lead to significant scarring and, when suspected, the skin should be cultured and treated without delay. Milia, or small inclusion cysts, may occur during healing and are best treated with extraction using a no. 11 blade. Preoperative and postoperative tretinoin has been shown to reduce the incidence of milia formation.[346]

Dermabrasion and Microdermabrasion

Dermabrasion and microdermabrasion work by mechanically removing the epidermis along with the papillary or upper reticular layer of the dermis and are considered as medium to deep resurfacing procedures.[377,378] They may be used as soon as 6 weeks following injury, with almost complete disappearance of some scars treated at this time, and have similar outcomes to the use of lasers for the treatment of perioral rhytides but are associated with less postoperative crusting and more rapid reepithelialization of the skin.[379,380] They work by increasing the density of types I and III collagen and TGF-β in the papillary dermis and reorienting collagen to a more parallel plane to the axis of the epidermis, which arises from the deeper, less photodamaged cells.[381-384] In addition, upregulation of tenascin expression throughout the papillary dermis and an increase in a6b4 integrin subunits of keratinocytes of the stratum spinosum are seen on histologic examination.[385] They have been shown to be equally or more effective than 5-fluorouracil (5-FU) in the treatment of actinic keratosis and other premalignant lesions.[346,386]

Indications for dermabrasion related to trauma include the treatment of post-traumatic keloids, and hypertrophic or depressed scars[378] Dermabrasion is contraindicated in patients with a history of abnormal wound healing, hypertrophic scarring, recent isotretinoin treatment, deep thermal injury, active herpes simplex or human papilloma virus infection, and congenital ectodermal defects.[387] As with chemical peels, because of sebaceous gland atrophy, patients with a history of isotretinoin use may be at increased risk for scarring.[331,388] However, there have been reports of patients undergoing dermabrasion following isotretinoin use without any adverse effects,[345,389] and laboratory studies have failed to show any side effects of isotretinoin with regard to collagen synthesis or fibroblast activity of the skin. Patients should be informed about the risks and benefits of dermabrasion therapy and combination with other scar revision techniques should be considered.[390]

The technique of mechanical dermabrasion includes using a diamond fraise or wire brush burr, moved in a direction perpendicular to the rotating burr with a courser grit penetrating deeper than finer grit. It can be performed using local anesthesia, nerve blocks, and/or cryogenic freezing. Freezing the skin to −40° C (−40° F) in 25 seconds produces a firm, anesthetized, bloodless surface, without distorting skin contours. The disadvantages of freezing, however, is that it is technique-sensitive, with thermal injury resulting from temperatures lower than 30° C inducing a frostbite type wound, leading to hypopigmentation. If too much pressure is placed on the

skin with the operating handpiece sloughing of the skin may ensue. As an alternative to freezing, local anesthesia tumescence may be used as an aid to increase skin turgor and rigidity to abrade.[391] For larger areas, to reduce the risk of lidocaine toxicity, lidocaine diluted in saline, epinephrine, and bicarbonate may be used in a larger volume. For depressed scars, it may be helpful to paint the area with 1% gentian violet so that dermabrasion can be carried to the greatest depth of the scar. Gentian violet combined with daily petrolatum dressings is also useful after dermabrasion for tattoo removal, causing the pigment to leach out of the wound. This creates an inflammatory response–stimulated phagocytosis of the remaining pigment.

The treatment should be planned in the order of cosmetic units performed, moving from unit to unit with taut skin tension placed on the skin by the assistant, and using a 4 × 4 gauze soaked in 2% lidocaine with 1 : 000,000 epinephrine, with mild pressure on treated sites for hemostasis. The surgeon may begin centrally beside the nose, working outward and moving from just below the jawline up to the infraorbital area and posterior to the preauricular area. Unabraded skin such as the eyebrows and first few centimeters of hair may be treated with 35% TCA for an even blending of the dermabrasion. The end point for dermabrasion is the development of uniform pinpoint bleeding, which will emanate from the papillary dermis. However, some may use the superficial reticular dermis as an end point because dermabrasion at this level is less likely to produce superficial scarring. This level is apparent by the transition between the appearance of white parallel bands and the protrusion of short white fibers.[392] At this level, a yellowish appearance resulting from the sebaceous glands at the border between the reticular and papillary dermis may be present.[337] This endpoint may be used for more significant scars, however carries the risk of further scarring.

Postoperative Care. Following hemostasis, a topical antibiotic, followed by a nonadherent dressing, absorbent gauze layer, and more rigid stabilizing layer outside should be applied. Patients should keep the head elevated; ice packs are recommended. Patients with a history of herpes simplex infection should begin prophylaxis 24 hours preoperatively and continue for 5 days postoperatively. Patients should be advised to soak their face twice daily and not pick at any crusts. Epithelialization following dermabrasion takes approximately 7 days. Following healing after dermabrasion, the area may be blended with surrounding tissues using superficial dermabrasion or a chemical peel. As with chemical peels, the application of 0.5% tretinoin for several weeks before and after dermabrasion has been shown to accelerate wound healing in the postoperative period.[346] In addition, the use of occlusive dressings in dermabraded wounds has been shown to increase healing time by up to 40% and may have a positive effect on collagen synthesis when compared with wounds left open to air.[393] Pain is also reduced with the use of occlusive dressings.[394] In patients with a history of dyschromia, topical hydroquinone and tretinoin may be started, with 1% hydrocortisone being added for patients with persistent hyperpigmentation. The skin appears normal skin in approximately 1 month.

Complications. Postoperative edema will usually resolve in 4 to 6 weeks. Complications following dermabrasion include milia, rebound oil production, and acne formation, but these are usually transient. Infection, hyperpigmentation, persistent erythema, and scarring are also unforeseen potential risks of dermabrasion. Scarring may be operator- or patient-dependent. Histologic changes following dermabrasion are similar to that of a deep chemical peel or CO_2 laser treatment.[395,396] Clinically, lasers penetrate the same depth as a phenol deep chemical peel and dermabrasion.[397] However, when compared with chemical peels one study has found that dermabraded skin may be less stiff at 6-month follow-up.[398] In addition, patients undergoing dermabrasion may heal significantly faster, with less erythema, than when treated with CO_2 laser.

Microdermabrasion. Microdermabrasion involves the frictional resurfacing of the epidermis and dermis by circulating a stream of aluminum oxide crystals that are pulled and blown across the skin. Other types of granules may be used, but aluminum oxide is the most common. This is useful for post-traumatic scars; patients may see beneficial results in as little as four treatments.[399] Skin biopsies taken in patients who have undergone multiple aluminum oxide microdermabrasion demonstrate a thicker epidermis and dermis, more hyalinization of the papillary dermis, and more newly deposited collagen and elastic fibers when compared with controls.[381,382] Clinically, patients show improvement in dyschromias, actinic changes, and fine rhytides when compared with controls.

Before the procedure, the skin should be cleansed in a similar manner as for chemical peeling. No anesthesia is required. Each facial unit should be treated one at a time. Patients may cleanse the skin and resume wearing makeup and normal activities immediately after the procedure. As with dermabrasion, the skin should be stabilized with firm pressure. A potential concern of microdermabrasion is a resulting granulomatous response from deep dermal penetration of the aluminum oxide crystals; however, this reaction has not been seen in the clinical setting.[399]

Lasers

Light amplification by stimulated emission of radiation (LASER) works by emitting electromagnetic radiation in the form of a stream of photons that travels at the speed of light. Lasers used clinically emit radiation in a fixed spectrum of wavelengths (Table 22-7). Materials used include dye, crystal, gas, or other media. They require an energy source in the form of radiation, electric current, or flashlamp, which emits the electrons at an excited, higher energy level. After returning to their steady state orbits, electrons release quantums of energy in the form of photons traveling at a certain wavelength; this stimulates other electrons to release photons that travel along an optical axis, reflecting back and forth, and causing a cascading effect of energy and photon release. The resulting light is amplified and a laser beam is produced.

Lasers allow the surgeon to select a wavelength for a particular targeted tissue, or chromophore. The two most commonly used lasers in facial resurfacing are the

TABLE 22-7 Lasers and Their Clinical Applications

Laser	Wavelength (nm)	Indications
CO_2	10,600	Resurfacing, atrophic scars
Er:YAG	2,940	Resurfacing, atrophic scars
Long pulsed ruby	694	Hair removal
Long pulsed alexandrite	755	Hair removal
Long pulsed diode	800	Hair removal
Long pulsed Nd:YAG	1064	Hair removal
Q-s Nd:YAG	1064	Pigmented skin, lesion, or tattoo
Q-s ruby	694	Pigmented skin, lesion, or tattoo
Q-s alexandrite	755	Pigmented skin, lesion, or tattoo
Pulsed dye	510	Pigmented skin, lesion, or tattoo
Q-s Nd:YAG (frequency- doubled) KTP	532	Pigmented skin, lesion, or tattoo
Argon-pumped tunable dye	577, 585	Vascular lesions
Flashlamp-pumped dye	585	Vascular lesions, hypertrophic scars
Pulsed Nd:YAG (frequency-doubled) KTP	532	Vascular lesions

CO_2 and Er:YAG lasers, which are absorbed by water. Water constitutes 90% of the epidermis, so CO_2 and Er:YAG are optimal for skin resurfacing procedures. Unfortunately, although the targeted chromophore may absorb a particular wavelength, so may adjacent chromophores. All laser energy is converted to heat in a process called photothermolysis, causing damage to adjacent tissue by heat dissipation, or thermal bleed. Type 1 collagen denatures at 70° C (158° F); therefore, the underlying dermis is susceptible to collateral thermal damage, which may lead to scarring.

Histologically, skin treated with lasers looks similar to skin treated with phenol. It demonstrates a zone of tissue vaporization and ablation of the epithelium, with an adjacent area of a collateral basophilic zone of thermal necrosis of the dermis. An initial pass of the laser will be absorbed by the water in the epidermis but because of the low water content of the collagen of the dermis and subsequent less absorption, more heat is produced, causing thermal injury with each successive pass.[400,401] Following laser resurfacing, regeneration of epithelium and elastic fiber in the papillary dermis occurs and glycoprotein tenascin is expressed as new collagen. Facial units such as the cheeks, chin, forehead, and perioral areas can tolerate deeper resurfacing, whereas other areas such as the jawline and other sharp demarcations should undergo one pass or lighter resurfacing.

CO_2 lasers were the first lasers available for skin rejuvenation, first being reported by Patel in 1964.[402] This type of laser is most useful for the correction of severe photodamaged skin, sebaceous hyperplasia, fine or moderate rhytides, and dyschromias. The mechanism of action is by causing thermal injury and subsequent vaporization of the dermis and epidermis, thereby leading to collagen coagulation and shrinking. New collagen production continues for up to 2 to 3 months postoperatively. The continuous pulse CO_2 laser works at a wavelength of 10,600 nm, vaporizing approximately 50 µm of tissue.[323,403,404] However, it can also can result in up to 100 µm of thermal injury, which causes erythema, pigment changes, and scarring. The superpulsed CO_2 laser use higher peak powers and shorter pulse duration to minimize collateral thermal injury while maximizing tissue ablation. The ultrapulse CO_2 laser, reported by Fitzpatrick in 1993, has an even shorter pulse duration (1000 microseconds) than the superpulsed CO_2 laser, leaving behind as little as 50 µm of adjacent thermal damage. Reepithelialization following CO_2 laser resurfacing takes place in approximately 7 to 14 days. Erythema, which is typical after CO_2 laser treatment, tends to fade over 1 to 6 months.[338]

The Er:YAG has a wavelength of approximately 2940 nm and has a higher affinity for water than CO_2 lasers, so most of its energy is focused on water vaporization, minimizing thermal damage and tissue debris.[338] It produces a more superficial ablation with less postoperative erythema and a shorter recovery phase than other lasers. It is useful for treating moderate to severe rhytidosis associated, with moderate to severe photodamage, and has been shown to stimulate collagen formation.[404] It has an ablation depth of 10 to 40 µm and leaves a zone of thermal spread less than 50 µm, which is less than that of the CO_2 laser. This leaves most patients with superior cosmetic results with fewer side effects, such as scarring and erythema. Complete reepithelialization takes place in 4 to 10 days; postoperative erythema usually resolves in 4 weeks. The use of the Er:YAG laser is most beneficial for Fitzpatrick types IV to VI skin and may be used as an adjunct in vaporizing the area of thermal necrosis following ultrapulse CO_2 resurfacing, resulting in faster healing and less erythema in these areas.[405] The CO_2 and Er:YAG lasers have also been combined, optimizing the advantages of both lasers.[406-408] These include the precision and low thermal ablation of the Er:YAG laser and deep ablation of the CO_2 laser.

The 585-nm flashlamp-pumped PDL is indicated for hypertrophic scarring and keloids. It is especially useful in post-traumatic injuries, resulting in less recurrent scar formation while significantly improving scar contour, height, pruritus, texture, color, and pliability.[57,267,269-273,409] In addition, PDL reduces scar microcirculation, subsequently preventing excess scar formation. Its mechanism of action is by acting on hemoglobin-containing structures, rather than directly ablating scar tissue.

Lasers should be used in accordance with the safety standards of the American National Standards Institute

(ANSI).[410] These include the use of protective eyewear for the patient, surgeon, and staff, nonflammable drapes, smoke evacuator, and laser masks. It is safe to use an aqueous-based solution for skin preparation; alcohol-based preparations should be avoided. Laser resurfacing may be performed using general, regional, local, or topical anesthesia, or sedation. Preoperatively, pretreatment with hydroquinone, tretinoin, or glycolic acid has been shown to reduce postradiation side effects, such as hyperpigmentation.[411] It is important to know the end points with laser resurfacing. For CO_2 laser resurfacing, a pink color may be seen once the papillary dermis is entered. The use of saline gauze to remove vaporized skin between passes is helpful for visualizing the full depth of treatment. Because of the hourglass shape of the pilosebaceous gland, pores increase in diameter as the level of ablation deepens. Variations in the thickness of skin among individuals and the facial subunit of each individual require the surgeon to pay attention constantly to the laser settings with each pass and movement to a different subunit.

Postoperative Care. Immediately following laser resurfacing, cool gauze soaked with topical lidocaine should be applied. In the postoperative period, a semiocclusive dressing should be used for 3 days and has been shown to reduce pain and decrease the duration of reepithelialization to as little as 5 days.[412-416] Antiviral prophylaxis should be continued for 7 to 10 days and any preoperative antibiotics, if used, may be discontinued. Antifungal prophylaxis with fluconazole has not been used routinely for ablative procedures; however, some believe that it should be recommended to patients with a history of oral, vaginal, or nail candidiasis. After reepithelialization is complete, sun should be avoided for 2 to 3 months. In patients with severe postoperative edema, corticosteroids have shown to be of benefit and skin moisturizers should be used after reepithelialization.

Complications. Excess passes of the laser will lead to an unintended depth of tissue penetration, which leads to significant scarring; this typically occurs in the area of excessive thermal injury or infection. Areas that remain persistently red for longer than 3 weeks should raise concern for infection.[417] These areas usually appear dusky red or red-purple, rather than the bright red initially seen following resurfacing. For progressive scarring, 585-nm flashlamp-pumped PDL therapy can be performed every 4 weeks.[418]

A variety of laser systems use a computerized scanner generator, allowing large surface areas to be scanned in a systematic manner and minimizing overuse of the laser in one area.[419] It is not uncommon to see a 2- to 6-week period of hyperpigmentation following laser resurfacing, especially in Fitzpatrick skin types IV to VI. Topical steroids used two or three times daily, tretinoin, or hydroquinone may be beneficial for treating pigmented skin. Hypopigmentation that results after laser resurfacing is often unpredictable and permanent. As depth of injury increases, more melanocytes in hair-bearing areas are destroyed.[417] This occurs in 10 % to 30% of patients and is usually difficult to treat.[420]

Other side effects include acneiform eruption, herpes simplex outbreak, bacterial infection, yeast infection, prolonged erythema beyond 1 month, and contact dermatitis.[421-423] Infections should be suspected when there is increased pruritus, burning, or pain in the first 3 postoperative days.[338,418] Gram staining, Tzanck smear, or immunofluorescence and potassium hydroxide examination should be done by direct smear of the areas in question, when indicated. Any occlusive dressing should be removed and empirical antibiotic treatment begun, including gram-negative coverage and antiviral, and antifungal therapy; this should not be delayed because of the significant long-term effects of infection. Prolonged erythema with skin thickening should be treated with intralesional injections with triamcinolone, 5-fluorouracil,[276] or a combination of both. Dermabrasion and CO_2 laser resurfacing should not be used in the lower two thirds of the face because this area contains fewer adnexal structures and has a higher chance of scarring. Extreme caution should be used when considering resurfacing around the upper neck and jawline.

GENERAL POSTOPERATIVE CARE FOR ALL ABLATIVE TECHNIQUES

Postoperative care for specific procedures is discussed earlier in this chapter. In general, occlusive dressings are recommended for almost all wounds. As noted, reepithelialization is enhanced in a moist environment.[5] Wounds with excess crusting and scarring heal more slowly than wound meticulously kept clean because epithelium travels along the most hydrated route[28] (Fig. 22-40). Granulation tissue formation is also hastened in a moist environment.[29] Therefore, occlusive dressings and antibiotic ointment use are recommended so that epithelial migration proceeds in directly and efficiently. Antibiotic ointment should be applied following surgical scar revision procedures,[34] reducing pain, infection, and scarring.[424,425] Antibiotic ointment, however, may be associated with a higher incidence of contact dermatitis. A cold compress on postoperative day 1 may be used. Once initial healing is complete, patients should be instructed to use a soapless cleanser to wash the face for the next 2 months. Patients may resume wearing makeup once reepithelialization has occurred. Sunblock (SPF > 30) is recommended for at least 1 year and encouraged indefinitely following resurfacing to minimize the risk of hyperpigmentation and skin cancer. Hydroquinone is a useful adjunct to prevent and/or treat hyperpigmentation. This can usually be started as soon as 2 to 3 weeks postresurfacing.

ADJUNCTIVE SCAR REVISION PROCEDURES

TISSUE EXPANSION

Tissue expansion is indicated for scars that cannot be revised by immediate local or regional flap techniques without causing significant donor site morbidity and is especially useful for post-traumatic avulsive defects sustaining significant tissue loss. It is most often used for the scalp and forehead because of the inelasticity of tissue in these areas. In general, the goal of tissue expansion is to

increase the size of normal tissue enough to cover a local scarred defect.

Unlike serial scar excision, which relies on mechanical creep and tissue elongation, tissue expansion works by biologic creep and tissue generation. It was popularized by Radovan for mastectomy reconstruction.[426] Expansion of the skin results in the creation of a new epidermal layer, with thinning of the dermis and subcutaneous tissues. Hair follicle morphology will remain the same, which is a critical advantage of scalp expansion.[427] Alternatives to tissue expansion include full- and partial-thickness skin grafting, serial scar excision, and the use of local regional and distal flaps; however, tissue expansion can be used alone or in combination with these techniques. Tissue expansion has the benefits of maintaining identical color, texture, thickness, and hair-bearing aspect of skin, and improves the vascularization of the native tissue bed. This proliferation of blood vessels increases the survivability of flaps raised in expanded tissue by 117% over flaps raised in nonexpanded tissue.[428] Disadvantages of tissue expansion include the need for multiple operations, longer duration of treatment, making the patient live longer with the deformity, and loss of skin elasticity, along with implant capsule formation. Also, wounds treated by tissue expansion may experience delayed contracture of expanded tissue; this may be associated with sensation changes of the overlying skin.[429-433]

Tissue expanders should not be placed in areas of scar, atrophied skin, or irradiated skin because of the increased risk of skin breakdown and necrosis in these areas.[434] Complications include wound dehiscence, port failure, implant exposure, seroma formation, implant rupture, and infection.[429,431] These might be avoided by using multiple drains, avoiding custom implant use because they tend to deflate, and using more than one expander if space allows. In addition, expanded skin of the neck should not be advanced past the mandibular border. This could lead to scar widening, lower eyelid ectropion, or lip ectropion. Skin expanded in the neck and cheek region should not cross multiple facial units because differences in skin characteristics may result in unsightly outcomes and be associated with more complications. Normal unexpanded cheek tissue is ultimately the best donor for scar revision of the cheek and skin overlying the mandibular body and inferior border. Inferior advancement of expanded cheek skin is associated with a much lower risk of ectropion than superior advancement than tissue of the upper facial third. Advancement or rotational flaps used in the neck should be performed with the neck in extension because this decreases skin tension.

Ideally, if tissue expansion is needed, it is combined with other revision techniques, such as free tissue transfer,[435] skin resurfacing,[436] serial scar excision,[437] Z-plasty,[438] and W-plasty.[439] Variations include endoscopic tissue expander placement, which allows for placement of expanders away from the site of interest, using small incisions to reduce the risk of wound dehiscence.[440]

Technique. There are a variety of customized and standardized implants of various sizes and shapes that can be used in the head and neck region. However, a detailed discussion of tissue expander placement is beyond the scope of this chapter.

In general, incisions should be made in normal healthy skin, just adjacent to the scarred area and preferably radial to the implant to minimize wound dehiscence. Passive placement of the implant into the cavity is essential. It should be placed within the subcutaneous plane, in the subgaleal plane in the scalp and just above the platysma in the neck. Only normal tissue should be expanded and the scar should remain the same size, with the ultimate goal of scar excision. In general, inflation of the implant begins 1 to 2 weeks after placement and increased in size at 1- to 2-week intervals. Expansion should not take place in the presence of excess pain or blanching on follow-up.[429] When more rapid expansion sequences are used, it is advisable to use local pulse oximetry to monitor for tissue hypoxia.[433] As a general rule, tissue should be expanded at least 2.5 to 3 times the desired size to allow for recoil and tension-free final closure after scar excision.[441,442] Although lower in elasticity than neighboring skin, expanded skin may be rotated, transposed, or advanced. It is not recommended to excise the capsule that develops during expansion because of its rich vascularity. It may, however, be scored, advance, and rotated. The patient should be informed that expanded skin will usually soften and skin characteristics such as color and texture will improve usually 3 months after final expansion.[443]

STEROIDS

Intralesional steroid injections are useful for the treatment of keloids and hypertrophic scars (see earlier).[276] They are also useful in reducing scar volume and height, pruritus, edema, and erythema and increasing scar pliability,[57,274,275] and are a useful adjunct to surgical scar revision. They may be started as soon as 2 weeks postoperatively.[277] The most commonly used agent is insoluble triamcinolone acetonide (10 to 40 mg/mL).[268] There are few short-term side effects; long-term side effects include tissue atrophy leading to skin thinning and widening.[57,278,279] Injections may be repeated every 3 to 4 weeks until the desired effect is achieved, but the dosage must be adjusted if side effects develop.[444]

FILLER MATERIALS

Filler materials are used to replace tissues below the epidermis, such as dermal collagen or subcutaneous tissue.[445-447] They are used to treat a variety of defects, most notably the depressed scar. Xenograft fillers such as injectable bovine collagen (Zyderm, Zyplast), which is derived from cowhide and is the purified suspension of bovine dermal collagen, may be used. The antigenic potential of the material is reduced by 95% to 98% in a series of purification steps.[448] When these materials are used, , the double skin test may be performed 4 weeks apart to test patient sensitivity. This is done by injecting 0.1 mL of the material in the volar forearm; if erythema or induration develops, the material should not be used. The skin test is positive in approximately 3% of patients.[449] Zyplast is collagen cross-linked with glutaraldehyde which is longer lasting and less reactive than Zyderm, which is placed within the dermis.[444] Long-term complications of bovine

dermal fillers include local skin necrosis, granulomatous reactions,[446,450,451] and abscess formation.[452-454] Other popular nonhuman filler materials include gelatin matrix implants[455] and hyaluronic acid fillers, such as hylan B[456-458] and Restylane (Medicis Aesthetics, Scottsdale, Ariz).[459-461]

Allograft fillers include AlloDerm and/or Cymetra (LifeCell). AlloDerm is available as sheets and Cymetra as the injectable form. For post-traumatic lip scarring secondary to deficient tissue volume, AlloDerm may be used by creating a submucosal tunnel along the anteroinferior aspect of the lip, pulling the appropriate-sized piece of AlloDerm through. AlloDerm may also be injected in areas of minor defects. Although silicone is no longer approved for use in the face, other allogenic fillers that may be used include homologous injectable collagen.[462] However, more studies are needed to evaluate the usefulness of this material.

First described for ophthalmologic procedures,[463] autologous dermal grafts are useful after subcision and undermining of scars for preventing reattachment of the anchoring fibrous bands from the overlying skin. Dermal grafts should be used 2 to 6 weeks after subcision.[464] Optimal donor sites include the postauricular dermal tissue because of its inconspicuous nature. Side effects include hematoma formation, infection, edema, pain, and graft migration. Grafts will mature to final size in 1 to 6 months.[465] Other but less used autologous fillers include cultured human fibroblasts[466-469] and autologous injectable collagen[470-472]; however, few clinical studies have been carried out on the use of these materials.

Autografts such as autologous fat graft or injection may be used for severely depressed scars. These are especially useful for post-traumatic defects and scarring. However, their effects are unpredictable and patients may experience a 45% weight loss within 1 year.[473] Fat grafts harvested surgically maintain volume better than those suctioned[474,475] and, because of expected absorption, overcorrection of defects by 30% to 50% is recommended.[476,477] Microlipoinjection is performed by first using a tumescent local anesthesia technique and then harvesting with a blunt-tipped microcannula or syringe. After serosanguineous material is separated, the fat is then washed with sterile saline. After injection, the fat is massaged to a smooth fill. This technique is useful for the treatment of defects around glabellar furrows, lips, melolabial and nasolabial folds, hemifacial atrophy, or post-traumatic head and neck scarring. As a method to restore natural skin layers, subcutaneous fat may be used to restore lost subcutaneous tissue and dermal fillers may be used to restore the deficient dermis. The epidermis may then be restored as described earlier. Lipocytic dermal augmentation is a modification of fat injection; the remaining intracellular fibrous setae following adipocyte rupture and triglyceride removal is used as collagen filler.[478,479] This seems to be beneficial for atrophy around the mouth and subcutaneous scars. The process involves freezing harvested fat in liquid nitrogen and then rapid thawing in warm water. The supernate is then centrifuged to remove the remaining triglycerides. This material has been shown to be equal to Zyplast in longevity for the treatment of atrophic skin and, like microlipoinjection, overcorrection should be done because of expected absorption.[480]

Other permanent materials such as expanded polytetrafluorethylene (e-PTFE; Gore-Tex, WL Gore, Flagstaff, Ariz),[481-483] may be used as permanent fillers in facial augmentation and are extremely useful in the lips because they will not resorb, like other implants.[484] However, these materials are associated with side effects such as extrusion into surrounding tissue layers, especially in the upper lip because of complex motions, with the resulting implant creating a capsule of fibrosis around it.[485] Other synthetic permanent materials used as fillers in the head and neck region include liquid silicone,[486] polymethylmethacrylate (PMMA),[487] and a biphasic polymer (Bioplastique).[488] Although silicone has shown to be inert, PMMA and Bioplastique have been associated with side effects such as granuloma formation.[489-491]

BOTOX

Botulinum toxin is used for simple facial wrinkles, blepharospasm, and post-traumatic muscle contraction. It weakens or paralyzes muscle by binding to presynaptic cholinergic terminals and blocking the release of acetylcholine at the neuromuscular junction.[492] It comes in two different preparations, onabotulinumtoxinA (BTX-A) and rimabotulinumtoxin B (BTX-B). BTX-B is useful for the treatment of cervical dystonias[493,494] and facial spasm.[495,496]. BTX-A injections are useful for a variety of procedures, including the treatment of wrinkles,[497] masticator muscle hypertrophy,[498] temporomandibular disorders,[499] salivary secretion disorders,[500] facial nerve palsy,[501] and post-traumatic scars secondary to significant muscle contraction.[502,503]

Botulism toxin is useful in combination with resurfacing procedures and dermal fillers. The resulting decrease in muscle pull allows for new collagen formation in areas of treatment. In addition, BTX-A injection lengthens the duration of effect of injectable dermal fillers. Its effects are noticeable 2 to 3 days after injection, with maximal weakness occurring 1 to 2 weeks after injection. Although BTX-A permanently impairs neuromuscular junctions, repair of muscle, axonal sprouting, and production of new neuromuscular junctions limit its effects to 3 to 6 months. It is available in a vial form and must be reconstituted with sterile, nonpreserved saline prior to IM injection. Each vial contains 100 units of *Clostridium botulinum* toxin type A, 0.5 mg of human albumin, and 0.9 mg of sodium chloride. The 100 units of Botox (Allergan Irvine, Calif) are significantly below the median lethal dose (LD_{50}) in an average 70-kg (150-lb) human.

Complications of Botox injection are usually reversible. These include ptosis after glabellar injection; as a result of migration of the toxin through the orbital septum, affecting the levator muscle,[504] eyebrow dropping following forehead injection may occur. Other complications include bruising, asymmetry, dysphagia, neck weakness, perioral droop, and diplopia from lateral rectus involvement. Permanent side effects include globe perforation, lagophthalmos, and keratosis. Treatment of lid ptosis includes eye drops consisting of 0.5% apraclonidine (Iopidine) to stimulate Müller's muscle.[505]

SUMMARY

Treatment of the trauma patient begins at the initial encounter and often continues for the life of the patient. Limitation in function and associated cosmetic deformities may leave the patient with significant debilitation. Skill, knowledge and, most importantly, dedication on behalf of the surgeon and patient are necessary to achieve the most optimal outcome.

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CHAPTER

23 Management of Human and Animal Bites

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OUTLINE

Incidence of Bites
Human Bites
Animal Bites
Pathophysiology
Human Bites
Animal Bites
Treatment of Bite Injuries
Initial Presentation
Antibiotic Prophylaxis
Surgical Management
Postsurgical Management
Forensic Bite Mark Recognition, Evidence, and Analysis
Bite Mark Recognition
Bite Mark Evidence
Bite Mark Analysis

Human and animal bites are relatively common occurrences and clinicians should have an understanding of the incidence, pathophysiology, virology, bacteriology, surgical and medical therapy, and proper sequence of treatment to provide comprehensive care for patients with these potentially complicated injuries. An understanding of possible injury demographics and microbiology of common bite injuries assists the practitioner in developing a clinical pathway to aid in the decision making process regarding medical and surgical treatment. Forensic bite mark examination continues to play an important role in criminal investigation, along with an understanding of DNA-based evidence gathering and criminal identification.

INCIDENCE OF BITES

HUMAN BITES

Human bites are the third most common bite injuries following dog and cat bites; it is likely that 50% of the U.S. population will have been bitten by a human or animal during their lifetime.[1-3] It has been estimated that as many as 150,000 human bites of humans occur annually but go unreported.[4] This estimate includes bites to the hand that occur when an assailant punches a victim in the teeth. The population of human bite victims is usually between 20 and 30 years of age.[5] Males are affected more frequently than are females.[6-8]

Human bite injuries can be found in almost any anatomic location, including the face and head, upper and lower extremities, breasts, lips, cheeks, nose, ears, abdomen and thorax, and genitalia; more than 50% occur to the hands and are clenched fist injuries.[4,6,9-12] Human bites are generally inflicted as a result of interpersonal violence and are typically associated with murder or attempted murder, rape, sexual assault, child abuse, burglary, and kidnapping.[10] The incidence of human bite infections ranges from 4% of facial injuries to 50% of bites to the hand.[5,13-16] Human bites more commonly become infected than dog or cat bites.[2,17]

ANIMAL BITES

Approximately 4.5 million persons are bitten by animals each year; 15% of these will seek treatment from healthcare providers.[1,6,9,18] Many of these wounds go unreported because some injuries may appear innocuous or because of social concerns. The overwhelming offender is the domesticated dog (85% to 90%), followed distantly by the domesticated cat (6% to 8%). In 2001, an estimated 368,245 patients were treated for dog bite–related injuries in hospital emergency departments.[19] Wild animals, including skunks, raccoons, bats, foxes, and others, account for less than 6%. Other domesticated animals, including horses, cattle, and sheep, account for less than 3% of these bites. The rate of injury is higher among boys than girls; most victims are between 5 and 14 years of age, but many are younger than 5 years (Table 23-1).[20-23]

Injury severity scales of children who are admitted to the hospital with dog bite injuries range from 1 to 25, with an average of 4 ± 5; the head and neck area is involved in almost 70% of these injuries.* This is in contrast to adult victims, who suffer only 5% to 15% of animal bites to the head and neck[19,26,27] (Fig. 23-1).

As many as 50% of dog bites may be reported as "unprovoked" (a possible indication of rabies infection), and more than 80% of these injuries involved the family's or a neighbor's pet.[18] Breeds most often involved included pit bulls (25%), golden retrievers (12%), Labrador retrievers (12%), Chow Chows (10%), German shepherds (10%), Rottweilers (10%), and Doberman pinschers (5%) (Table 23-2).

*References 6, 9, 18-20, 24, and 25.

TABLE 23-1 Number, Percentage, and Rate* of Nonfatal Dog Bite–Related Injuries 2001

Characteristic	No.	(%)†	Rate	(95% CI‡)
AGE GROUP (YR)				
0-4	48,153	(13.3)	253.8	(218.9-288.7)
5-9	56,146	(15.2)	278.2	(234.8-321.6)
10-14	49,326	(13.4)	236.2	(203.1-269.4)
5-19	27,820	(7.6)	137.3	(108.6-166.0)
20-24	26,161	(7.1)	133.0	(105.9-160.2)
25-34	45,133	(12.3)	114.0	(99.2-128.7)
35-44	46,658	(12.7)	103.6	(89.1-118.2)
45-54	32,613	(8.9)	83.2	(72.4-94.0)
55-64	16,185	(4.4)	64.0	(49.5-78.4)
≥65	19,005	(5.2)	53.9	(45.5-62.2)
Unknown	25§	—	—	
GENDER				
Male	202,735	(55.1)	145.0	(126.5-163.5)
Female	165,510	(44.9)	114.2	(103.8-124.5)
TREATMENT MONTH				
January	21,994	(6.0)	7.7	(5.7-9.7)
February	24,945	(6.8)	8.8	(6.4-11.1)
March	27,511	(7.5)	9.7	(7.3-12.1)
April	36,108	(9.8)	12.7	(10.1-15.3)
May	34,284	(9.3)	12.0	(9.8-14.2)
June	34,742	(9.4)	12.2	(11.3-13.1)
July	40,828	(11.1)	14.3	(11.3-17.4)
August	34,716	(9.4)	12.2	(10.9-13.5)
September	32,983	(9.0)	11.6	(9.6-13.6)
October	27,372	(7.4)	9.6	(7.3-11.9)
November	25,011	(6.8)	8.8	(7.2-10.4)
December	27,749	(7.5)	9.7	(7.9-11.6)
WORK-RELATED				
Yes	16,526	(4.5)	5.8	(4.1-7.5)
No	350,554	(95.2)	123.1	(109.9-136.3)
Unknown	1,165	(0.3)§	—	—
BODY PART INJURED				
Head, neck	83,946	(22.8)	29.5	(26.0-32.9)
Face	55,867	(15.2)	19.6	(17.0-22.2)
Mouth	17,029	(4.6)	6.0	(5.2-6.8)
Ear	5,475	(1.5)	1.9	(1.4-2.5)
Head	3,669	(1.0)	1.3	(0.8-1.8)
Other (neck, eyeball)	1,906	(0.5)	0.7	(0.3-1.0)
Upper trunk (includes shoulder)	5,036	(1.4)	1.8	(1.2-2.3)
Lower trunk	14,432	(3.9)	5.1	(3.8-6.4)
Arm/Hand	166,756	(45.3)	58.6	(54.2-62.9)
Hand	66,969	(18.2)	23.5	(20.6-26.4)
Lower arm	45,482	(12.4)	16.0	(14.7-17.3)
Finger	34,787	(9.4)	12.2	(10.3-14.1)
Upper arm	8,645	(2.3)	3.0	(2.3-3.7)
Wrist	8,029	(2.2)	2.8	(2.2-3.4)
Elbow	2,843	(0.8)	1.0	(0.7-1.3)
Leg/Foot	94,848	(25.8)	33.3	(26.2-40.4)
Lower leg	54,388	(14.8)	19.1	(14.3-23.9)
Upper leg	25,379	(6.9)	8.9	(7.3-10.5)
Knee	5,317	(1.4)	1.9	(1.3-2.5)
Foot/Toe	5,063	(1.4)	1.8	(0.9-2.6)
Ankle	4,700	(1.3)	1.7	(1.1-2.2)
Other	2,328	(0.6)§	—	—
Unknown	899	(0.2)§	—	—
DIAGNOSIS				
Contusion, abrasion, Hematoma	22,016	(6.0)	7.7	(5.7-9.7)
Laceration	90,926	(24.7)	31.9	(27.3-36.5)
Puncture	148,180	(40.2)	52.0	(34.1-70.0)
Fracture/Dislocation	1,386	(0.4)	0.5	(0.2-0.8)
Amputation/Avulsion/Crush	2,854	(0.8)	1.0	(0.7-1.4)
Cellulitis/Infection	5,559	(1.5)	2.0	(1.0-2.9)
Unspecified dog bite/Other	97,324	(26.4)	34.2	(20.4-48.0)
DISPOSITION				
Treated and released	361,692	(98.2)	127.0	(113.3-140.7)
Hospitalized, observed, transferred	5,921 (1.6)	2.1	(1.6-2.6)	
Unknown	631	(0.2)§	—	—
Total	**366,245**	**(100.0)**	**129.3**	**(115.9-142.7)**

*Per 100,000 population. These bites were treated in U.S. hospital emergency departments, by selected characteristics.
†Numbers might not sum to total because of rounding.
‡Confidence Interval.
§Estimate might be unstable because the coefficient of variation is >30%.
From Centers for Disease Control and Prevention (CDC): Nonfatal dog bite-related injuries treated in hospital emergency departments—United States, 2001. *MMWR Morb Mortal Wkly Rep* 52:605–610, 2003.

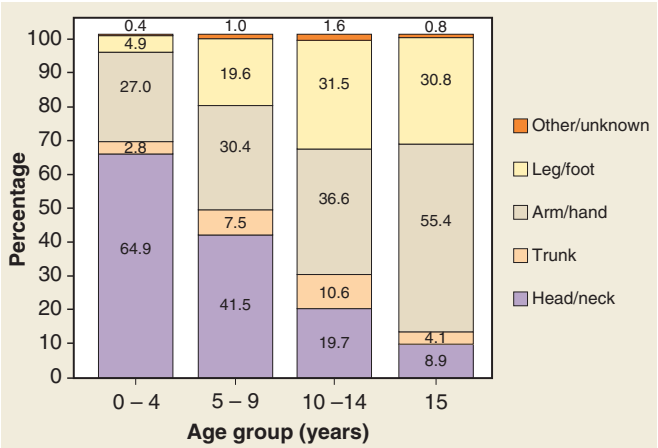


FIGURE 23-1 Percentage of nonfatal dog bite–related injuries treated in U.S. hospital emergency departments, by primary body part affected and age group—National Electronic Injury Surveillance System All Injury Program, United States, 2001. (Data from Centers for Disease Control and Prevention [CDC]: Nonfatal dog bite-related injuries treated in hospital emergency departments—United States, 2001. *MMWR Morb Mortal Wkly Rep* 52:605–610, 2003.)

TABLE 23-2 Dog Bite Injuries by Breed of Dog

Breed	Bite Injuries (% of all Bite Injuries)
Pit bulls	25
Golden retrievers	12
Labrador retrievers	12
Chows	10
German shepherds	10
Rottweilers	10
Doberman pinschers	<5

Data from Gandhi RR, Liebman MA, Stafford BL, Stafford PW: Dog bite injuries in children: A preliminary survey. *Am Surg* 65:863–864, 1999.

There were 1164 deaths caused by nonvenomous animals from 1979 through 1990.[28] From 1995 to 1996, 279 human deaths occurred as a result of dog attacks.[29] During 1997 and 1998, at least 27 dog bite–related fatalities occurred.[30] Although some deaths involved hunters who were attacked by large animals, such as bears, most of the deaths were caused by large domesticated dogs, especially Pit bulls, Rottweilers, and German shepherds.[23] The most frequent reason for death is exsanguinating hemorrhage. The victims are usually infants (60%) or older individuals.[20]

PATHOPHYSIOLOGY

HUMAN BITES

When humans chew, they generate forces in the range of 1.6 to 26.7 kg.[31-34] Maximum molar bite forces range from a low of 1 kg to a high of 443 kg. Although human incisors shear, canines tend to puncture and molars crush. Thus, human bites can penetrate, avulse, and crush the tissue. These injuries are contaminated by a wide range of microorganisms (Box 23-1).*

BOX 23-1 Common Microorganisms Transmitted by Human Bites

AEROBIC AND FACULTATIVE ISOLATES	ANAEROBIC ISOLATES
Acinetobacter spp.	*Bacteroides fragilis* group
Corynebacterium spp.	*B. oralis*
Eikenella corrodens	*B. urealyticus*
Enterococcus spp.	*Bifidobacterium* spp.
Haemophilus influenzae	*Eubacterium* spp.
H. parainfluenzae	*Fusobacterium nucleatum*
Klebsiella pneumoniae	*F. neorophorum*
Neisseria spp.	*Peptostreptococcus*
Nocardia spp.	*asaccharolyticus*
Proteus spp.	*P. magnus*
Pseudomonas	*Peptococcus niger*
Staphylococcus aureus	*Prevotella melaninogenica*
S. epidermidis	*P. intermedia*
Streptococcus	*Veillonella* spp.
α-hemolytic	
γ-hemolytic	
Group A β-hemolytic	
Non–group A β -hemolytic	

OTHER RARE PATHOGENS

Hepatitis B virus
Herpes simplex virus
Human immunodeficiency virus
Mycobacterium tuberculosis
Treponema pallidum

More than 42 different species of bacteria have been reported in human saliva. Consideration must be given to all normal oral flora and common pathogens in the treatment of these contaminated crush injuries. Although the amount and type of inoculum may be minimal in the case of pristine dentition, they are of significant concern in the neglected mouth. More than 190 species have been isolated when gingivitis or periodontitis is present. The most common bacteria associated with human bite infections include *Streptococcus, Staphylococcus aureus, Eikenella, Prevotella, Peptostreptococcus, Fusobacterium,* and *Candida* spp. Immunocompromised patients, alcoholics, and diabetics are at a higher risk for infection by human bites. Other transmissible human diseases that are of concern include herpes, hepatitis B, hepatitis C, tuberculosis, and syphilis.[37,38] Acquired immunodeficiency syndrome (AIDS) transmitted by saliva is considered to be of low or no risk, according to the Centers for Disease

*References 6, 9, 11, 12, 35, and 36.

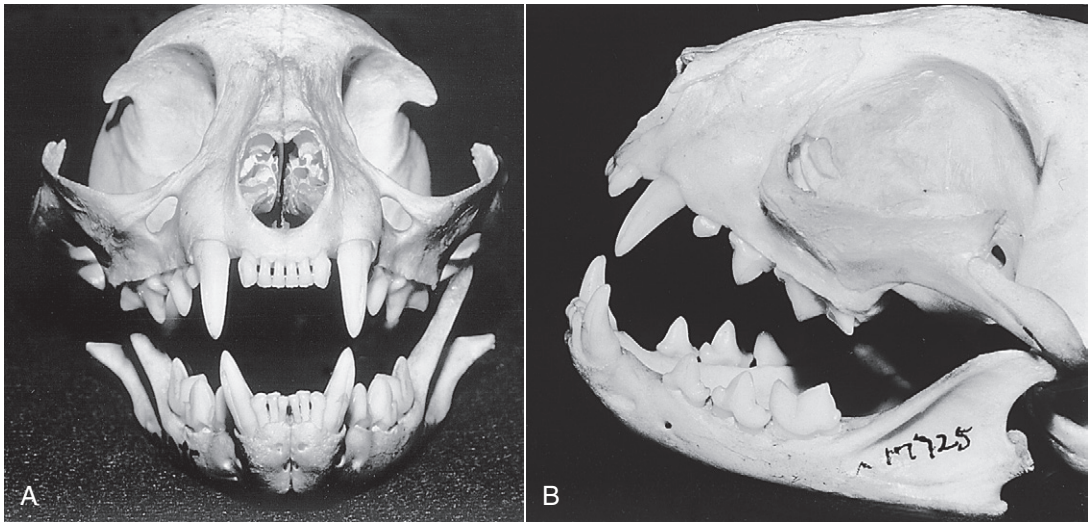


FIGURE 23-2 The long, thin, sharp teeth of the domesticated cat tend to cause puncture injuries, which are more frequently associated with infections.

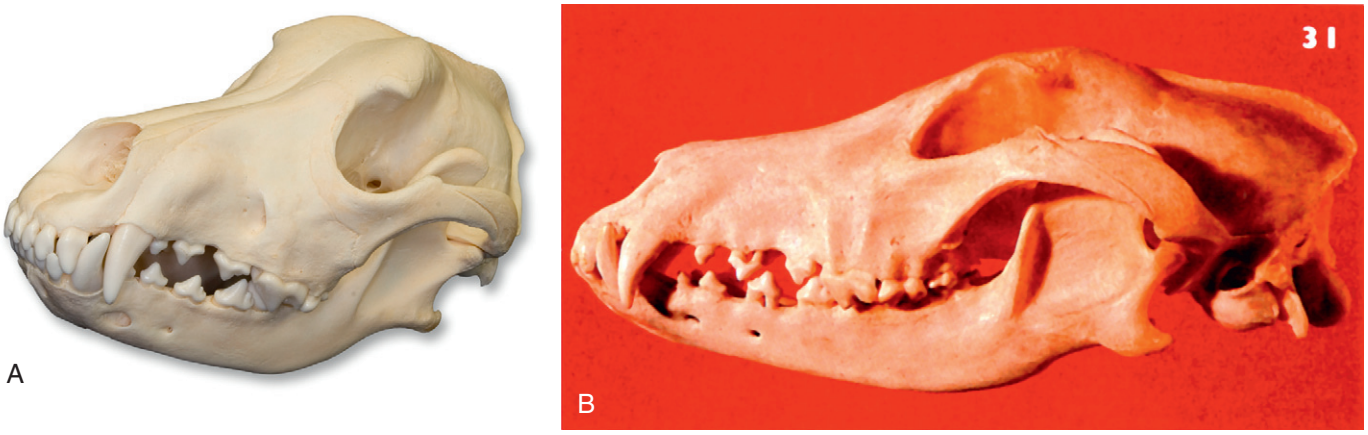


FIGURE 23-3 **A, B,** Shorter, rounder, blunter teeth tend to penetrate and crush, as in the case of the domesticated dog. (**B** from Boyd J, Paterson C: *Colour atlas of clinical anatomy of the dog and cat*, London, 2001, Mosby.)

Control and Prevention (CDC). However, a number of case reports have listed a human bite as the likely event responsible for transmission of AIDS infection and the use of postexposure prophylaxis has been advocated.[2,39,40]

ANIMAL BITES

The force delivered by an animal's jaw when biting can be as high as 1800 psi.[41,42] This can result in devitalized tissue caused by crushing, tearing, avulsion, or a combination of all three. Furthermore, these wounds are contaminated by a broad spectrum of microorganisms and can cause a wide range of zoonotic diseases. Sharp, thin, long teeth more frequently cause puncture injuries, as in the case of the domesticated cat[6,20] (Fig. 23-2). Shorter, blunter, rounder teeth frequently cause a combination of penetrating and crush injuries, as in the case of the domesticated dog (Fig. 23-3). Flat teeth cause crush and tear injuries, as in the case of the domesticated horse (Fig. 23-4). Animal bites may cause fractures in addition to soft tissue injury, a thorough examination should be performed to prevent missing critical injuries, especially in infants, children, and older adults (Fig. 23-5).

A list of microorganisms from all animal bite sources is presented in Box 23-2.* No species seems to be associated with any particular type of bacterial variant, except that animal bites differ from human bites in the presence of *Pasteurella multocida* in as many as 50% to 75% of cases of infection. Consistent with the heterogeneity observed between feline and canine oropharyngeal *Pasteurella* strains, *P. canis* biotype 1 is the predominant isolate from dog bites, whereas *P. multocida* subsp. *multocida* and *septica* have been isolated much more frequently from cat bites.[49]

The aerobic bacteria most frequently associated with infected animal bites include alpha- and beta-hemolytic streptococci (24% to 46%), *S. aureus* and *S. epidermidis* (10% to 25%), and *Escherichia coli*, *Pseudomonas*, and *Moraxella* spp. (10% to 25%).† The most common

*References 6, 9, 20, 35, 36, and 43-48.

†References 9, 35, 36, 43, 47, 48, and 50.



FIGURE 23-4 The domesticated horse has very flat incisors **(A)** and molars **(B)** and is capable of causing significant crush injuries.

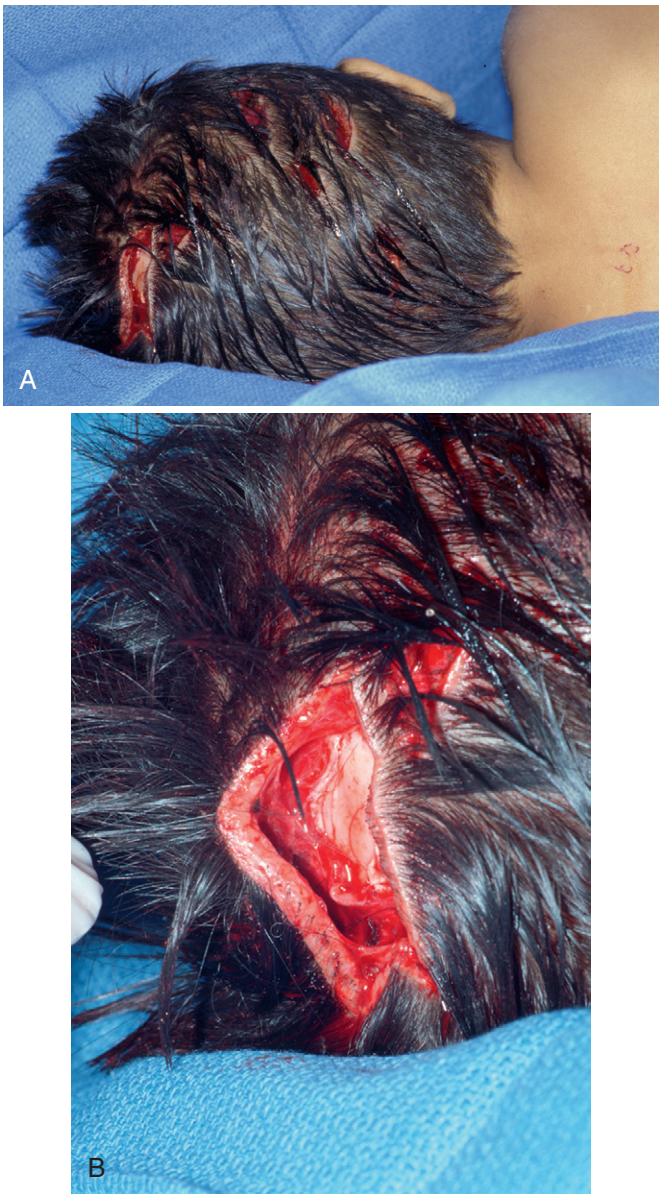


FIGURE 23-5 **A,** Multiple scalp lacerations caused by a dog bite in this young child. **B,** Injury extends to the subperiosteal plane.

anaerobic bacteria isolated from infected animal bites are the *Bacteroides, Prevotella,* and *Fusobacterium* spp. (13% to 76%). Oral and environmental fungi may also contaminate bite wounds. *Candida* spp. have been isolated from 8% of infected human bites, but their pathogenic role remains unclear.[51,52]

Tetanus

Tetanus is caused by *Clostridium tetani*, a gram-positive, anaerobic rod. *C. tetani* spores are resistant to heat and antiseptics and are found widely in soil and in the intestinal tract of domestic animals, including dogs and cats. Humans have also been known to harbor the organism.

The CDC has stated that 201 cases of tetanus were reported from 1991 to 1994, with an annual incidence rate of 0.02 cases/100,000.[53] There was a reported case fatality rate of 25%. Of these injuries, 49% resulted from puncture wounds, 20% from lacerations, and 12% from abrasions. Only 5 patients reported injury by an animal.

No bite injury should be considered clean or minor. Although the incidence of the tetanus may seem low, the high rate of mortality in patients who contract it makes the treatment of tetanus a priority. If adequate history of immunization cannot be elicited, the patient should be treated according to the Advisory Committee on Immunization Practices (Table 23-3).[53]

Rabies

The incidence of rabies has declined steadily since the 1950s.[54] Approximately 36 cases have been reported in the United States since 1990.[1,55] Only one case of rabies in a human was reported to the CDC during 2001. This viral disease is most frequently carried in wild animals (especially bats, raccoons, and skunks). Domestic animals accounted for 6.7% of all rabid animals reported in the United States during 2001; these were mainly cats, dogs, and cattle. Vaccination of domestic animals, vaccination programs targeting wildlife, and ongoing educational programs have contributed to the very low incidence of rabies in humans. Approximately 20% of human cases occur in the United States because of exposure abroad. Of the almost 80% of cases that occurred within the United States from 1990 to 2001, 92.3% of infected persons were found to have viral variants associated with bats.

The rabies virus is a bullet-shaped rhabdovirus.[56] After entering its new host, it travels to the central nervous system through the peripheral nerves, proliferating in

BOX 23-2 Common Microorganisms Transmitted by Animal Bites

AEROBIC BACTERIA
Acinetobacter spp.
Aeromonas hydrophila
Bacillus subtilis
Bordetella spp.
Brucella canis
Capnocytophaga canimorsus
Chromobacterium spp.
Clostridium perfringens
Corynebacterium spp.
EF-4
EF-7
Eikenella corrodens
Enterobacter spp.
Flavobacterium spp.
Haemophilus aphrophilus
Klebsiella
Micrococcus spp.
Moraxella catarrhalis
M. weaveri
Moraxella spp.
Neisseria spp.
Pasteurella multocida
Proteus mirabilis
Pseudomonas
Serratia marcescens
Staphylococcus aureus
S. epidermidis
S. saprophyticus
Streptococcus spp.
Weeksella zoohelcum

ANAEROBIC BACTERIA
Arachnia propionica
Bacteroides spp. (many species)
Eubacterium spp.
Fusobacterium spp.
Leptotrichia
Peptococcus
Peptostreptococcus spp.
Propionibacterium acnes
Veillonella spp.

OTHER RARE PATHOGENS
Bartonella henselas
Clostridium tetani
Francisella tularensis
Hepatitis B virus
Rabies virus
Herpes simplex virus
Leptospira spp.
Rio Bravo virus
Simian herpes B virus (macaque monkeys only)
Spirillum minus
Streptobacillus moniliformis
Yersinia pestis

the gray matter. It then spreads centrifugally to the salivary glands, adrenal glands, musculature, and heart. This mechanism accounts for the signs and symptoms associated with the different phases of the rabies infection. There is a long incubation period in rabies, typically lasting 20 to 90 days and rarely exceeding a year or more.[57] The prodromal phase usually ranges from 3 days to 3 months, the excitement phase lasts from 3 months to 6 months, and the brainstem dysfunction phase ranges from 6 to 12 months. Symptoms of rabies in animals include an unprovoked attack or bizarre behavior in domestic animals. Signs of infection in humans include symptoms of cerebral dysfunction, anxiety, confusion, and agitation, progressing to delirium, abnormal behavior, hallucinations, and insomnia.

If the bite has been inflicted by a human, chipmunk, guinea pig, gerbil, hamster, squirrel, rat, mouse, rabbit, or horse, there is little to no risk of rabies infection.[1,55] If the bite is from a healthy domesticated dog, cat, or ferret, the animal should be confined and observed for 10 days. Any sign of illness prompts sacrifice and testing of the animal and postexposure prophylaxis for the victim. Any bite by a wild animal, such as a raccoon, skunk, fox, or coyote is considered high risk and rabies prophylaxis must be given. Interestingly, bats are the most common source of exposure to rabies in the United States. Currently, postexposure prophylaxis is recommended for anyone with a bite, scratch, or mucous membrane contact with a bat.

TABLE 23-3 Summary Guide to Tetanus Prophylaxis in Routine Wound Management

	Clean, Minor Wounds		All Other Wounds*	
History of adsorbed tetanus toxoid (doses)	Td†	TIG	Td†	TIG
Unknown or <three‡	Yes	No	Yes	Yes
≥Three	No§	No	No¶	No

*Such as, but not limited to, wounds contaminated with dirt, feces, soil, and saliva, puncture wounds; avulsions, and wounds resulting from missiles, crushing, burns, and frostbite.
†For children < 7 yr old; DTP (DT, if pertussis vaccine is contraindicated) is preferred to tetanus toxoid alone. For those ≥7 yr of age, Td is preferred to tetanus toxoid alone.
‡If only three doses of fluid toxoid have been received, then a fourth dose of toxoid, preferably an adsorbed toxoid, should be given.
§Yes, if >10 yrs since last dose.
¶Yes, if >5 yrs since last dose. (More frequent boosters are not needed and can accentuate side effects.)

For victims at risk for rabies, management must begin immediately (Table 23-4); 20 IU/kg (0.133 mL/kg) of rabies immune globin (RIG) should be administered. Most of the dose should be injected in and around the site of the wound as much as possible, avoiding vascular compromise. The remainder should be given IM.[1,58] Rabies vaccine should also be administered in five doses IM on the day of the attack and on days 3, 7, 14, and 28 after the attack.[3,7,59] RIG should not be administered to patients who have been previously vaccinated, but they should receive additional vaccine. Three inactivated rabies vaccines are licensed for use in the United States. They are human diploid cell vaccine (HDCV), rabies vaccine absorbed (RVA), and purified chick embryo cell vaccine (PCEC).

TREATMENT OF BITE INJURIES

Management of bite wounds to the face is not unlike managing other trauma to the head and neck. Because bite wounds can cause a wide variety of injuries, and because they may not be isolated injuries, appropriate attention to complete evaluation and adherence to Advanced Trauma Life Support (ATLS) protocols is important; plain film or computed tomography (CT) scans should be ordered, when appropriate.[60] Certain aspects of management of these wounds are slightly different. The following section will highlight these differences and offer a protocol for treatment.

INITIAL PRESENTATION

Whether inflicted by humans or animals, the basic principles for the management of maxillofacial bite injuries are the same. ATLS protocols should be followed. Cervical spine immobilization and neurologic evaluation are particularly important because a large animal can grasp the underlying cranium with its fangs and crush the cranium of children and infants or shake them violently, thereby injuring the cervical spine.[61]

TABLE 23-4 Rabies Postexposure Prophylaxis Guide—United States, 2008

Animal Type	Evaluation and Disposition of Animal	Postexposure Prophylaxis Recommendations
Dogs, cats, and ferrets	Healthy and available for 10 days observation. Rabid or suspected rabid. Unknown (e.g., escaped).	Persons should not begin prophylaxis unless animal develops clinical signs of rabies.* Immediately vaccinate. Consult public health officials.
Skunks, raccoons, foxes and most other carnivores; bats	Regarded as rabid unless animal proven negative by laboratory tests.†	Consider immediate vaccination.
Livestock, small rodents, Lagomorphs (rabbits and hares), large rodents (woodchucks and beavers), and other mammals	Consider individually.	Consult public health officials. Bites of squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, other small rodents, rabbits, and hares almost never require antirabies postexposure prophylaxis.

*During the 10-day observation period, begin postexposure prophylaxis at the first sign of rabies in a dog, cat, or ferret that has bitten someone. If the animal exhibits clinical signs of rabies, it should be euthanized immediately and tested.
†The animal should be euthanized and tested as soon as possible. Holding for observation is not recommended. Discontinue vaccine if immunofluorescence test results of the animal are negative.

After initial assessment and stabilization, a comprehensive head to toe examination should be performed. This is followed by a thorough review of the past medical history of the victim, including the status of the patient's tetanus vaccination. The time of the bite and circumstances surrounding the bite should be noted and whether the animal was wild or domestic, whether the attack was provoked or unprovoked, and the number of bites and/or injuries.[1,29]

Once the secondary survey has been accomplished and a thorough history has been obtained, a complete physical examination should be performed. All organ systems should be evaluated. Wounds to the head, neck, or thoracic inlet deserve special attention, with a particular focus on the great vessels of the neck.[41] If present, consideration should be given to special imaging techniques, such as CT angiography or magnetic resonance imaging (MRI) angiography. The specialized structures of the face, including the parotid ducts, facial nerve, and nasolacrimal system, should be assessed individually. Care should be taken to evaluate the globe and an ophthalmologic consultation should be obtained, if indicated (Fig. 23-6).

A proposed classification of facial bite wounds, based on the extent of injury, is presented in Table 23-5 (Figs. 23-7 to 23-9).

ANTIBIOTIC PROPHYLAXIS

Human bites to humans have received considerable attention and a bad reputation. The reason for this is that most human bites affect the interphalangeal joints from a clenched fist hitting human teeth when a punch is thrown.[31,60] Oral flora is inoculated into the joint capsule. When the hand relaxes, the joint capsule becomes sealed. A dark moist region that is rich in nutrients and contaminated with microorganisms is the result. Because many of these patients are unreliable, they infrequently seek care until well after an infection has developed. When considering antibiotic prophylaxis, the microorganisms listed in Box 23-1 should be considered. Large, randomized clinical trials comparing antibiotics in human and animal bites have not yet been carried out. Some studies have suggested that empirical therapy should be amoxicillin with clavulanic acid.[11,50] Alternatives include fluoroquinolones, carbapenems, or combinations of clindamycin and penicillin or second- or third-generation cephalosporins.[35]

Antibiotic prophylaxis for animal bites continues to be debated, with few well-designed prospective studies available to shed any light on the controversy.[44,62-64] Although some studies have indicated that prophylactic antibiotics make a difference in the incidence of infection, others claim no efficacy. A review of randomized controlled trials comparing antibiotics with placebos in mammalian bites failed to show the effectiveness of prophylactic antibiotics for dog and cat bites in anatomic areas other than the hand.[63] Although documentation in support of prophylactic antibiotics for animal bites was not found in this review, only eight publications were included in the study and only dogs were included in more than one study. It seems clear that confirmatory research is necessary, but it is prudent to consider the type of wound, its location, and additional contamination or foreign bodies when deciding on possible antibiotic use. Amoxicillin with clavulanic acid is the drug of choice for prophylactic coverage.[58] If the patient is allergic to penicillin, treatment options include fluoroquinolones or clindamycin in combination with second- or third-generation cephalosporins. Azithromycin is probably the most appropriate choice for penicillin-allergic pregnant women or children, for whom tetracyclines, fluoroquinolones, and sulfa compounds are contraindicated.[65]

SURGICAL MANAGEMENT

The primary focus in the management of bite wounds is thorough wound cleansing by high-pressure, pulsed irrigation with copious amounts of normal saline[1,20,66] (Fig. 23-10). Some have used povidone-iodine (Betadine)

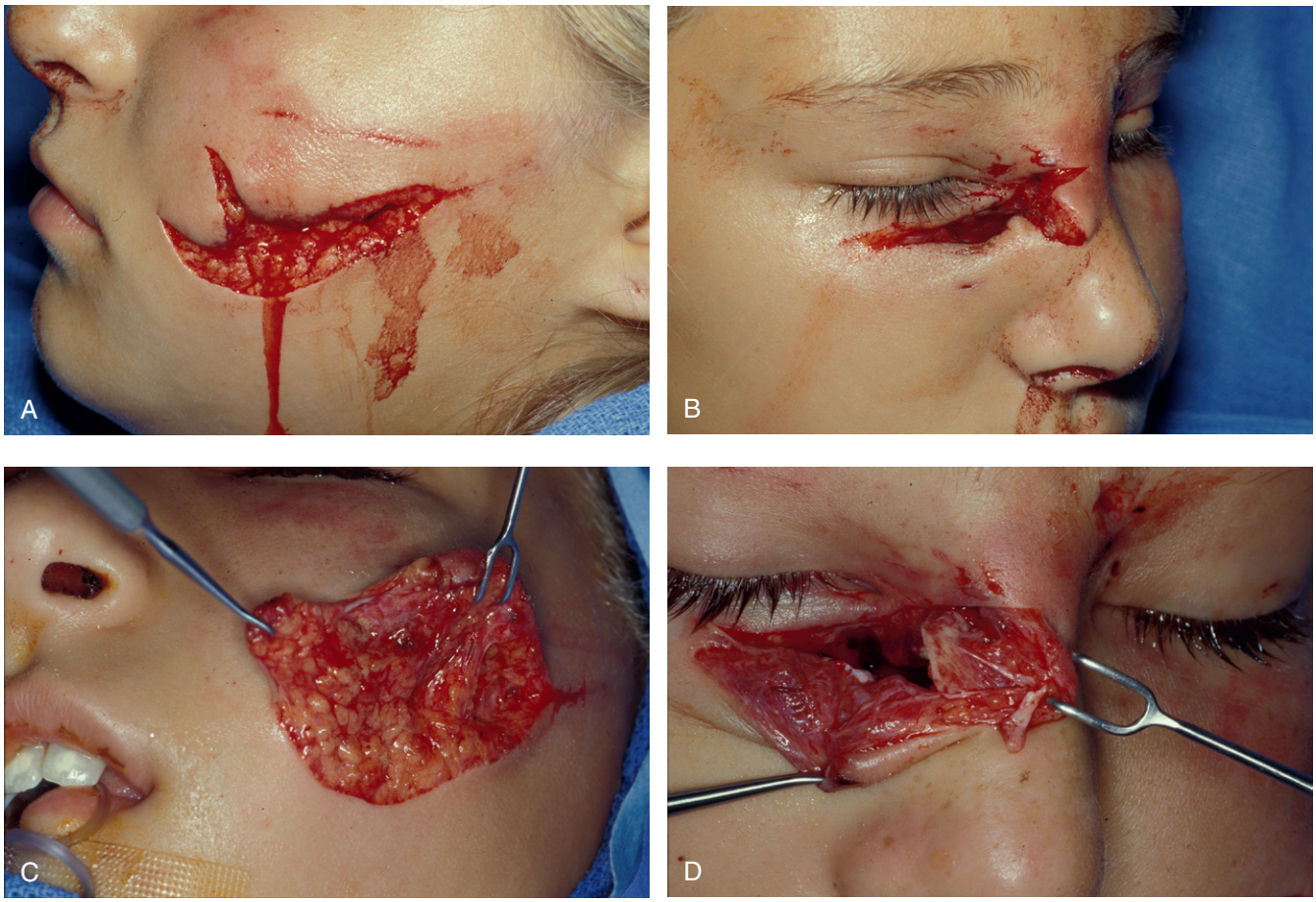


FIGURE 23-6 **A, B,** These lacerations were the result of a dog bite to the face. **C, D,** Exploration of significant structures should be performed when indicated. In this case, these would include the facial nerve, parotid duct, and the lacrimal system. Note that this injury includes fractures of facial bones. CT scanning is indicated in this situation. (Courtesy Dr. Edward Ellis III.)

TABLE 23-5 Classification of Facial Bite Injuries

Type	Clinical Findings
I	Superficial injury without muscle involvement (Fig. 23-7)
IIA	Deep injury with muscle involvement
IIB	Full-thickness injury of the cheek or lip with oral mucosal involvement (through and-through wound)
IIIA	Deep injury with tissue defect (complete avulsion)
IIIB	Deep avulsive injury exposing nasal or auricular cartilage (Fig. 23-8)
IVA	Deep injury with severed facial nerve and/or parotid duct (Fig. 23-9)
IVB	Deep injury with concomitant bone fracture

From Stefanopoulos PK, Tarantzopoulou AD: Facial bite wounds: Management update. *Int J Oral Maxillofac Surg* 34:469–472, 2005.

and/or hydrogen peroxide irrigation initially.[22] If these are used, normal saline should be used for a third irrigation to remove any remaining povidone-iodine. Iodine left in the wound can cause local tissue necrosis and inflammation. Gross necrotic tissue, foreign bodies, dirt, and debris must be removed. Wound infections caused by animal bites are typically of mixed flora and a wound culture has been advocated[58] (Fig. 23-11). Puncture wounds should be irrigated with the same solutions. This can be facilitated by the use of a flexible angiocatheter attached to a syringe, allowing access to the deepest part of the wound. An alternative is to take a scalpel and extend the puncture wound with two releasing incisions, creating a laceration.[20] This injury can then be irrigated more easily. Good wound cleansing is absolutely imperative. After the wound has been thoroughly cleansed, it should again be inspected. With debris, dried clots, and blood removed, better visualization is routinely afforded.

However, sustained high-pressure irrigation should be avoided in areas containing loose areolar tissue, such as the eyelids or children's cheeks, because this could cause tissue disruption and excessive edema. In general, 250 to 500 mL of solution provides an adequate cleansing effect for most facial bite wounds.[67,68]

After irrigation and reassessment, appropriate imaging techniques should be used if there is suggestion of a fracture. Hemorrhage should be controlled and necrotic tissue should be débrided. Specialized structures, such as the facial nerve, parotid duct, or nasolacrimal apparatus, should be repaired as indicated.

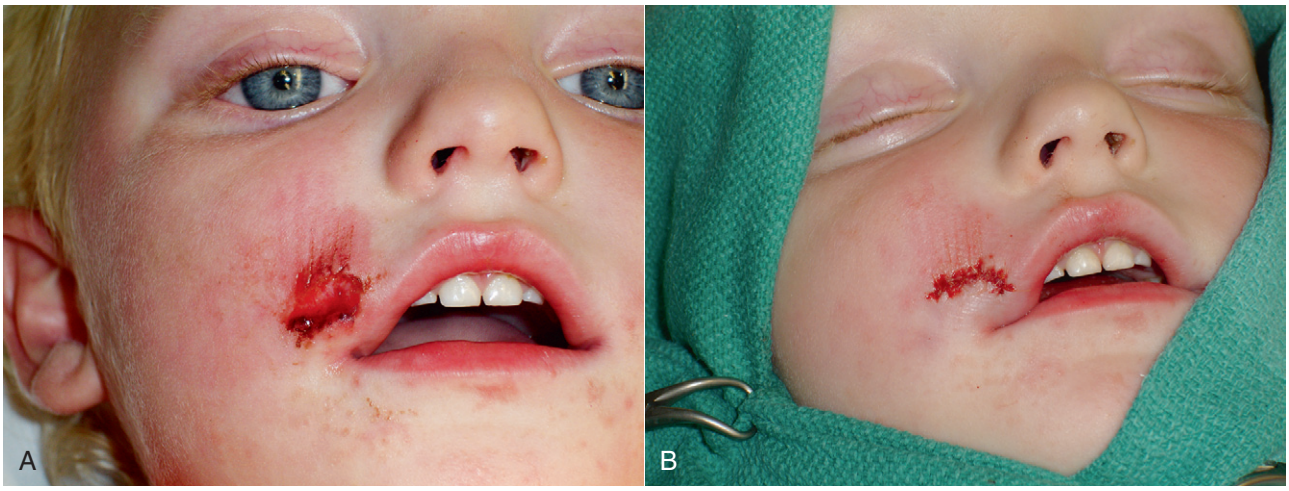


FIGURE 23-7 **A,** Small dog bite, facial injury classification type I, with no muscle involvement. **B,** Small dog bite injury treated with primary closure and topical antibiotics.



FIGURE 23-8 **A,** Human bite facial injury classification type IIIB, demonstrating partial avulsion and exposure of underlying cartilage nasal tip. **B,** Staged musculocutaneous dorsal nasal flap advancement, reconstructing the avulsed nasal tip. **C,** 4-week postoperative view of nasal tip reconstruction.



FIGURE 23-9 **A,** Large dog bite facial injury classification type IVA, demonstrating deep tissue injury in the region of the facial nerve and or parotid duct. **B,** Large dog bite injury demonstrating primary closure with residual areas of tissue avulsion. **C,** Large dog bite injury 1 year postoperative demonstrating scarring and soft tissue deformity and managed with intralesional steroids.

It has been recognized for at least 4 decades that uncomplicated lacerations from bites to the face can be closed primarily within the first 24 hours—the golden 24-hour period—without fear of increased rates of infection.[69-74] This also produces the most favorable aesthetic results, although this opinion has been challenged, particularly in human bites to the face resulting in exposed cartilage.[5] A layered closure should be performed with the minimum number of deep resorbable sutures necessary to eliminate dead space and tension, followed by closure of the skin with nonresorbable monofilament sutures.[20]

Two additional factors pertaining to the face can render the management of bite wounds in this area problematic. The first is a substantial risk of occult oral communication with bite injuries of the cheek because of the nature of the animal occlusion. The second is the presence of the relatively avascular buccal fat pad, which is well developed in children and, once exposed, does not resist infection well. Thus, in cases of deep bites to the cheek, especially in children, antibiotic prophylaxis should be started as soon as possible after careful exploration and irrigation, usually with the first dose administered IV.

Puncture wounds should be left open or converted to lacerations and then closed as described. Specialized techniques of wound pexis, skin grafts, and regional flaps should be considered when appropriate.[21,25,75,76] Avulsed tissue may require microvascular repair or tissue banking.[77,78] Adherence to the principles of irrigation, meticulous wound care, minimal débridement, and primary closure can provide favorable results[20,21,73] (Fig. 23-12; Box 23-3).

POSTSURGICAL TREATMENT

Postsurgical management includes local wound care and antibiotic therapy for 5 to 7 days after surgery.[1] Depending on the type of attack and age of the victim, psychiatric

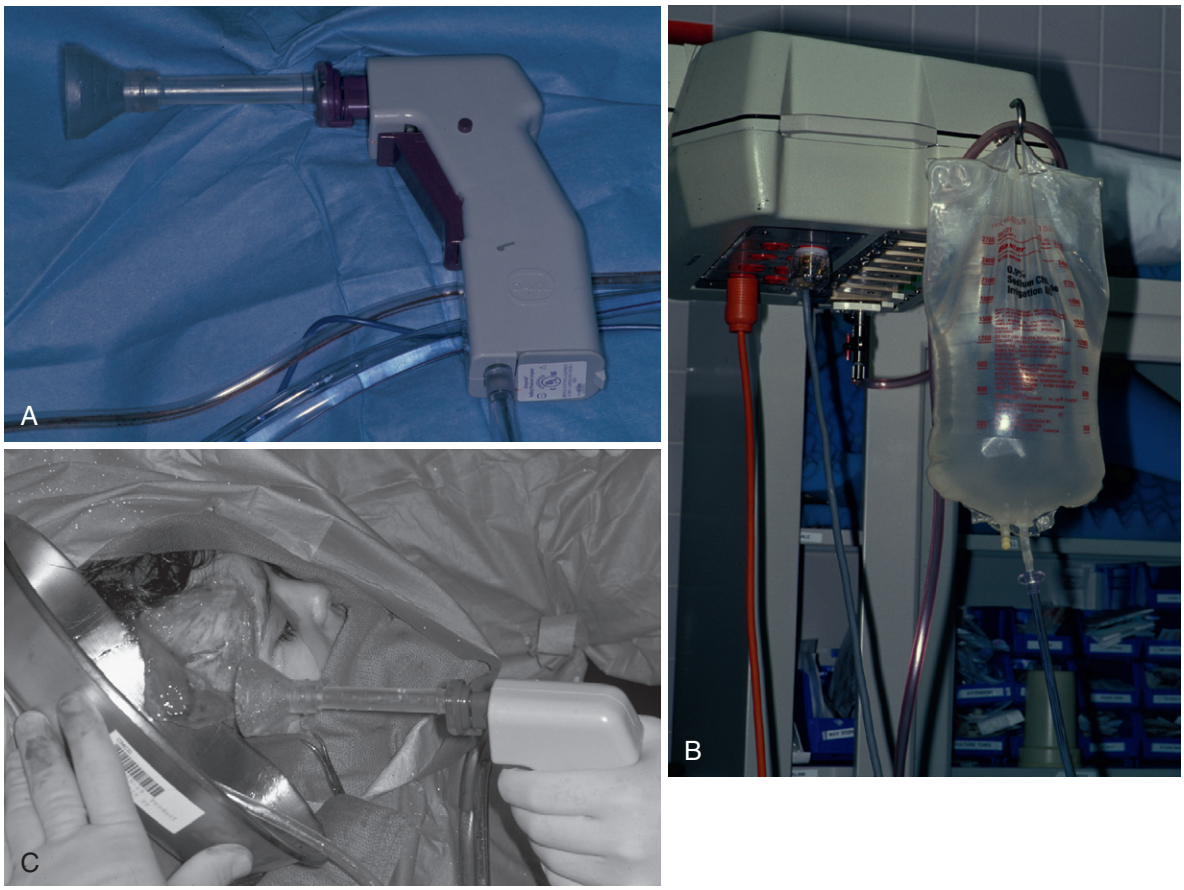


FIGURE 23-10 **A, B,** Pulsatile irrigation system—3-liter bags of saline are pumped through a handpiece that provides pulsation and suction during wound irrigation. **C,** Use of the pulsatile irrigation system for wound cleansing.

or social counseling may be required. This is especially true for children or victims of sexual assaults.[4] Signs and symptoms of infected wounds and even zoonotic diseases should remain a concern.[58] Finally, scar revision and secondary reconstruction can be considered as soon as 6 months after the injury.

FORENSIC BITE MARK RECOGNITION, EVIDENCE, AND ANALYSIS

Bite mark evidence has been widely accepted by courts in the United States for many years on the basis that the human dentition is unique to each person. The conviction of mass murderer Theodore Robert Bundy was secured by bite mark evidence and bite mark analysis testimony (Bundy was executed in Florida on January 24, 1989). Bite mark evidence has become a powerful investigational tool in the United States. However, bite mark evidence and analysis are not without controversy. This section will provide the practitioner with an overview of bite mark recognition, evidence, and analysis.

BITE MARK RECOGNITION

A cutaneous human bite mark, as defined by the American Board of Forensic Odontology (ABFO), is an injury on the skin caused by contacting teeth that shows the representational pattern of the oral structures.[79] A human bite mark, therefore, is a patterned injury to the skin similar to a tool mark patterned injury. In most cases, human bite marks are associated with violent crimes, such as homicide, rape, sexual assault, child and spousal abuse, and assault.[10,80,81] With the violent nature in which human bite marks are usually received, it is important that medical and dental personnel, day care and nursing home workers, teachers, criminal investigators, and medical examiners have proper training in the recognition of patterned injuries on the skin as possible bite mark injuries.

Knowledge of the anatomic location of human bite marks is pivotal for all who are involved with examining the victims (living and deceased) of violent crimes. Hospital-based studies of biting incidence have indicated that the extremities, especially hands, are the most commonly bitten areas.[4] However, information from coroners' data has suggested that bites are more commonly delivered to breasts. One study of 101 human bite mark cases, using the Lexis legal database, has concluded that females were four times more likely to be bitten than males and that bites occurred more frequently on breasts than on extremities.[10]

To recognize a human bite mark, one must be familiar with the shape of this particular patterned injury on the skin. In general, skin is a poor impression medium for



FIGURE 23-11 **A-D,** This purulent infection developed 1 week after treatment of this dog bite, despite pulsatile irrigation and IV antibiotics during closure.

recording tooth imprints because of its rebounding nature over time. Therefore, the evidence that remains on the skin generally takes the form of bruising patterns. Bite marks usually appear as oval or circular contusions or abrasions, with or without indentations or lacerations, and are described as having a dotted or broken line pattern (Fig. 23-13). Any distinguishing factor, trait, or pattern in a given bite mark is referred to as a characteristic of the mark. There are two categories, class characteristics and individual characteristics.[80]



FIGURE 23-12 2-month postoperative results of the patient seen in Figure 23-6. (Courtesy Dr. Edward Ellis III.)

BOX 23-3 Treatment Protocol For Common Facial Bite Wounds

1. Skin preparation; anesthesia
2. Pressure irrigation; irrigation of puncture wounds
3. Resection of skin tags
4. Removal of visible foreign particles
5. Suturing (exceptions listed below)
6. Consideration of tetanus prophylaxis
7. Follow-up within 24 to 48 hours

ALSO RECOMMENDED:

- Normal saline irrigation (1% povidone-iodine should be reserved for grossly contaminated wounds)
- Antibiotic prophylaxis
- Culture of problematic wounds (failure to respond to initial antibiotic therapy or presence of serious infection)

NOT RECOMMENDED:

- Routine debridement (if attempted, it should not exceed 1 mm of tissue)
- Suturing in the presence of overt infection, gross edema, foreign bodies, or visible contamination (consider delayed closure)
- Culture of fresh uninfected wounds, because it depicts the polymicrobial flora of the wound rather than the causative organisms of any subsequent infection

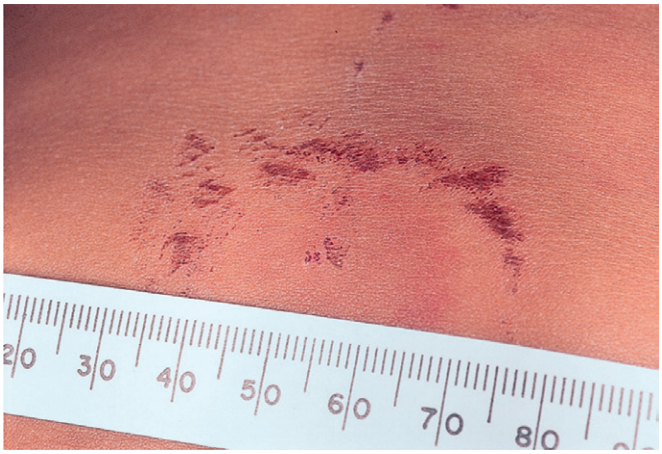


FIGURE 23-13 Human bite mark showing the circular pattern, with the broken line bruising containing an area of central ecchymosis. (From Swartz M: *Textbook of physical diagnosis: History and examination*, ed 6, Philadelphia, 2010, Saunders.)

Class Characteristics

A class characteristic is a feature, trait, or pattern seen in, or reflective of, a given group. Teeth are divided into morphologic classes—incisors, canines, premolars, and molars. Each class will represent a somewhat different pattern in the bite mark injury. Incisors, for example, usually produce linear or rectangular contusions, whereas canines typically leave triangular bruises. Maxillary incisors will produce larger patterns because of their size, which helps distinguish the maxillary arch from the mandibular arch. Arch size differences are important in determining whether a bite mark was produced by a child or adult.

Individual Characteristics

An individual characteristic is a feature, trait, or pattern that represents a variation in the class characteristics. Examples include but are not limited to rotations, fractures, missing restorations, malalignment, and spacing between marks. The number of discernible individual characteristics present in the bite mark is used to distinguish between two different dentitions. The higher the number, the more distinctive the bite mark and the higher the confidence level that a particular suspect made a given bite mark.

BITE MARK EVIDENCE

Ordinarily, a forensic odontologist should be called on to perform a preliminary examination when a human bite mark is suspected. Several basic questions must be answered during this initial evaluation:

1. Is the patterned injury a bite mark?
2. If the injury is a bite mark, did human teeth cause the injury?
3. Is the bite mark consistent with the type of crime and time of occurrence?
4. Does the bite mark contain distinct or unique individual characteristics of the biter's teeth?

Once it has been ascertained that the patterned injury is a bite mark, it is important to determine that an animal

did not produce the bite mark. Domesticated animals and wild animals, such as rats, may have access to a crime scene. Typically, animal bites are V-shaped and have deep lacerations, but do not contain areas of ecchymosis within the confines of the bite mark. In contrast, human bites are circular or elliptical, have superficial abrasions, and commonly contain ecchymosis within the bite mark injury secondary to positive pressure from closing the teeth and damage to capillaries or to negative pressure from sucking and tongue thrusting[81] (see Fig. 23-13). Intercanine distances less than 2.5 cm indicate that a child may have caused the injury. Bite marks with intercanine distances more than 3 cm were likely caused by adults.

Although bite marks are usually associated with violent crimes, bite marks may occur at other times, such as during sexual relations not associated with a crime. The age of the bite mark must be consistent with the time of the crime occurrence to be used as evidence in the case. The bite mark should contain enough individual characteristics to allow the forensic dentist to prove or disprove a physical match to a suspect's teeth with a high degree of confidence. At the conclusion of the preliminary examination, it is the responsibility of the forensic odontologist to make a judgment about to the evidentiary value for recovering and analyzing the bite mark.[77]

Bite Mark Evidence Recovery

After it has been decided to recover bite mark evidence from the victim, it must be collected expeditiously because the appearance of the bite mark starts to change quickly, especially on the living victim. Most forensic dentists follow a standard protocol for bite mark evidence recovery that includes photographing the bite mark, swabbing the bite site, making impressions of the injury, and obtaining tissue specimens.

Photography. Extensive photographing of the bite injury, before any manipulation, is generally the first step in evidentiary collection. Color and black and white print film should be used. Color prints represent a realistic reproduction of the bite mark, whereas black and white prints tend to make the bite mark more evident.[82] At times, color photographs may be considered inflammatory and not admitted into court as evidence, whereas black and white photographs are routinely admitted. Because the bite mark injury appearance changes over time, it is important to photograph the bite mark at regular intervals, usually every 24 hours, for several days.

Photographs should be made with the lens plane parallel to the plane of the bite mark to decrease perspective distortion. This is a challenging task because most surfaces on the human body are curved. A scale should be used to estimate the amount of photographic distortion. The ABFO no. 2 reference scale has been specifically designed for this purpose and has been recommended by the ABFO[83,84] (Fig. 23-14). Photographs should be made with and without the scale in place. The photograph without the scale demonstrates to the court that it does not cover any relevant evidence.

Swabbing. Swabbing the bite mark may provide valuable salivary trace evidence. Approximately 85% of the general population are secretors who reveal their ABO blood group classification in their saliva. In addition, recovery of human DNA evidence is possible through swabbing the bite mark injury.[85] Evidence is collected by using the double-swab technique,[86] which involves moistening the bite injury with a sterile cotton swab moistened with sterile distilled water, followed by moisture removal from the site with a second dry swab. Control swabbings should be made on other areas far removed from the bite mark.

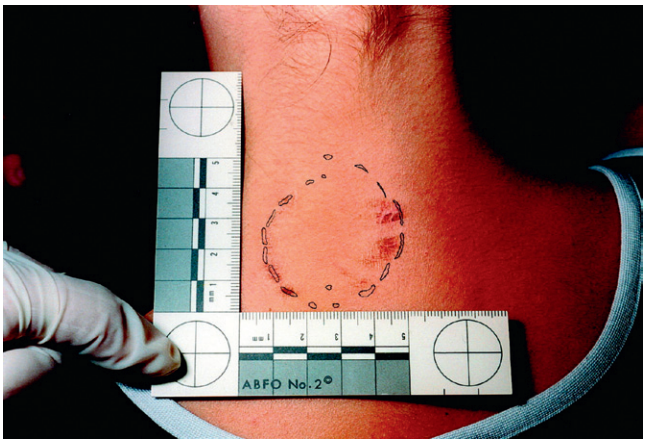


FIGURE 23-14 ABFO no. 2 reference scale. (From Byard R, Corey T, Henderson C: *Encyclopedia of forensic and legal medicine*, Kidlington, England, 2005, Academic Press.)

Impressions. After photographs and swabbings have been completed, impressions of the bite mark surface should be made when it appears that valuable information will be provided (ABFO Bite Mark Analysis Guidelines).[79] All hair should be removed from the bite mark location and the site washed, rinsed, and dried. A low viscosity impression material (one that meets American Dental Association [ADA] specifications) is put on the area and allowed to set completely. The impression material must be reinforced with a rigid backing material to reproduce the anatomic contour accurately. Commonly used backing materials include dental stone, acrylic resin tray material, thermoplastic tray material, and orthopedic mesh.[87] Once the selected backing material has set, the impression is poured in an ADA type IV dental stone, using the manufacturer's instructions. The master cast is used for court demonstrations and casts produced from subsequent pours are used for evaluation and analysis.

Tissue Specimens. With approval from the medical examiner or coroner, bite mark injury patterns on deceased individuals may be excised for photography with transillumination and preservation for future analysis and comparison.[88] One technique for bite mark removal is to use a custom-fabricated acrylic stabilizing ring and permanently attach it to the skin using cyanoacrylate and interrupted sutures.[89] Vinyl polysiloxane (VPS) impression material is injected on the surface of the skin within the stabilization ring to serve as a backing for the anatomic contour of the area to be excised. Once the impression material is set, the entire complex (ring, backing, and skin) is dissected and removed from the body. The entire specimen is placed in 10% buffered formalin fixative for at least 10 hours. After fixation, the

VPS backing material may be removed while leaving the ring and sutures intact. The tissue specimen should be placed in a sealed plastic bag and stored in a refrigerator for future analysis.

Protocol of Evidence Recovery. Before collecting evidence from a suspect, the forensic dentist must ensure that the proper legal documentation has been obtained. Extraoral photographs should include head and neck portrait and profile views. Intraoral photographs should include the frontal view and lateral views at maximum intercuspation (MI), maximum interincisal opening, close-up of the anterior teeth in MI and protrusion, and occlusal views of the maxillary and mandibular arches. The intraoral photographs should be made with and without the ABFO no. 2 reference scale in place. Other evidence to collect from the suspect would include an extraoral and intraoral examination, salivary swab, blood sample, two impressions of each arch, and sample or test bites in various materials.

When the bite mark is located in an area accessible to the victim, impressions of the victim's dentition and comparison to the marks is advisable. On occasion, victims do bite themselves during the passion of an intense fight. If an investigator fails to perform this task, the defense often will bring forth this possibility to the embarrassment of the prosecution.

BITE MARK ANALYSIS

Human bite mark analysis is possibly the most challenging aspect of forensic odontology. Combining the subtle nature of bite mark evidence with the highly subjective interpretation of this evidence is a major problem related to bite mark analysis. Because of this problem, differences in expert opinions—even among board-certified forensic dentists—should be expected during trial.

Comparative analysis involves the examination of the bite mark evidence and comparison against suspect evidence to determine whether a positive identification is possible. There are many comparison methodologies used by forensic dentists in the analysis of bite mark evidence, including visual comparison, life size overlays, test bites (exemplars), digital bite mark overlays, scanning electron microscopy, and metric analysis. The most commonly used comparison methods involve some type of overlay technique and metric analysis.

Fabrication of Bite Mark Overlays

Many methods for fabricating bite mark overlays have been published.[90-92] One technique is described as follows:

1. Life-sized (1:1) photographs are produced of the bite injury (Fig. 23-15*A*).
2. Once stone casts of the suspect's teeth have been generated and duplicated, the incisal edges and cusp tips are inked on the duplicate cast. Life-sized (1:1) photographs are made of the suspect's casts, with a scale in place. Two Xs are placed on the photograph to serve as reference points. A transparent acetate sheet is placed over the life-sized photograph of the suspect's cast and the incisal edges are traced and labeled. The Xs are also traced to prevent the overlay from inadvertently being reversed. On the flip side of the transparency, each tooth tracing is given its respective tooth number (see Fig. 23-15*B*).
3. The transparency is placed just short of exact superimposition over the 1:1 photograph of the bite mark to view concordant points (matching points) of the suspect's teeth with the bite mark (see Fig. 23-15*C*).
4. Exact superimposition of the acetate overlay of the suspect's teeth over the life-sized photograph of the bite mark is used to determine the degree of match (see Fig. 23-15*D*).

The use of hand tracings from life-sized photographs and photocopies of the suspect's dental stone casts has decreased over the years with the introduction of a more accurate, less subjective technique, the computer-generated bite mark overlay.[91]

Metric Analysis

In addition to the fabrication of an overlay, careful inspection of the suspected biter's teeth is necessary. With metric analysis, once casts and exemplars have been generated, each characteristic of the suspect's teeth is measured accurately using calipers and recorded.[83] This includes the size of each tooth, intercanine distance, size of any diastema, degree and direction of any rotated and/or malaligned teeth, and notation of any missing teeth. Each characteristic of the suspect's teeth that is registered in the bite mark will also undergo a similar analysis. Then, there is a comparative analysis of the measurements recorded from the bite mark and that of the suspect's teeth to determine the degree of match.

Results of Analysis

After bite mark analysis and evaluation of other evidence, including DNA evidence, crime scene photographs, and written medical treatment reports, the forensic dentist must prepare a report of the bite mark analysis results and form an opinion. The opinion may link a suspect to the bite mark, exclude a suspect as the biter, or neither.[82] Specific bite mark terminology has been recommended by the ABFO to prevent miscommunication when used as a conclusion in a report or testimony.[79] The terms relate the degrees of certainty when describing the link between the bite mark and suspect.

Although the conviction of a criminal based on bite mark evidence alone is extremely rare, bite mark evidence provides additional information in connecting a suspect to a crime or an injury produced during a crime. It is the bite mark evidence in conjunction with the other evidence that leads to a conviction. Bite mark analysis continues to be a challenging yet fascinating and important aspect of forensic odontology.

SUMMARY

Human and animal bites are complicated wounds. They consist of crush injuries with punctures, lacerations, and avulsions that are contaminated by a plethora of microorganisms. Underlying fractures and concomitant multiorgan systems injuries must not be overlooked. Antibiotic prophylaxis using amoxicillin with clavulanic acid is encouraged. Because these are neither clean nor minor wounds, tetanus prophylaxis must be considered. Rabies

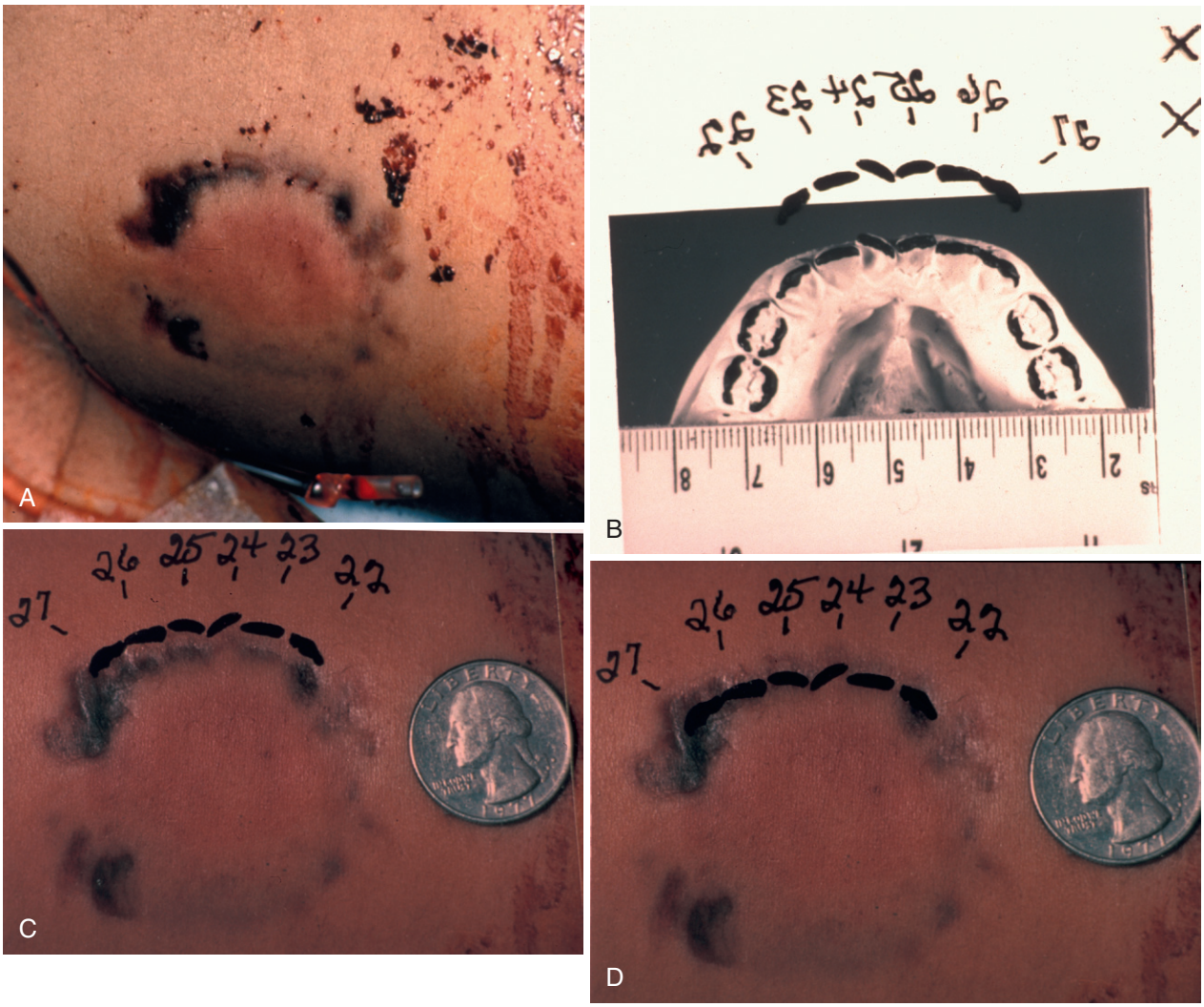


FIGURE 23-15 **A,** Victim with a bite mark on torso. **B,** Photograph of the suspected biter's cast with the mandibular teeth inked and acetate overlay tracing. **C,** Acetate overlay tracing shown slightly above bite injury to visualize concordant points. **D,** Exact superimposition of overlay on life-sized bite mark injury photograph. (Courtesy Dr. Douglas Damm.)

prophylaxis is based on the incidence for the particular species in a specific geographic area or on laboratory confirmation in the animal or victim. The single most important principle in the management of these injuries is absolute, meticulous wound cleansing. Irrigation with copious amounts of saline or povidone-iodine solution and saline, along with vigorous scrubbing, conservative débridement, and careful foreign body removal, cannot be overemphasized. Lacerations in the maxillofacial region should be closed primarily. Postoperative management includes local wound care along with keen observation for infection and the transmission of local or systemic diseases, or both. Post-traumatic psychological counseling should be considered in the pediatric patient population or for victims of sexual assaults.

ACKNOWLEDGMENTS

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CHAPTER

24

Diagnosis and Management of Traumatic Salivary Gland Injuries

Brian Bast | Norman J. Betts | Michael P. Powers

OUTLINE

Anatomy
Parotid Gland and Duct
Submandibular Gland and Duct
Sublingual Gland and Duct
Minor Salivary Glands
Mechanism of Salivary Gland and Ductal Injuries
Diagnosis of Salivary Gland Injuries
Treatment of Salivary Gland Injuries
Minor Salivary Gland Injuries
Submandibular Gland Injuries
Sublingual Gland Injuries
Parotid Gland Injuries
Isolated Glandular Injury
Ductal Injury
Management of Salivary Gland Injury Complications
Iatrogenic Injuries to the Salivary Gland
Parotid Sialocele and Fistula
Submandibular Gland Mucocele

Trauma to the facial soft tissues and fractures of the maxillofacial complex may involve the salivary structures, but the relative incidence of injury to the salivary structures is low. Salivary gland injuries are usually associated with penetrating injuries or complex fractures of the facial bones.[1,2] Salivary gland trauma can be classified as acute (blunt, lacerating, avulsion, or blast injuries) or chronic (inflammatory damage associated with infection, foreign bodies, sialoliths, or irradiation). Management of salivary gland injuries may vary according to factors such as the mechanism of injury, site of injury, degree of contamination, associated injuries, and general medical status of the patient. Lacerations in the cheek region posterior to the anterior border of the masseter muscle along a plane from the tragus of the ear to the middle of the upper lip (Fig. 24-1) may involve the parotid gland or duct, various branches of the facial nerve, and the facial artery and veins. Soft tissue injury in the floor of the mouth or beneath the mandible can damage the submandibular or sublingual glands, their associated ducts, or both. However, the incidence of injury to these salivary glands is lower than that for the parotid gland because of the protection of these salivary structures by the body of the mandible.

Salivary gland injuries can be serious and frequently associated with long-term morbidity. Delay or failure to diagnose may result in morbidity secondary to soft tissue scarring and disfigurement, sialocele, cutaneous fistula, or gustatory sweating associated with Frey's syndrome.[3,4] Surgical and nonsurgical approaches are used in the treatment of salivary gland and ductal injuries. Nonsurgical approaches are generally used only when the gland parenchyma is contused but the ductal structures remain intact. Surgical techniques are used to repair extensive injuries. Unfortunately, injuries to the salivary glands and/or supporting structures are often overlooked or underestimated in patients who have suffered significant or massive facial trauma. Thorough assessment and proper management of these injuries can prevent costly and unpleasant sequelae as acute treatment is usually much less difficult than late management.[5,6]

ANATOMY

PAROTID GLAND AND DUCT

The parotid gland is a unilobar structure with superficial and deep portions. These two segments of the gland are connected by a small isthmus, within which lies the facial nerve. The gland is encapsulated by a fascia formed by a splitting of the superficial layer of the deep cervical fascia. The bulk of the parotid gland overlies the ramus of the mandible and is bounded superiorly by the zygoma and anteriorly by the masseter while posteriorly abutting the external auditory canal and the anterior superior aspect of the sternocleidomastoid muscle. The parotid tail curves posteriorly around the mandibular ramus and is rarely involved in traumatic injury. The parotid duct, or Stensen's duct, exits from the anterolateral portion of the gland and passes beyond the anterior border of the masseter muscle, parallel with a plane drawn from the tragus of the ear to the midpoint of the upper lip. Approximately 1 cm anterior to the anterior border of the masseter muscle, the duct turns medially and penetrates the buccal fat pad and buccinator muscle, opening into the mouth at the level of the maxillary second molar. The facial nerve emerges from the stylomastoid foramen, passes into the substance of the gland, and subdivides into multiple branches that emerge anteriorly from the gland substance. Stensen's duct is divided into the glandular portion, the proximal (masseteric) portion and a distal (buccal) portion.[7] Generally, the buccal branch of

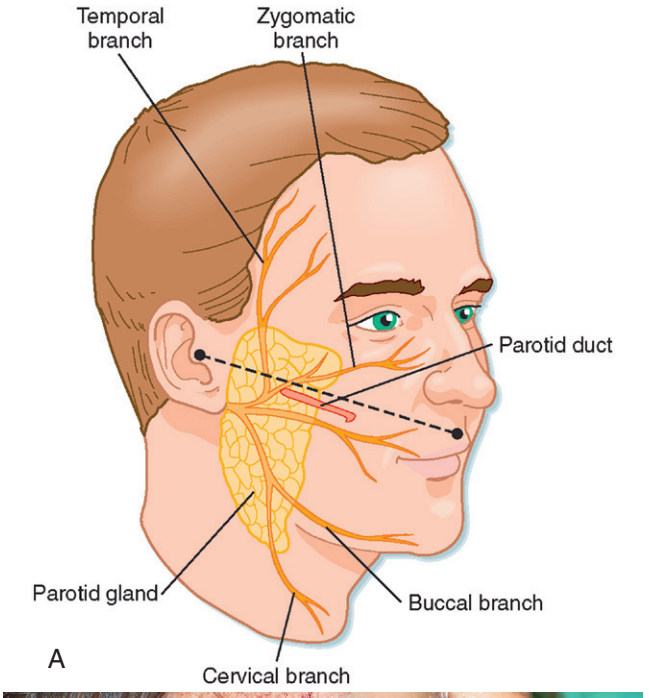


FIGURE 24-1 **A,** A line drawn from the tragus of the ear to the middle of the upper lip approximates the course of the parotid duct. Injury to the parotid gland duct is usually located at or distal to the anterior border of the masseter muscle along this line. **B,** Facial laceration with high risk of injury to parotid gland and duct. (Part A from Adams J, Barton E, Collings J, et al.: Emergency medicine, St. Louis, 2008, Saunders.)

the facial nerve can be found 5 to 10 mm below the parotid duct as it exits the gland. The nerve and duct are especially vulnerable to injury as they pass relatively unprotected over the superficial surface of the masseter muscle and exit the anterior portion of the parotid gland before descending within the fibers of the orbicularis oris and buccinator muscles.[4,8] Several vascular structures are associated with the parotid gland, including the retromolar and external jugular veins and superficial temporal, external carotid, and facial arteries.

SUBMANDIBULAR GLAND AND DUCT

The submandibular gland lies below and in front of the angle of the mandible and occupies most of the posterior portion of the submandibular triangle. The gland lies deep to the platysma muscle, bounded anteriorly and inferiorly by the digastric muscle, the myohyoid muscle deep, and the hypoglossus muscle posteriorly. The greater part of the gland is superficial, but it folds around the posterior border of the mylohyoid muscle so that a small portion of the gland is found in the posterior floor of the mouth. The facial artery is intimately associated with the superficial portion of the gland. The submandibular duct, or Wharton's duct, arises from the deep portion of the gland, is about 5 cm long, and has relatively thin walls and a constricted orifice. The duct leaves the submandibular triangle by passing above the posterior border of the mylohyoid muscle traversing along the floor of the mouth and opening in the anterior floor of mouth. In the floor of the mouth, the duct passes from lateral to medial on its way to the sublingual caruncle. Here, the duct has an intimate relation to the lingual nerve, which crosses it twice on its forward path (Fig. 24-2).[9,10] The facial vein and artery are in close association with the submandibular gland and generally lie just posterior to the gland. The marginal branch of the facial nerve provides function to the depressor muscles of the lower lip and runs over the fascia superficial to the facial artery, facial vein, and gland and can extend to the most inferior level of the gland.

SUBLINGUAL GLAND AND DUCT

The sublingual gland is the smallest of the three major salivary glands. It is narrow and flattened and lies below the mucosa bilaterally in the floor of the mouth, above the mylohyoid muscle. About 12 small sublingual ducts (ducts of Rivinus) emanate from the superior border of the gland and open separately in the floor of the mouth.[10-12]

MINOR SALIVARY GLANDS

A discussion of the anatomy of salivary glands would not be complete without mention of the minor salivary glands. Minor salivary glands provide the bulk of lubrication for the mucosal surfaces of the oral cavity. These glands are located throughout the oral cavity, with the majority in the palate, the labial and buccal mucosae, the ventral surface of the tongue (Blandin and Nuhn's glands), and the alveololingual glands in the lateral floor of the mouth. These minor glands lack a well-developed ductal system. Instead, these glands, which are located in the submucosal layer, have short ducts that open directly on the mucosal surface.[10,13]

MECHANISM OF SALIVARY GLAND AND DUCTAL INJURIES

Sharp penetrating trauma is the usual mechanism that results in acute injury to the salivary glands and surrounding anatomical structures. Lewis and Knottenbelt have reported the incidence of parotid ductal injury as 0.21% (33 of 15,419) of all trauma cases evaluated over a 6-month interval, with most of these injuries being assaults with a knife or bottle.[1] The most common cause of parotid gland injury is penetrating trauma from assault weapons or broken glass from a bottle or an automobile

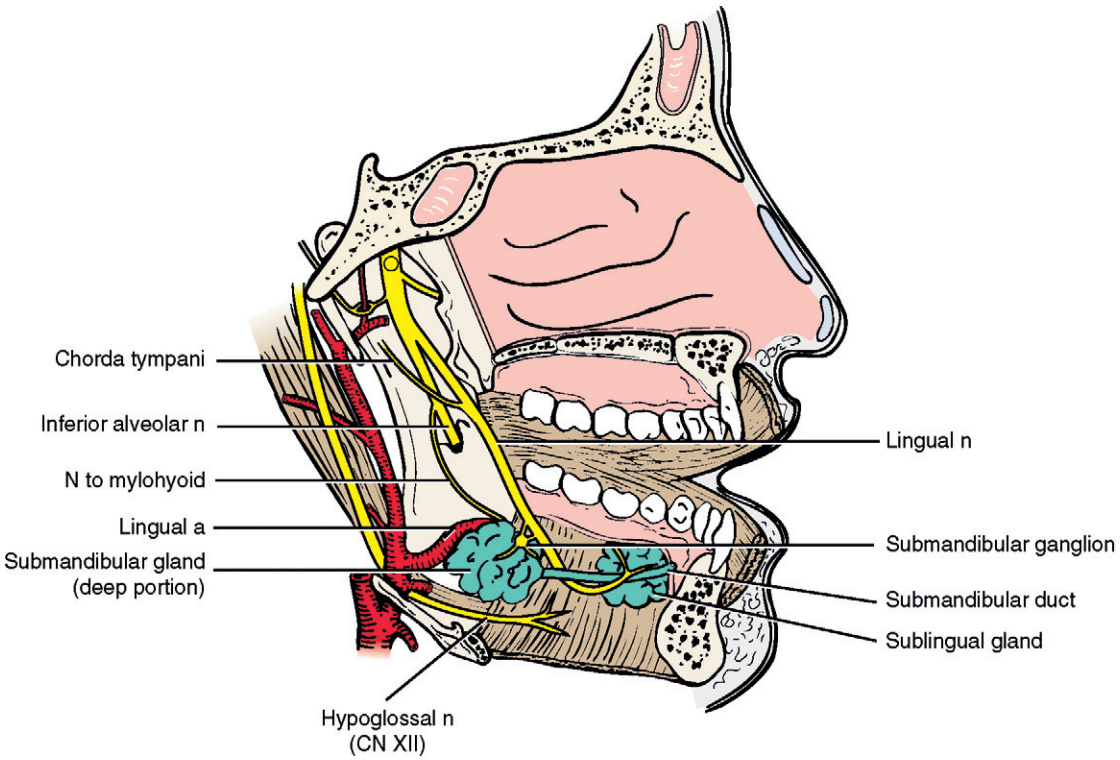


FIGURE 24-2 Anatomy of the floor of the mouth. Note the intimate relationship between the lingual nerve and submandibular duct. The submandibular duct originates from the portion of the gland that folds around the posterior border of the mylohyoid muscle. (From Liebgott B: The Anatomical basis of dentistry, ed 3, St. Louis, 2011, Mosby.)

windshield.[3] The largest documentation of salivary gland injury in the literature was reported by Morestin, who published his World War I experience with 62 parotid gland injuries.[14] Blunt or compressive injury to the salivary glands is rare, usually requires significant amount of force that results in injury to the facial bones and facial soft tissues, and typically is associated with motor vehicle accidents (MVAs), but also has been reported from sports-related injuries, strangulation, blows from fists or feet, and excessive manipulation.[15,16] Blunt trauma may result in the formation of a parenchymal contusion, hematoma, and sometimes a sialocele or a mucocele. This type of injury usually involves the parotid gland because of the protective nature of the mandible body for the submandibular and sublingual glands.[17] However, rupture of major salivary glands can occur after blunt force, even in the absence of a cutaneous defect or laceration.[15,18] The demographics of salivary gland injury are similar to those of other maxillofacial injuries in that the incidence is greater for men in the third decade of life.[1,3,19] In a collective review of 32 patients by Tachmes et al, 56.3% of ductal injuries were acute, whereas the remainder appeared several days following the injury with a sialocele, fistula, or both, often with associated superinfection.[2]

DIAGNOSIS OF SALIVARY GLAND INJURIES

The diagnosis of salivary gland injury starts with increased suspicion of a potential injury based on the location and mechanism of trauma. The history and physical examination often provide useful information in the workup of possible salivary gland injury. Questions should be directed toward the presence of any suspicious swelling or drainage from the wound that might represent saliva, as well as the effects of food if the patient is taking an oral diet. Any penetrating cheek injury has the potential to involve the parotid gland and duct, the facial nerve, or all these structures. Figure 24-3 demonstrates a useful anatomic classification of parotid duct injuries first described by Van Sickels.[7] Site A represents the most proximal portion of the duct, where it emerges from the substance of the parotid gland to the posterior edge of the masseter muscle. Site B is directly over the masseter muscle. In this location, the duct is relatively straight, superficial, and unprotected by any other overlying structure. Consequently, site B is the most common location of duct injury and the easiest to repair.[1] Lastly, site C represents the portion of the duct distal to the anterior border of the masseter muscle, which subsequently dives into the substance of the buccinator muscle to terminate in the mouth adjacent to the maxillary second molar.

Parotid duct injuries are not easily diagnosed and are often missed on initial examination.[1,19] In addition, other severe traumatic injuries are often associated with parotid duct injuries. Thus, delay in diagnosis of salivary injury is common and, if diagnosis is made, immediate repair is often postponed until the patient is stable.[2,3] When the patient's condition permits, the optimal sequence of treatment for suspected parotid gland ductal injury should include the following[20,21]:

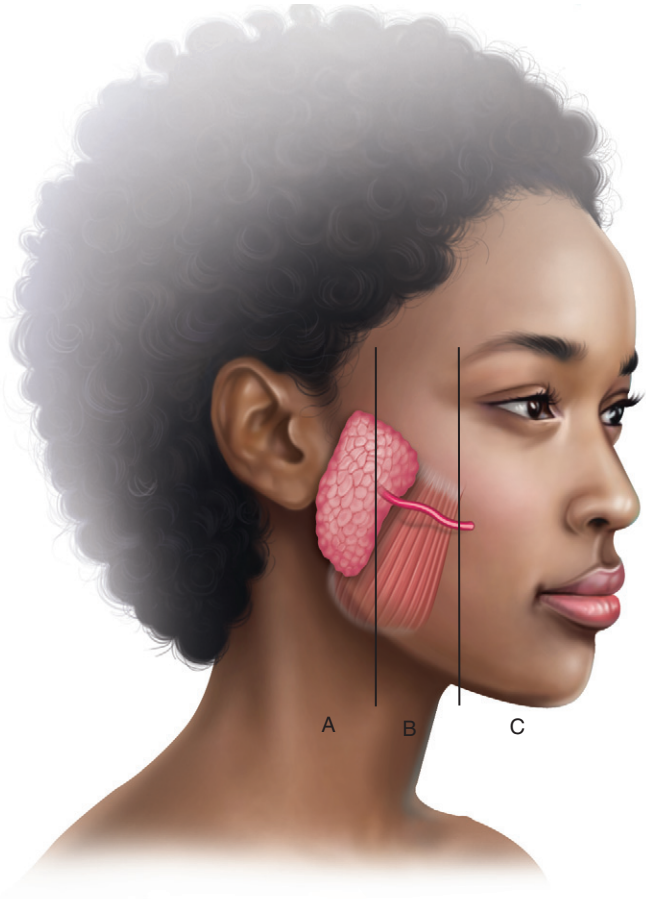


FIGURE 24-3 Anatomic classification of parotid duct injuries first described by Van Sickels and Alexander. The path of the duct is divided into three regions—A, B, and C. The most common site of injury and the easiest to repair is in region B over the anterior border of the masseter muscle.

- Detailed history and physical examination.
- Complete otologic examination when injury to the parotid gland is suspected.
- Palpation of the skeletal support of the face and the temporomandibular joints.
- Functional testing of each branch of the facial nerve (the administration of local anesthetic should be delayed until this examination is completed).
- Control of hemorrhage with pressure until adequate exploration of the wound under general anesthetic can be attempted; blind attempts to control bleeding should be avoided because the main blood vessels to the parotid gland lie deep to the facial nerve.
- In deeply penetrating injuries to the parotid gland, one must rule out injury to the carotid artery and a thorough neurologic examination must be undertaken. Repeat examination is indicated if the patient is intoxicated or unable to participate with the examiner.
- Emergent exploration of the wound if nerve or ductal injury is suspected.

Inspection of the injury and surrounding structures should be compared with the contralateral side for symmetry. After control of hemorrhage, a complete examination consists of attempting to identify collections of clear fluid in the depths of the wound. The sensitivity of this examination can be improved by pressing on the gland to express saliva into the field. When injuries are suspected with the submandibular gland, an assessment of tongue sensation and mobility may be helpful. Facial nerve function can be assessed with a nerve stimulator to help determine if the nerve is intact. Ductal injuries can best be delineated by direct inspection following cannulation of the duct with a lacrimal probe from its distal oral opening. Retracting the cheek wall in an outward direction, which straightens the sharp bend in the distal duct, can facilitate cannulation of Stensen's duct. Retrograde injection of methylene blue can be used to aid in the diagnosis of ductal injury when inspection and probing fail to locate the duct. The duct opening is located intraorally and cannulated with a small gauged angiocatheter. A small amount of methylene blue is injected retrograde while observing the wound. Areas of extravasation can then be explored for ductal injury. A small amount should be injected because an excessive amount of dye in the wound can make for difficult evaluation and identification of stained anatomic landmarks.[6,22,23] Saline, sterilized milk,[24] or propofol solution has been advocated to assess the integrity of the duct and avoid the staining problems associated with the use of methylene blue.

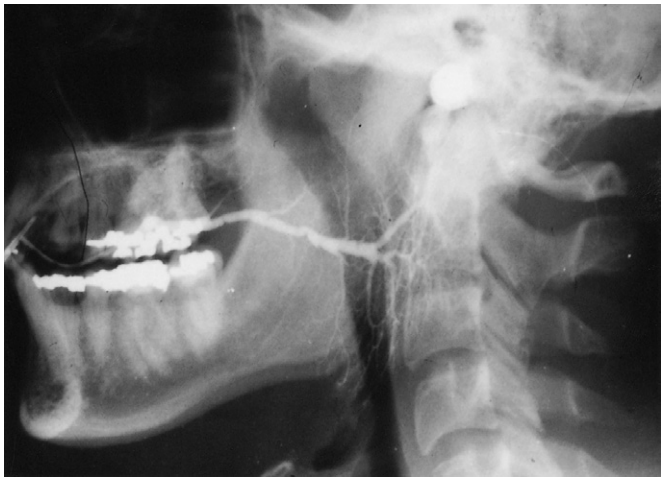


FIGURE 24-4 Lateral cephalometric image of a patient following a parotid sialogram. Note the presence of the catheter in the parotid duct orifice.

Radiographic imaging can been used to support the clinical suspicion of salivary gland and ductal injury. Magnetic resonance imaging (MRI) has been the standard technique for imaging soft tissue pathology, including infections, but is not the study of choice for salivary gland trauma because it is difficult to delineate the integrity of the ductal system. Sialography with a water-soluble contrast medium remains a highly sensitive test of ductal abnormalities, with or without (Fig. 24-4) computed tomography (CT) imaging.[13,17,25-27] Direct visualization of ductal filling under fluoroscopy can be useful, particularly using digital subtraction techniques. Cannulation of the parotid duct is normally straightforward, but access

to the submandibular duct, especially in the trauma patient, may be difficult and its use with possible sublingual gland injury is not possible. Several authors have argued that the study of sialography, although costly and time-consuming, is important because it can delineate the exact location of glandular and ductal injuries, which has significant prognostic implications.[1,3,28] Patients with facial trauma may also have concomitant injuries that prohibit transferring the patient to the radiology department for a lengthy study. Sialography is best used in cases that present some time after the traumatic event or in which ductal injury may have been overlooked.[23] Sialography is also useful in the postoperative period to evaluate the results of treatment. If cannulation of the duct is unsuccessful, salivary gland function may be quantified by time-activity curves with ^{99m}Tc-pertechnetate scintigraphy and may distinguish between the functioning, obstructed, and nonfunctional gland.[29] MRI sialography is an imaging technique using heavily T2-weighted sequences that is performed without the injection of a contrast medium and is considered a noninvasive, painless, and safe technique. Ductal injuries can be demonstrated by MRI sialography, which is dependent on stimulated ductal salivary flow as the contrast medium, and correlates well with or even improves on conventional sialography.[24,30]

TREATMENT OF SALIVARY GLAND INJURIES

Specific recommendations on the management of ductal injury remain controversial. There is a consensus in the literature that acute parotid injuries should be explored primarily and the injured structures repaired, if possible. The aims of primary repair of salivary gland injuries should be the reapproximation of anatomic structures and return of normal function. Nonsurgical management of parotid duct injuries has been supported by Lewis and Knottenbelt, who reported their experience with 19 patients with a confirmed parotid duct injury. Nine patients had an uncomplicated duct healing and the remaining patients developed a sialocele or fistula that eventually resolved without surgical management.[1]

MINOR SALIVARY GLAND INJURIES

Most minor salivary gland injuries occur during penetrating intraoral trauma. Appropriate treatment during the closure of intraoral lacerations includes the removal of any traumatized minor salivary glands within the wound. Complications of such intraoral or labial injuries may result in mucus retention phenomena (or mucocele; Fig. 24-5) caused by extravasation of fluid into the surrounding tissue from injury to the duct of a minor salivary gland. The treatment of this complication is accomplished by aggressive removal of the involved and surrounding minor salivary glands and the associated fluid-filled cavity, followed by primary closure of the mucosa.[9,13]

SUBMANDIBULAR GLAND INJURIES

Direct injury to the submandibular gland parenchyma is unusual because of the protective nature of the body of the mandible and position of the gland. Typical injuries include intraoral lacerations of the duct, penetrating trauma to the gland, trauma to the gland associated with fracture of the mandibular body, or direct blunt trauma with fracture of the gland (usually seen following MVAs). Complications may include salivary extravasation into tissue, an expanding neck mass or, less commonly, cutaneous fistula formation.[13,31] Treatment of parenchymal injuries usually consists of conservative management with pressure dressings and aspiration of any fluid collection. If significant damage to the gland is present or if conservative measures fail, excision of the gland is appropriate.[28] The most frequent management for intraoral lacerations of Wharton's duct is marsupialization at the site of injury (Fig. 24-6). This is the treatment of choice because primary repair of the duct is difficult and unnecessary and may result in secondary stricture formation.[9]

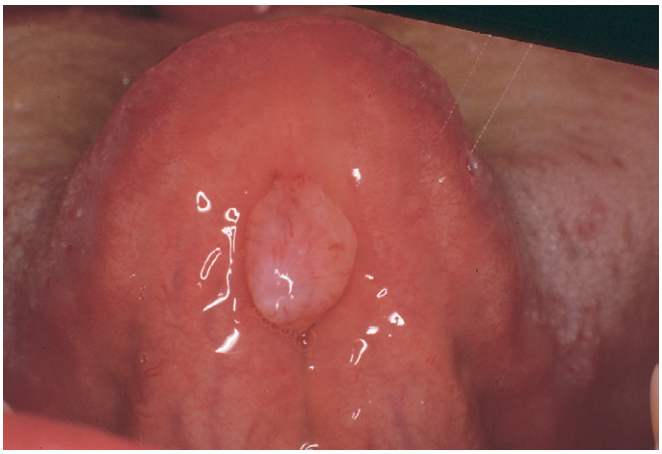


FIGURE 24-5 Atypical location of a mucocele (salivary extravasation phenomenon) on the ventral surface of the tongue. A mucocele can occur anywhere minor salivary glands are present. They commonly occur following injury to the minor salivary gland duct, which leads to extravasation of saliva into the surrounding tissue.

SUBLINGUAL GLAND INJURIES

Injury to the sublingual gland is most commonly a result of blunt penetrating trauma in the floor of the mouth or injury associated with fracture of the mandible in the area of the sublingual glands. Again, the incidence is low because of the protective nature of the mandible. Depending on the severity of injury, repair consists of primary closure of the wound or gland removal. The most notable complication of this injury is ranula formation with treatment being gland excision or marsupialization[11,12,32,33] (Fig. 24-7).

PAROTID GLAND INJURIES

Defining the extent of parotid gland injury is helpful for predicting the development of complications and duration of healing. Consequently, it is useful to divide injuries into those that involve only glandular tissue, those that involve only the duct, and those that involve both the gland and the duct. Van Sickels has recommended that injuries that occur over the glandular region be treated with closure of the capsule, those over the

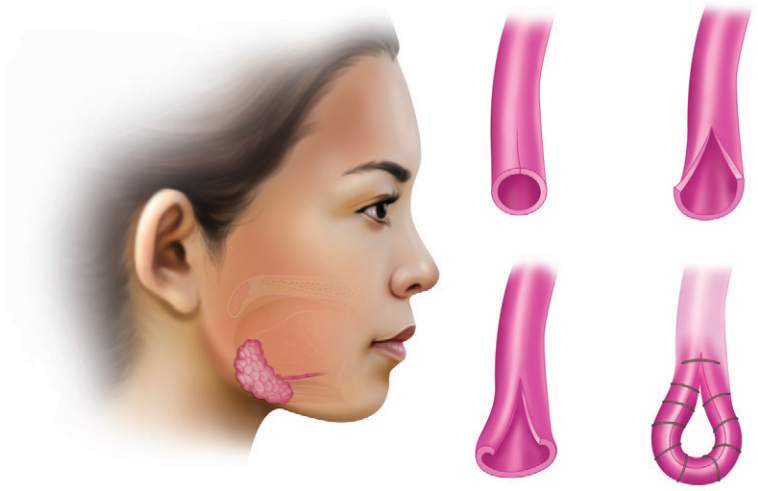


FIGURE 24-6 Marsupialization of the submandibular duct into the floor of the mouth in a more distal location following traumatic injury to the distal duct.

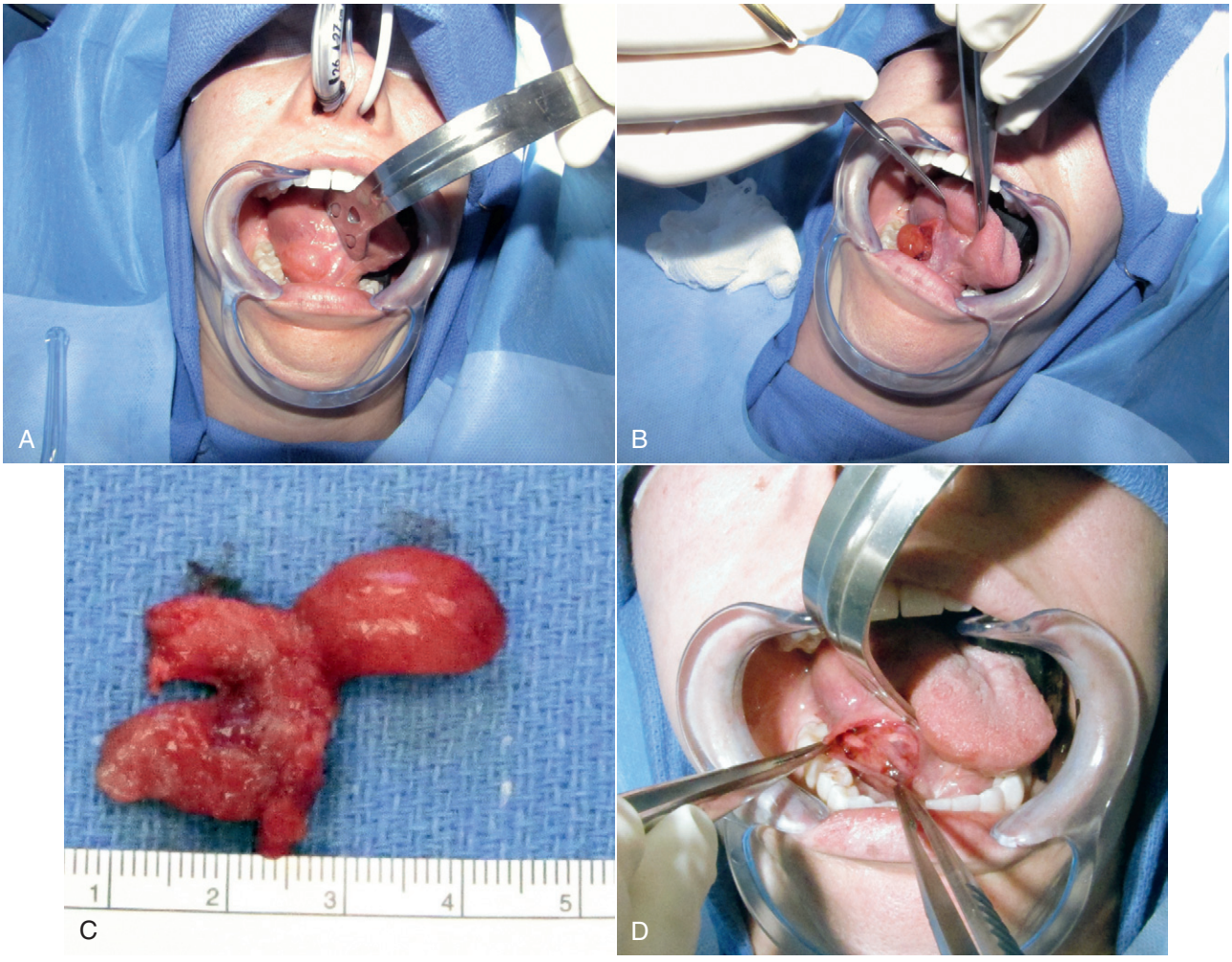


FIGURE 24-7 **A,** Ranula in the right side of the floor of mouth. This patient experienced penetrating injury to the floor of the mouth approximately 1 month before being seen by a physician. **B,** Initial incision with exposure of mucocele. **C,** Specimen containing sublingual gland and mucocele. **D,** Exposure of lingual nerve and submandibular duct after removal of sublingual gland and mucocele.

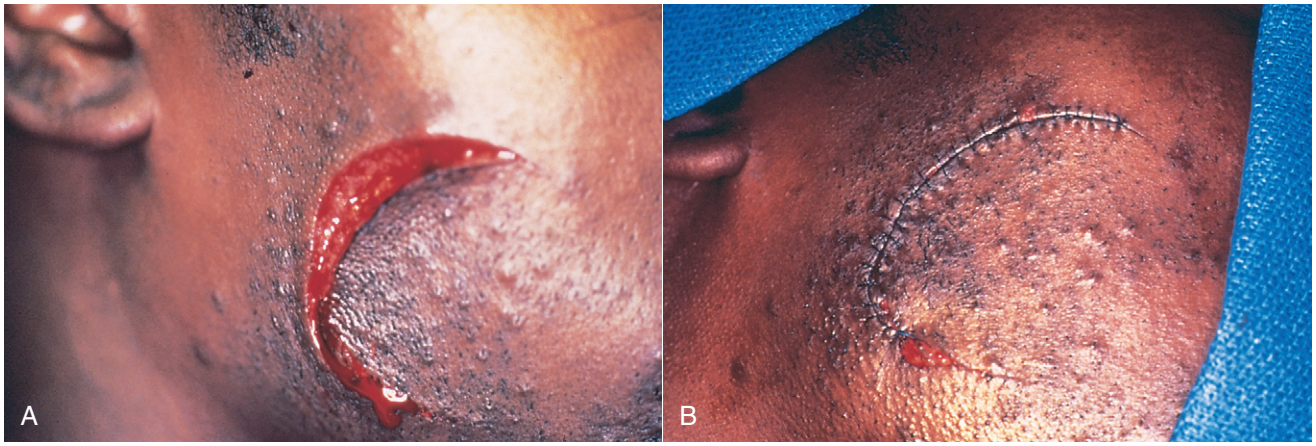


FIGURE 24-8 Injury to the parotid region involving only the glandular material. Note the circular form of the injury resulting from an assault with a broken bottle. The wound is closed after careful attention to a layered closure, paying particular attention to the gland capsule. An external pressure dressing will now be applied for 24 to 48 hours.

masseter muscle treated by direct repair of the duct, and those anterior to the masseter treated by repair of the duct or creation of an intraoral fistula.[7] Identifying the exact location of the ductal injury provides important prognostic information (see Fig. 24-3).[1,3]

Isolated Glandular Injury

Isolated glandular injury can be managed conservatively without any surgical intervention other than suturing. Thorough irrigation of the wound, débridement of necrotic tissue, and layered primary closure of lacerations should be carried out, with closure of the gland capsule being the most important. A pressure dressing often is applied to prevent a sialocele by attempting to direct the saliva back through the ductal system. Because of the potential for infection, these patients should be placed on antibiotics.

Primary management of glandular injury that includes a major interparotid duct also involves a layered closure of the wound (Fig. 24-8), application of pressure dressings, frequent recall with aspiration of any fluid accumulation, and techniques to reduce salivary flow (i.e., NPO and the administration of antisialagogues [e.g., propantheline, 30 mg PO every 6 hours]). Oral intake is restricted as Enfers[34] has demonstrated that resting parotid secretions are negligible and the major impetus for parotid salivary flow is gustatory and mechanical stimulation. Lesions confined to the parenchyma or minor ducts heal notably faster than glandular disruption that includes a major intraparotid duct. For glandular injuries that disrupt major intraparotid ducts, lack of oral stimulation has been shown to facilitate healing significantly.[3]

Ductal Injury

Acute trauma to the parotid gland rarely produces a complicated surgical problem unless Stensen's duct has been injured. When managing an injury to the parotid duct, many factors must be considered, including the degree and anatomic location of the ductal damage, any associated facial nerve injury, time interval between injury and presentation, and presence of complications, such as sialoceles or salivary fistulas. There are three methods generally used to manage duct lacerations. Three categories of treatment include primary repair of the duct with microsurgical anastomosis, diversion of salivary flow by creation of an intraoral fistula, and suppression of salivary gland function.[23]

Controversy exists in the literature concerning the acute management of known parotid duct injury. One side argues for exploration of the wound and primary closure of lacerations to Stensen's duct, stating that the complications of sialoceles, fistulas, and salivary cysts are great and preventable[6,22] and, given the advancements in microsurgical technique, good results may be achieved.[35,36] Others believe that the hazards and inconvenience involved in immediate repair of parotid ductal injuries are not justified because complications such as sialocele and cutaneous fistula eventually heal with conservative management.[1,3]

The most important factors in the primary surgical management of parotid duct injury are the extent of injury and anatomic location of the lesion. In a study by Parekh et al, conservative management of parotid injuries resulted in an average healing time of 10 days for lesions in regions A and B (see Fig. 24-3) when only partial ductal injury was present. However, complete transection of the duct, whether in region B or C, resulted in a delayed healing time as long as 21.5 days with conservative management.[3]

Localization of the site of ductal injury can provide important prognostic data and help determine the method of treatment. Lesions appearing in the proximal portion of the duct or at the glandular-duct interface have fewer associated complications and a more expedient healing phase and therefore can be treated conservatively. In contrast, Parekh et al[3] have indicated that disruption of the glandular-duct junction has the worst prognosis. Lewis and Knottenbelt noted in their study that only 2 of 9 patients with site A injuries (glandular injury only) have complications following conservative therapy, whereas 8 of 10 patients with injuries in sites B

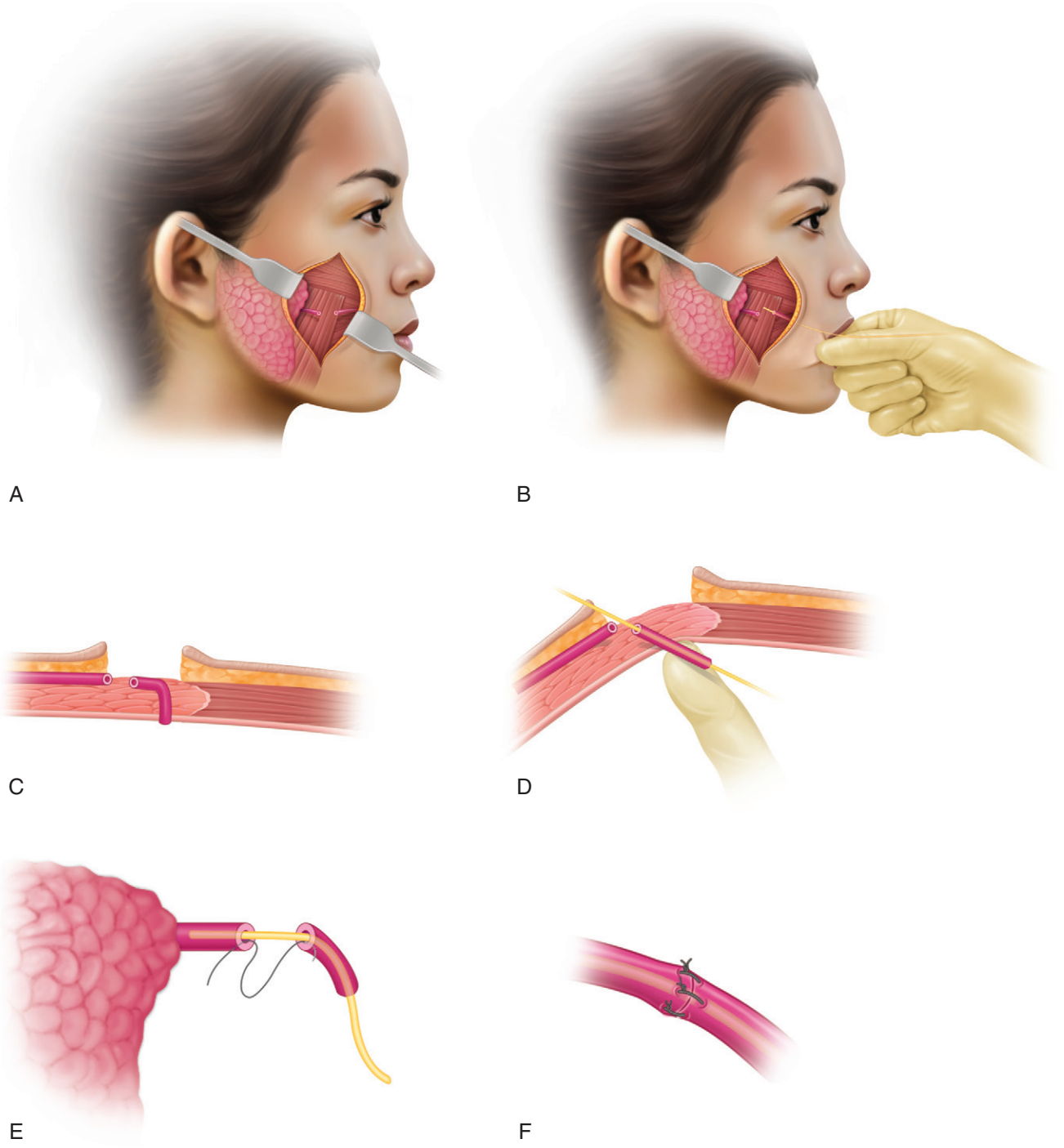


FIGURE 24-9 Steps for primary repair of parotid duct laceration. **A,** The parotid duct severed at the anterior border of the masseter muscle. **B,** A catheter is passed through the oral orifice of Stensen's duct into the wound. **C,** The sharp bend in the duct just before it penetrates into the oral cavity. **D,** Correctly placed intraoral finger pressure can straighten the duct to facilitate the threading of the catheter. **E,** Direct anastomosis of the cut ends of the duct using the catheter as a splint. **F,** Appearance of the duct after primary anastomosis with at least three sutures.

and C have complications.[1] Overall, in their study, 47% of ductal injuries healed without complication, 37% had salivary fistulas, 21% had sialoceles, and 1 in 19 had an infection that resolved following antibiotic administration. However, all complications healed within 3 weeks without surgical intervention. These figures give credence to the conservative management of partial parotid duct injuries, especially if they are located in regions A and B.

Most authors believe that the acute appearance of a parotid duct transection warrants immediate repair with stenting.[1,7,20,28,37]

Primary parotid duct repair should be sequenced as follows (Fig. 24-9):

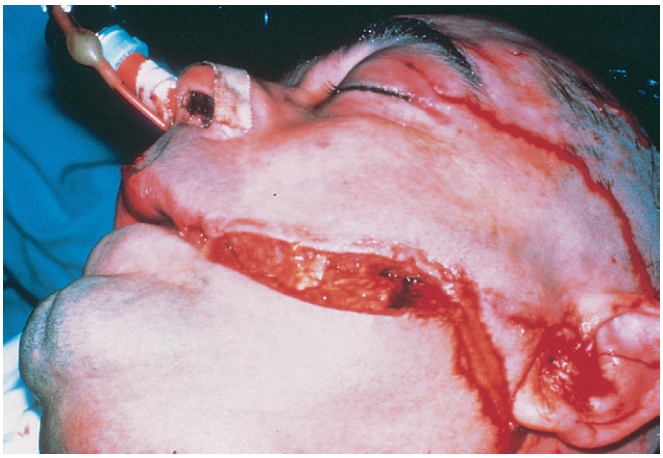


FIGURE 24-10 Severe through-and-through laceration of the left cheek from the commissure of the mouth almost to the ear following a motor vehicle accident.

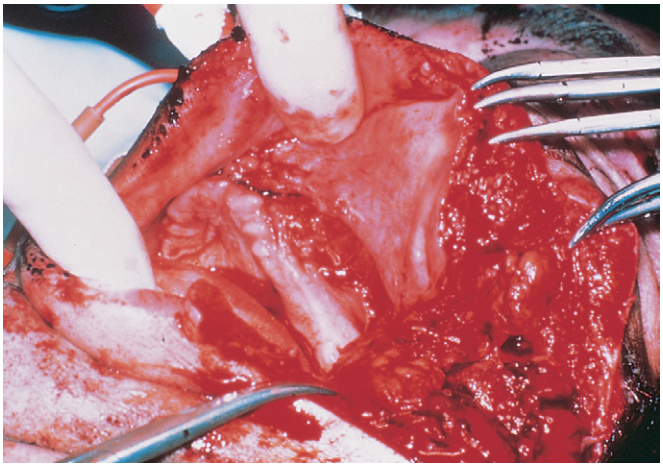


FIGURE 24-11 Visualization of the entire laceration after careful débridement and control of hemostasis.

1. Functional testing of the facial nerve should be performed before administration of local anesthetic. The wound should be gently irrigated and débrided. Bleeding should be controlled with pressure or surgical ties; avoid electrocautery and blind clamping (Figs. 24-10 and 24-11).
2. The cheek wall should be retracted outward. This maneuver straightens the angled course of the distal parotid duct and permits easy passage of the catheter.
3. A 16-gauge Intracath stent is passed through the distal portion of the duct from the oral orifice. If this is impossible, and if the cut end of the distal portion of the duct is visible in the wound, cannulation of the duct can be performed through the wound.
4. Identification of the proximal portion of the duct is facilitated by massaging the gland to express saliva. The proximal segment of the duct is then cannulated.
5. The severed ends of the duct are closed over the Intracath, with at least three small nylon or Prolene sutures (Fig. 24-12).
6. The wound is closed in layers, with careful closure of the gland capsule (Fig. 24-13). The catheter is sutured to the buccal mucosa (Fig. 24-14), and an external pressure dressing is applied for 24 to 48 hours. Antisialagogues (propantheline, 30 mg PO every 6 hours) can be considered. The catheter is left in place for 10 to 14 days. During this time, the patient should receive antibiotic therapy.[7,22,28]

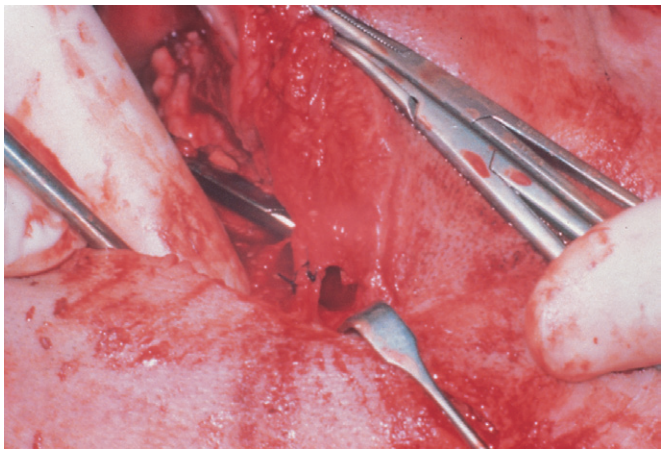


FIGURE 24-12 Repair of the parotid duct with three small Prolene sutures following identification and cannulation of the proximal and distal portions of the lacerated duct.



FIGURE 24-13 Final layered closure of the wound before application of an external pressure dressing.

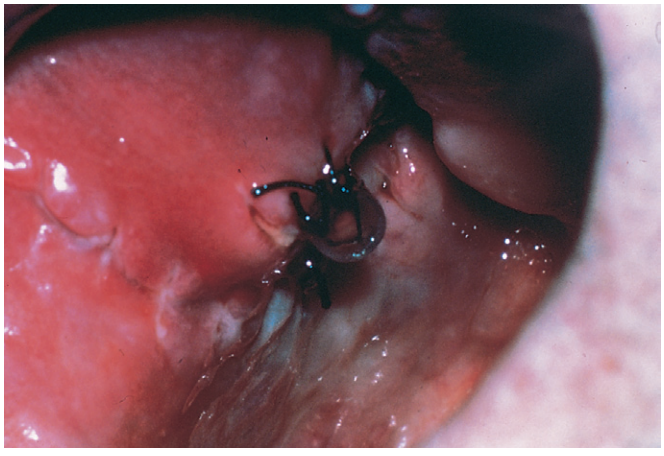


FIGURE 24-14 End of the catheter sutured in place intraorally in the left cheek. This catheter will be left in place for 10 to 14 days.

7. A ductal defect larger than 1 cm may prevent direct anastomosis, and an autogenous graft may be indicated.[35] Interpositional vein grafting in the repair of Stensen's duct has been reported.[38]

Unfortunately, no long-term study has looked at ductal patency or the incidence of parotid duct atrophy after primary repair. If ductal stricture occurs, balloon dilation can be performed to improve ductal patency.[39] Postoperative sialography or evaluation by sialoendoscopy may be used to examine the intraductal anatomy after trauma to the gland and/or duct.[35,40]

If there has been extensive tissue loss and direct anastomosis is not possible, the proximal parotid duct should be dissected free from the surrounding tissue to allow for surgical manipulation and diversion of the duct into the oral cavity may be possible. The proximal portion of the duct is brought through the buccinator muscle and oral mucosa into the oral cavity. If the duct enters the oral cavity without tension, a catheter within the duct may not be necessary and the duct can be sutured to the intraoral mucosa. Although use of a stent is not mandatory, a stent may help reduce the incidence of ductal stenosis consequent to developing granulation tissue and edema while maintaining patency of the duct and permitting salivary flow.[4] If the proximal duct cannot be advanced to enter into the oral cavity tension-free, a catheter can be inserted into the proximal portion of the duct and secured to the oral mucosa to allow for secondary epithelization around the catheter and formation of an oral fistulous tract for drainage of the salivary flow.

With severe avulsion of the soft tissues or during wound exploration, repair of the ductal injury is judged to be impossible; the proximal portion of the duct should be ligated. The resulting salivary obstruction leads to increased intraluminal pressure, which when transmitted to the parotid parenchyma results in decreased perfusion and eventual physiologic death of the gland.[6,14,28] Although effective, this process can result in temporary parotid inflammation and swelling, causing a significant amount of pain until the atrophy process is complete. A pressure dressing should be placed over the parotid gland to minimize swelling and further promote gland atrophy; antisialogogue medications should be used to decrease salivary gland function.[4,23] Concerns of facial asymmetry following glandular atrophy have been demonstrated not to be a long-term complication following parotid duct ligation.[41]

Some authors advocate a conservative approach and believe that the biggest risk of delayed treatment of ductal injuries is the development of a sialocele or salivary fistula, which in most cases can be managed conservatively.[1,3,28] The advantages of conservative management of acute ductal injuries include safety and ease of treatment, less risk of facial nerve injury, no necessity for special surgical skills, and the ability to manage these patients on an outpatient basis.

Another factor essential for determining the method of treatment for parotid gland injuries is the presence of known facial nerve injury. The facial nerve can be injured by transection or compression, or from a crushing injury. In a compression or crushing injury, the functional deficit may be without anatomic disruption. Electrical nerve testing may be useful to evaluate nerve potential and, in most cases, nonpenetrating injuries to the facial nerve can be managed conservatively with good results.[42] Most authorities agree that the diagnosis of transected facial nerve injury warrants immediate exploration and repair of the nerve and duct in an operating room setting using microsurgical repair.[1,2,5] Landau and Stewart[28] have reported a 20% incidence of facial nerve injury associated with isolated glandular injuries and a 55% incidence with parotid duct injuries. When a facial nerve injury occurs, the buccal branches are usually injured. This is because the buccal branches run parallel and just superior to the duct and occasionally may even cross lateral to the duct (Fig. 24-15). Severance of this nerve branch causes drooping of the upper lip. To determine whether a severed nerve branch should be repaired, the examiner

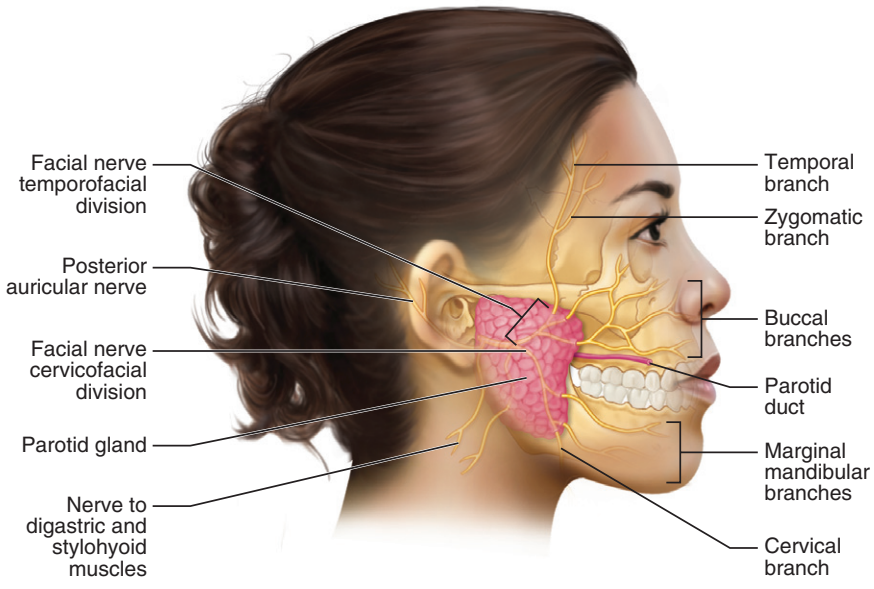


FIGURE 24-15 The relationship of the parotid gland and duct to the branches of the facial nerve. Note the close approximation of the buccal branches of the facial nerve to the parotid duct.

should compare the site of injury with a line from the outer canthus of the eye to the gonial notch of the mandible. Branches distal to this line do not require repair; however, careful attention to closure of the wound in layers is necessary to facilitate reestablishment of nerve function. For nerve injuries proximal to this line, isolation of the lacerated nerve stumps and primary repair are indicated, including possible nerve graft from the greater auricular or sural nerves.

The detection of parotid duct injuries is frequently delayed. Delay allows for granulation tissue formation as well as scarring. Finding and repairing a duct in such a presentation may be difficult or impossible and more conservative treatment modalities may be required. This is primarily because of the high incidence of severe musculoskeletal injuries associated with parotid injuries. Management of these injuries takes precedence, often precluding definitive diagnosis and treatment of parotid injuries in a timely fashion. Furthermore a definitive diagnosis of ductal injury is often not made in the trauma bay, because an accurate diagnosis often requires patient compliance and time-consuming procedures, such as local wound exploration and duct cannulation,[28] cannulation of the duct with injection of methylene blue dye,[1] or the use of sialograms with or without associated CT studies.[3,25,43] Delay in the treatment of parotid injuries may be beneficial in certain patients in whom notable hemorrhage or tissue distortion is present. In these patients, local wound exploration may be associated with a high risk of morbidity (e.g., facial nerve injury).[44]

MANAGEMENT OF SALIVARY GLAND INJURY COMPLICATIONS

The most common complications of major salivary gland injuries are a result of salivary fluid extravasation into the tissue leading to a sialocele, a nonepithelialized fluid-filled cavity (through continuous salivary secretion without proper drainage),[35] and finally to a salivary cyst, an epithelialized cavity filled with salivary secretions. Occasionally, a cutaneous salivary fistula may be formed from the extraoral flow of saliva from the injury site.[6] An external parotid fistula can develop within the first week after injury. In contract, a sialocele tends to develop more slowly, presenting 8 to 14 days after parotid gland trauma.[3] The duration of the complications and the prognosis are again directly attributed to the location and degree of the parotid injury, (i.e., parenchymal versus ductal injury and partial versus complete ductal transection).[44] Ideally, one would confirm the presence of salivary fluid in a sample of aspirate by determining the amylase level (>1000 U/liter), because these lesions may be mistaken for hematoma or infection.

Since secondary repair of the duct is difficult because of the presence of granulation and scar tissue,[6] the major aim of therapy is to resolve the complication with glandular atrophy. In the past, methods such as antisialagogue administration alone, radiation therapy, parasympathetic denervation (tympanic neurectomy), and superficial or total parotidectomy were used to accomplish this goal. Clinical studies have investigated the use of botulinum toxin at the site of a sialocele or fistula.[45,46]

The use of antisialagogues alone to treat salivary injuries has had mixed reviews in the literature. Antisialagogue therapy is a useful adjunct when attempting to decrease salivary flow. Their use may aid in the closure of salivary fistulas; however, some authors report success only in injuries not involving major intraparotid ducts. Their use alone may be questioned and the side effects associated with their use can be problematic.

In the past, radiation therapy was used to induce fibrosis and atrophy of the gland. Approximately 1800 rad is necessary to accomplish glandular atrophy. However, there is a high failure rate in patients with ductal injuries, and more than 6 weeks is required for gland atrophy to occur following radiation therapy.[3] The potential to induce malignancy (thyroid cancer, skin cancer, or salivary gland neoplasms) has led to general abandonment of this treatment method.[6]

Tympanic neurectomy (transtympanic sectioning of Jacobson's nerve) has a high failure rate. It has been shown that the use of this technique may not speed recovery and bears a high potential morbidity of delayed healing because glandular atrophy may take as long as 6 months to occur.[44,47,48]

Superficial or total parotidectomy has been advocated as a treatment method. However, the morbidity of this procedure, especially in the presence of granulation tissue and fibrosis, must be emphasized. This technique has also largely been abandoned because approximately 75% of these patients experience some postoperative facial palsy.[44]

The initial treatment for complications such as sialocele and cutaneous fistula is conservative management, including pressure dressings, repeated aspirations, limited intake by mouth, and the use of antisialagogues to decrease salivary flow (Fig. 24-16).[19,48] Landau and Stewart have reported uniform success using this conservative treatment regimen in a prospective study of 14 patients with sialoceles or cutaneous fistulas.[28] All the sialoceles and fistulas resolved within 14 days.[49,50] Other authors have shown similar results.[1,3,44,47] One study went further in the delineation of conservative therapy by placing patients on a strict NPO regimen while administering parenteral nutrition until the salivary lesion was completely healed. The investigators found a significant difference in the rate of healing when those on NPO status were compared with those allowed oral intake 5 days following repair. This method of treatment is successful because the parotid gland is kept in a resting state in which basal salivary flow rates are small. In addition to the preceding, Epker and Burnette have advocated the insertion of a catheter via Stensen's duct into the sialocele, with intent to direct drainage intraorally.[6] Studies of the treatment of selected salivary gland disorders with local injections of botulinum toxin type A (Botox, Allergan, Irvine, Calif) has been encouraging. Botulinum toxin is an exotoxin of *Clostridium botulinum*. It prevents the release of acetylcholine at the cholinergic synapse in salivary glands.[51] Ellies et al have reported on ultrasound-guided administration of Botox into salivary glands in the treatment of hypersalivation.[52] Significant suppression of

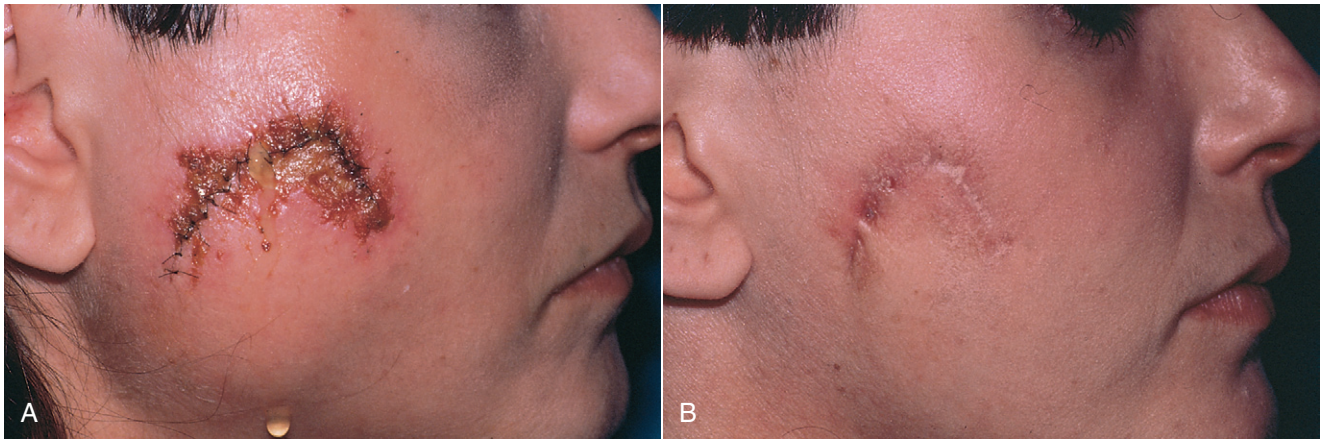


FIGURE 24-16 **A,** Patient with a parotid fistula appearing approximately 10 days following conservative repair of parotid gland and duct laceration. Note the drop of saliva from the fistula. **B,** Same patient 3 weeks after initiation of conservative treatment consisting of an external pressure dressing, administration of oral antisialagogues, repeated aspirations, and restriction of oral intake. Note healing of the fistula.

salivary gland function induced by botulinum toxin begins 1 to 2 weeks after injection and can last for 3 to 6 months. This allows time for healing of the salivary gland function and resumption of normal salivary flow.[53] The success of conservative treatment of the complications of salivary injury suggests that surgical intervention should be considered only after conservative therapy fails.

Various surgical techniques are described in the literature for refractory salivary fistulas and sialoceles, ranging from reexploration of the wound to identify and repair the parotid duct injury to techniques designed to redirect the salivary secretions intraorally (create an intraoral fistula).[19,50,54,55] With the exception of the last procedure, all these techniques run the risk of complications, ranging from delayed or poor healing to facial nerve injury.

Demetriades[19] has described the technique of internalization of salivary flow for the treatment of sialoceles and fistulas. The procedure is performed with the patient under local or general anesthetic using an incision incorporating the traumatic scar to gain access to the lesion. Next, blunt dissection is used to enter the floor of the cystic cavity and then to perforate through the masseter muscle into the oral cavity. A Jacques catheter (size 6) is positioned so that one end is within the cavity and the other exits through the oral mucosa where it is sutured in place. When a true fistula is present, the epithelium-lined tract must be excised. The skin wound is closed primarily in layers and a pressure dressing is applied. The intraoral drain is typically left in place for 10 days, oral antibiotics are prescribed, and the patient receives a liquid diet. Invariably, the secretions decrease in the first 5 to 7 days. The presumed cause of healing is scar formation within the cavity and duct, which results in glandular atrophy. The advantages of this procedure over the options described earlier are the ease of performance, minimal surgical risk, and minor cost.[19,22] A better prognosis for the sialocele or salivary fistula is expected when the injury is a result of glandular disruption rather than a duct-only injury.[35]

Frey's syndrome (gustatory sweating) is thought to represent the aberrant regeneration of postganglionic parasympathetic parotid nerve fibers, denervated during the surgical procedure or the traumatic injury, as they grow astray into the overlying skin and innervate the sweat glands. Diagnosis can be made based on a history of gustatory sweating or by means of the starch iodine test, in which the affected skin is painted with iodine and dusted with starch. The patient is then given a sialogogue and the resultant sweating is manifest by the appearance of black spots as the sweat interacts with the iodine. Treatment options for Frey's syndrome include the topical application of 2% glycopyrrolate cream, intradermal injection of botulinum toxin, and tympanic neurectomy.[4]

In general, salivary gland injuries are an infrequent occurrence. When they occur, they may be associated with other severe injuries, which can lead to delayed diagnosis and treatment. It may be advisable to obtain a sialogram if the patient is stable at the time, because the information derived from this diagnostic test can be important for determining the location and extent of injury and predicting the outcome of conservative treatment. If acute injury to the duct is detected, particularly when associated with a facial nerve injury, the wound should be explored and primary repair of the duct and facial nerve accomplished. If diagnosis is delayed (usually with the appearance of a sialocele or fistula), conservative management should be used. In most cases, this approach is successful. If conservative management fails, surgical intervention should be used to promote healing.

IATROGENIC INJURIES TO THE SALIVARY GLAND

Surgical approaches to the facial skeleton, particularly approaches to the mandible, may bring the surgeon into close proximity to the major salivary glands. Iatrogenic

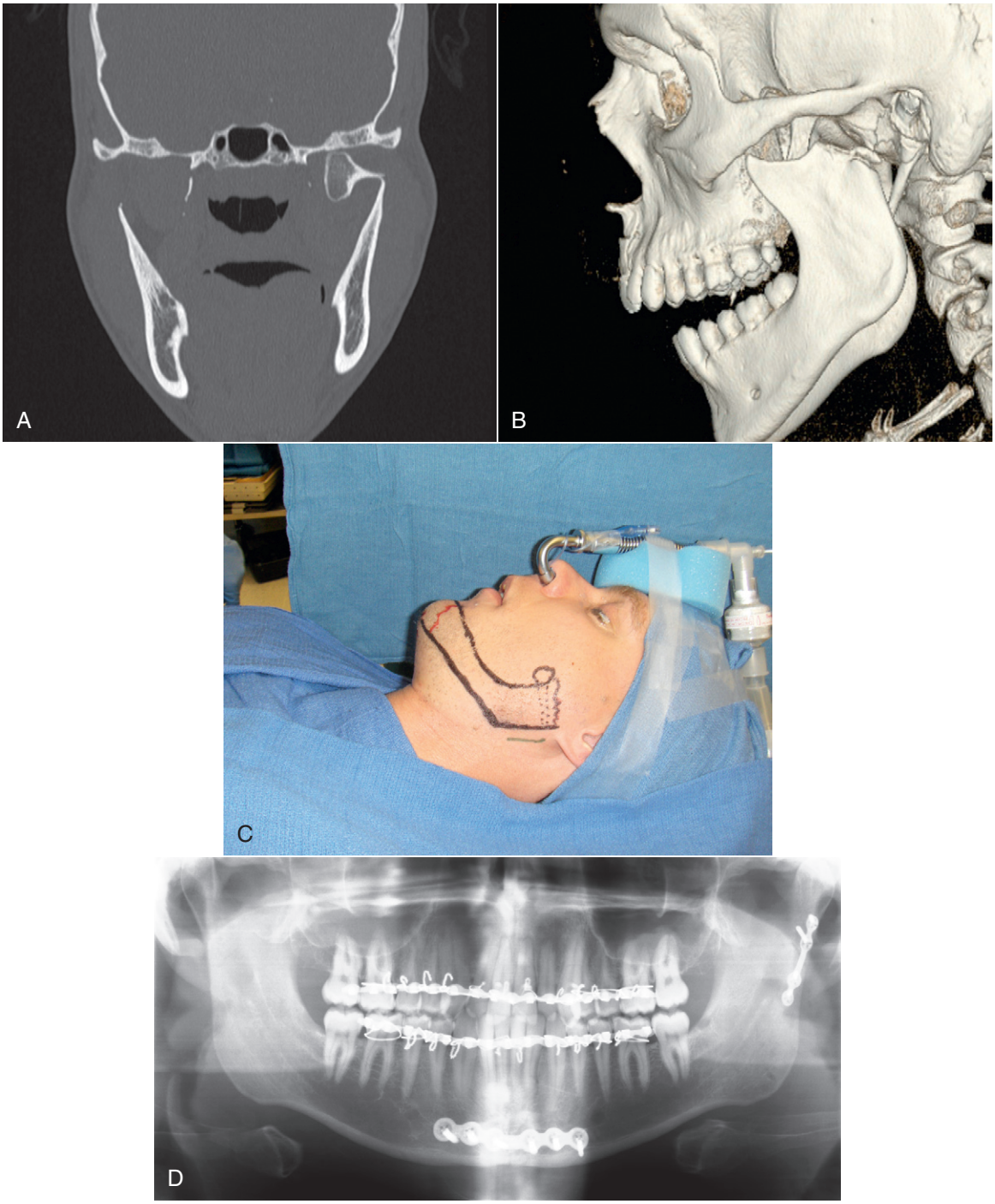


FIGURE 24-17 **A, B,** CT scans demonstrating left subcondylar fracture. **C,** Retromandibular inciscion. **D,** Postoperative panoramic radiograph showing open reduction and internal fixation (ORIF) of mandibular fractures with anatomic reduction.

Continued



FIGURE 24-17, cont'd **E,** Development of a parotid sialocele 2 weeks after surgery. **F,** Aspiration of sialocele. **G,** Resolution 1 month after aspiration and pressure dressing.

injury to the glands during these procedures is a rare possibility. It is important that the surgeon be able to recognize and manage these injuries should they occur.

PAROTID SIALOCELE AND FISTULA

The retromandibular incision initially described by Hinds[56] requires blunt dissection through the body of the parotid gland. This incision provides excellent exposure for the open reduction and fixation of mandibular subcondylar fractures. Dissection through the parotid gland carries a slight risk of the development of parotid sialocele or fistula.[57] The reported incidence of parotid sialocele and fistula associated with the retromandibular incision is between 2% and 8%. Careful dissection through the gland and tight closure of the parotid capsule have been advocated as preventive measures to avoid this complication.[58]

Diagnosis and management of a postoperative parotid sialocele are often straightforward. Clinical presentation would include a posterior neck swelling in the region of the prior surgery. The differential diagnosis would include infection versus sialocele. The patient with an infection will often present with fever and leukocytosis, which are absent in the sialocele. Aspiration of the sialocele reveals a straw-colored fluid. Laboratory tests would confirm a high level of amylase. Management involves decompression with needle aspiration of the saliva, placement of a pressure dressing, and starting the patient on an antisialagogue. Conservative management should continue until resolution of the swelling. This may take 4 to 5 weeks and may involve repeated needle decompressions[59] (Fig. 24-17).

SUBMANDIBULAR GLAND MUCOCELE

The submandibular or Risdon approach provides access to the mandibular body, angle, and ramus. During this dissection, the operator may encounter the submandibular salivary gland deep to the platysma muscle while progressing through the superficial layer of deep cervical fascia. During this dissection, the gland's capsule may be violated and the body of the gland injured. Submandibular gland mucocele is a rare complication.[60]

The diagnosis of a submandibular gland mucocele is largely clinical. The differential diagnosis may include

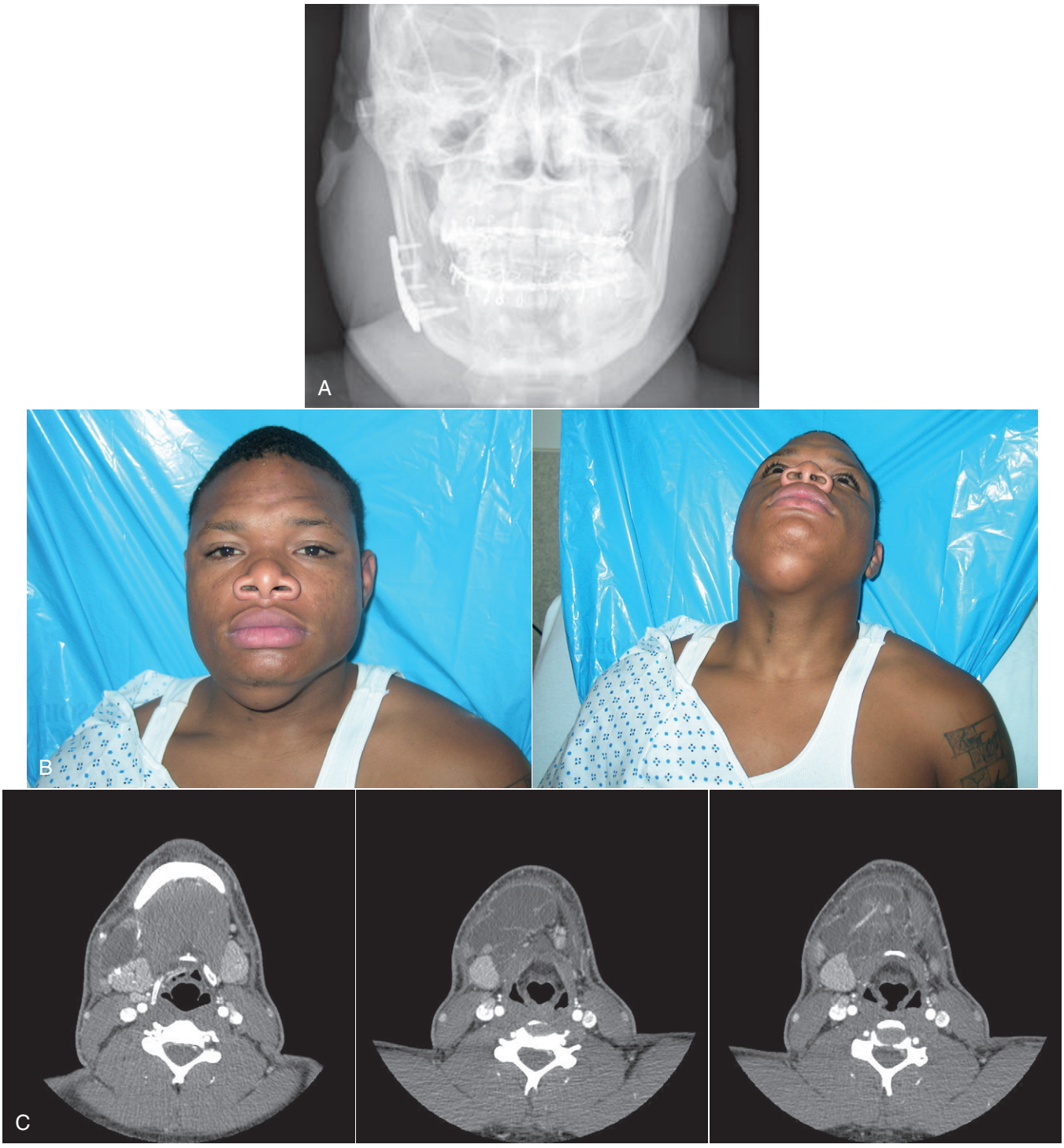
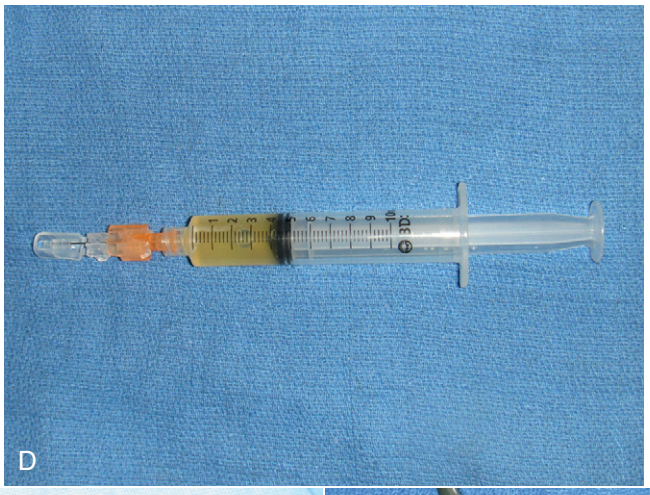


FIGURE 24-18 **A,** 3 years post-ORIF right mandibular angle fracture. **B,** Submandibular swelling 3 years after ORIF mandibular angle fracture. **C,** Axial CT scan showing fluid-filled collection in the submandibular space.

Continued

infection versus mucocele but could also include other neck swellings. Aspiration of straw-colored fluid positive for amylase coupled with a CT scan of the neck confirms the diagnosis. The treatment of a submandibular gland mucocele involves the removal of the mucocele along with the associated gland. In some cases, it may prove difficult to distinguish if the mucocele is associated with the sublingual or submandibular gland. In these cases, it is recommended that both glands be removed as definitive treatment[61] (Fig. 24-18).



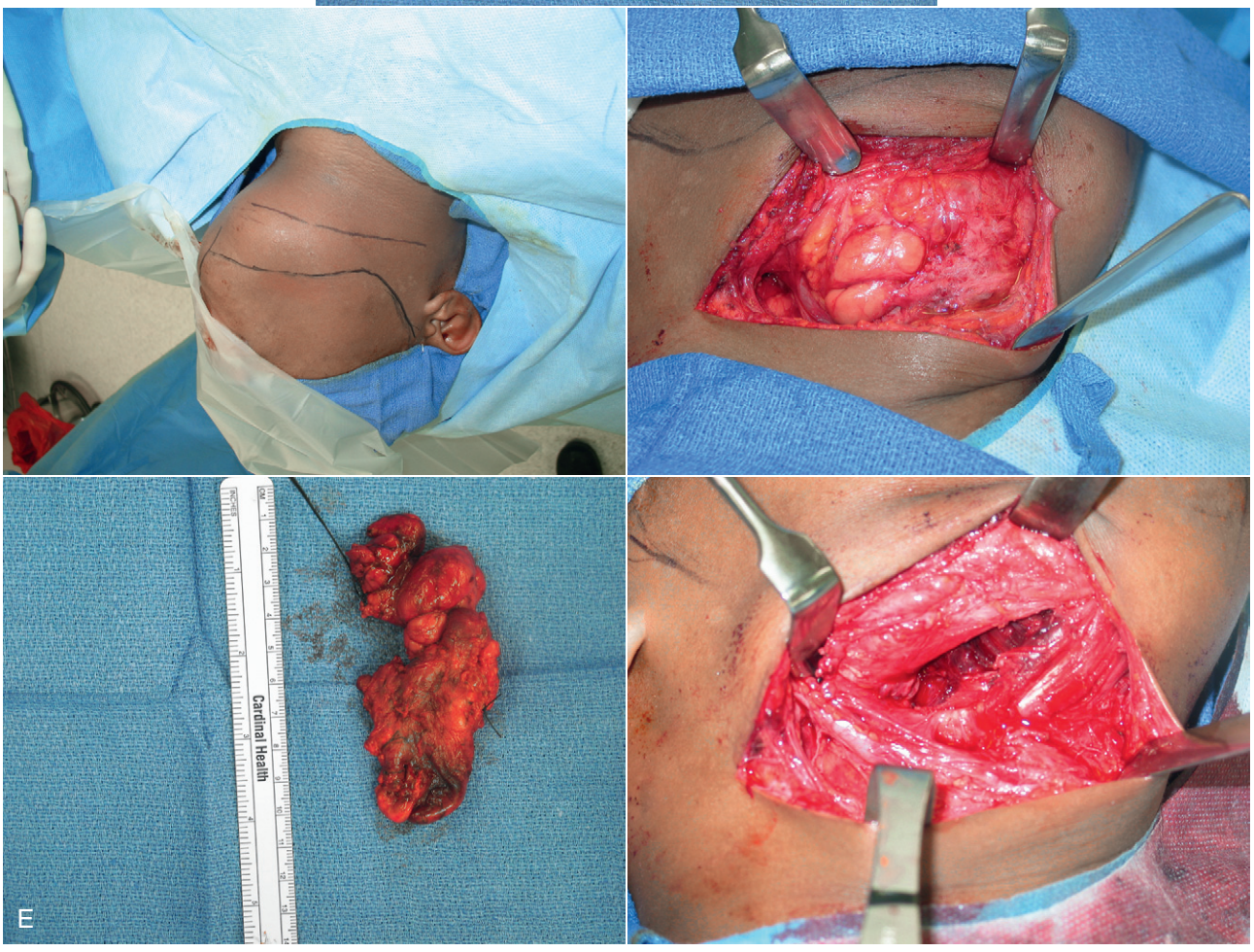


FIGURE 24-18, cont'd **D,** Aspiration of submandibular fluid collection (saliva). **E,** Excision of submandibular gland and mucocele.

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CHAPTER

25

Traumatic Injuries of the Trigeminal Nerve

Michael Miloro | Antonia Kolokythas

OUTLINE

Trigeminal Nerve Anatomy
Macroanatomy
Microanatomy
Trigeminal Nerve Imaging
Preoperative Risk Assessment
Postinjury Assessment
Postinjury Functional Assessment
Classification of Trigeminal Nerve Injuries
Seddon Classification System
Sunderland Classification System
Axonal and Cellular Response to Injury
Mechanisms of Injury to the Trigeminal Nerve
Local Anesthetic Injection
Third Molar Surgery
Dental Implant Surgery
Maxillofacial Trauma
Orthognathic Surgery
Maxillofacial Pathology
Endodontic and Chemical Injury
Clinical Neurosensory Testing
Microneurosurgery
Indications
Microneurosurgery for Trigeminal Nerve Injuries
Outcomes of Trigeminal Nerve Injury and Surgical Intervention

The face and perioral regions are among the body regions with the highest density of peripheral nerve receptors; this feature makes neurologic disturbances less tolerable in the head and neck than in other body parts. The trigeminal nerve is the largest cranial nerve, with three main divisions including the ophthalmic, maxillary, and mandibular branches. The trigeminal nerve is a mixed motor (special efferent and proprioceptive) and sensory (general afferent) peripheral nerve innervating the face, mouth, head, and neck regions. Injury to the peripheral branches of the trigeminal nerve can be devastating because of the effects on speech, deglutition, swallowing, mastication, and taste, as well as the impact on social interactions. Unfortunately, these injuries can occur rather easily from a traumatic event or from several commonly performed procedures in the maxillofacial region. Throughout their careers, oral and maxillofacial surgeons often encounter patients with trigeminal nerve injuries and should be proficient in diagnosing and managing these cases appropriately, because successful outcomes from nerve injuries are critically time-sensitive. In this chapter, the following topics are presented: an overview of the anatomy of the peripheral branches of the trigeminal nerve, relevant to traumatic injuries and surgical procedures in the maxillofacial region; an overview of nerve microanatomy to assist the reader's understanding of the events following injury and the nerve's regenerative properties; the available imaging modalities for risk assessment of nerve injury from commonly performed maxillofacial procedures, along with the possible mechanisms of injury from these procedures; neurosensory testing modalities available for appropriate evaluation and classification of nerve injuries that are paramount for appropriate management of these cases; and a description of microsurgery for trigeminal nerve repair and the expected outcomes.

The use of universally accepted terminology is crucial when discussing nerve injuries. It is imperative that the appropriate terms be used to describe and document each case for communication among clinicians, determination of management strategies, and medicolegal reasons (Table 25-1). In addition, a detailed description, including nerve mapping with illustrations of the region with associated sensory changes, should be documented for each patient and used to quantify the initial presentation and, most importantly, to follow the progress of the area of involvement (Fig. 25-1).

TRIGEMINAL NERVE ANATOMY

MACROANATOMY

Inferior Alveolar Nerve

The largest of the three branches of mandibular division of the trigeminal nerve (V3) descends between the medial pterygoid muscle and medial ramus and enters the mandible at the mandibular foramen (Fig. 25-2). At the lingula (the bony landmark for the mandibular foramina along the medial surface of the ramus), the inferior alveolar nerve (IAN) has the largest diameter, 2.4 ± 0.4 mm. At the mandibular foramen, the IAN diameter is 2.0 ± 1.1 mm. The mental nerve exits the mandible at the mental foramen and the course of the bony canal appears to be fairly predictable, based on cadaveric and conventional imaging studies.[1-3] Proximally within the ramus, angle, and body region of the mandible,

TABLE 25-1 Select Terminology of Sensory Alterations

Term	Meaning
Allodynia	Pain because of a nonpainful stimulus
Analgesia	Absence of pain from a painful stimulation
Anesthesia	Absence of any sensation from a painful or nonpainful stimulation
Dysesthesia	Abnormal sensation, spontaneous or evoked, that is unpleasant
Hyperalgesia	Abnormal response to a painful stimulus
Hyperesthesia	Increased sensitivity to stimulation, excludes special senses
Hyperpathia	Increased reaction to a (repetitive) stimulus.
Hypoalgesia	Diminished pain in response to a painful stimulus
Hypoesthesia	Decreased sensitivity to stimulation, excludes special senses
Neuralgia	Pain in the distribution of a nerve or nerves
Paresthesia	An abnormal sensation, spontaneous or evoked, that is not unpleasant

between the mandibular foramen, where the IAN enters the mandible to the second premolar region, the IAN follows an S-shaped course. This is where the nerve is most lateral in the third molar region and closest to the buccal cortex. It then approaches the lingual plate in the first molar region. Anteriorly, between the premolars, the IAN returns toward the buccal cortex prior to exiting the mandible through the mental foramen.

According to Cutright et al, the mental foramen was found to be apical to the second premolar in approximately 51% of cases; its distance from the midline was slightly different between whites and blacks, averaging 2.7 and 3.2 cm, respectively. It was noted that in whites the foramen is located between the premolars, whereas in blacks it is located distal to the second molar. In addition, the group noted small but significant differences between males and females for the three foramina (mental, infraorbital, and supraorbital).[4] In the sagittal plane, the course of the IAN in the mandible begins superiorly and lingually at the lingula, approximately 10 mm from the sigmoid notch, and it reaches its lowest point at the first molar to second premolar region, with the distance from the inferior border of the mandible of 7.5 ± 1.0 mm, where it splits into the incisive and mental nerve branches. The incisive dental branch continues



FIGURE 25-1 Facial photographs with the areas of involvement marked are useful for monitoring progression.

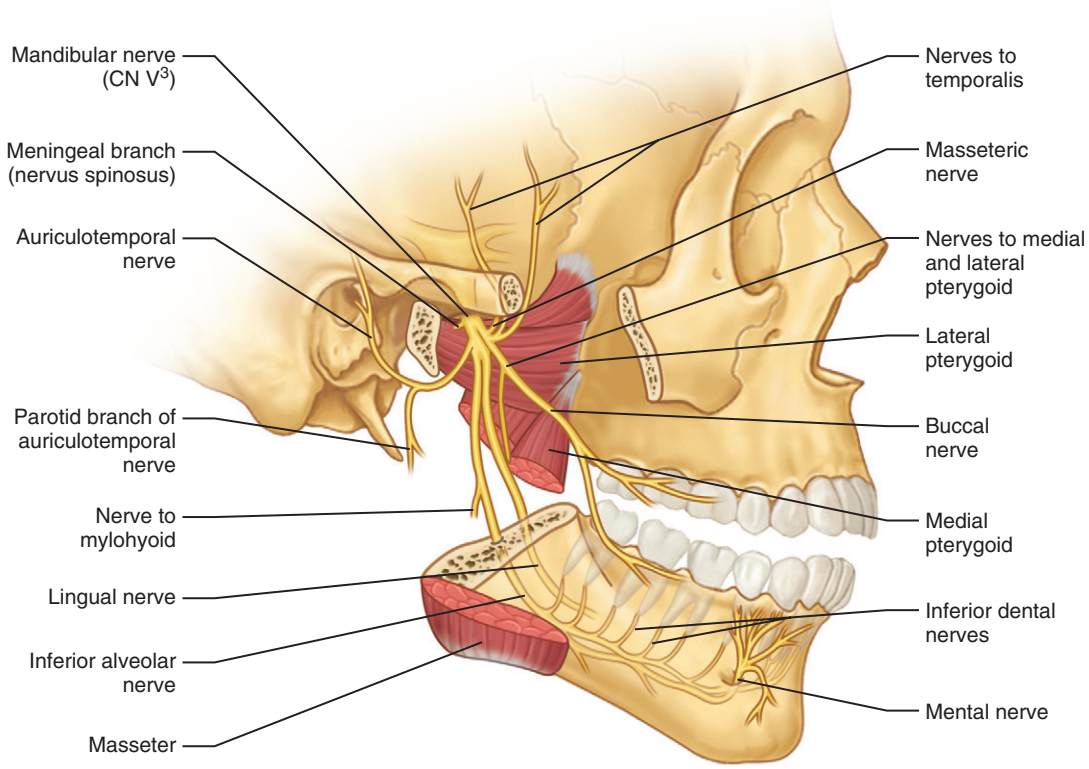


FIGURE 25-2 Regional anatomy demonstrating the peripheral trigeminal nerve system.

forward toward the incisors and the mental nerve ascends and loops backward for a variable distance (3 to 7 mm) to exit at the mental foramina through the buccal cortex.[5,6]

In their study of the mandibular canal, de Oliviera et al analyzed the bidimensional and tridimensional positions of the canal via multiplanar computer tomography (CT) morphometric analysis and provided useful average distances of the canal to the buccal and lingual cortices, inferior border, and alveolar ridge. The locations of the mandible for the measurements were selected to represent areas of commonly performed maxillofacial procedures associated with high risk for nerve injury, specifically extraction of third molars, sagittal split osteotomy, and dental implant placement. They did not find significant differences between the right and left sides, whereas in edentulous patients the typical values were lower in females than males and in older (from 51 to 75 years) than younger individuals (25 to 50 years).[7]

The mental nerve, the continuation of the IAN after it exits the mental foramen has two branches (range, one to four branches), with the largest being the labial branch running on the orbicularis oris muscle fibers. A smaller mucosal branch runs posteriorly on the buccinator muscle fibers. The labial branch innervates the lip and chin mucosa and overlying skin, but it may reenter the mandible to innervate the mandibular incisors and even cross the midline.[8] The mucosal branch innervates the buccal mucosa and gingiva of the mandible as far posteriorly as the first and second molar, where cross-innervation with the buccal nerve may occur. Throughout its course in the mandible, the IAN provides branches for innervation of the mandibular teeth.[9] Hu et al,[10] in a study of the branching pattern of the mental nerve, identified four branches (angular, medial inferior labial, lateral inferior labial, and mental) and five branching patterns. They believed that this information on the branching could help the clinician to predict the area of sensory disturbance after injury of the mental nerve more accurately, depending on the cause and location.

Lingual Nerve

The lingual nerve (LN) branches from the posterior division of the third division of the trigeminal nerve, medial to the lateral pterygoid muscle. It is located medial to the IAN and supplies sensory and special sensory function to the anterior two thirds of the ipsilateral tongue, floor of the mouth, and lingual mandibular gingiva. The LN is joined by the chorda tympani as it passes through the pterygotympanic fissure, which provides taste (anterior two thirds of the tongue) and carries special visceral afferents (preganglionic parasympathetic fibers from the facial) to the submandibular and sublingual glands. Kim et al,[11] in their study of the topographic relationship of the LN and IAN at the infratemporal fossa and paralingual space in 32 cadaveric hemisectioned heads, noted four patterns of furcation between the two nerves in relationship to the sigmoid notch and lingula (Table 25-2). In addition, the group measured the distance of the bifurcation to stable bony landmarks, including the foramen ovale and hamulus. The bifurcation was identified to occur on average 14.3 mm (range, 7.8 to 24.1mm) inferior to the foramen and 16.5 mm superior to the tip of the hamulus (range, 4.9 to 24.3 mm). This variation in the bifurcation patterns between the IAN and LN, as well as the collateral nerve branches at the retromolar region identified in 81.2% of the cases in their study, are

thought to contribute to the reported failure rate of mandibular anesthesia.[12]

The LN then passes into the pterygomandibular space between the ramus and medial pterygoid muscle, where it is surrounded by mesoneurial fat throughout its course. It enters the oral cavity over the superior pharyngeal constrictor, styloglossus, and mylohyoid muscles at the anterior border of the medial pterygoid muscle. As the nerve approaches the third molar region, it courses from a more lateral to a more medial position following the oblique flaring of the ramus. The exact location of the LN at the third molar region has been studied extensively, with some controversial findings among cadaveric studies, clinical observation during third molar surgery, and high-resolution magnetic resonance imaging (MRI) with regard to its proximity to the mandible.[13-20] Table 25-3 summarizes the reported distances and relationship of the nerve to the mandible at the third molar region. In this location, the nerve has a diameter from 2.0 to 5.0 mm and may be round, oval, kidney-bean shaped, elliptical, or ribbon-like in cross section, with a predominance of one shape versus another varying among studies.[11,18,20]

TABLE 25-2 Types of Furcation Of Inferior Alveolar Nerve And Lingual Nerve

Type of Furcation Between IAN and LN	Level of Furcation Between Sigmoid Notch and Lingula	No. per Type
Type I	Separation occurs above notch and inferior to otic ganglion	65.6% (21/32)
Type II	Separation occurs below notch and at superior half of distance between notch and lingula	28% (8/32)
Type III	Separation occurs below notch and at inferior half of distance between notch and lingula	3.1% (1/32)
Type IV	Separation occurs in a plexiform pattern	6.3% (2/32)

Kim SY, Hu KS, Chung IH, et al: Topographic anatomy of the lingual nerve. Surg Radiol Anat 26:128–135, 2004.

At the second molar region, the LN crosses Wharton's duct from a lateral to medial direction, traversing below the duct and hypoglossal nerve in the submandibular triangle. The nerve in this location gives off the preganglionic parasympathetic fibers from the facial nerve to the submandibular ganglion. These fibers are transected during submandibular gland excision so that the gland can be released from the nerve. It has been reported that the risk of LN injury during submandibular gland excision or sialodochoplasty of Wharton's duct ranges from 1.4% to 4.8%, and the nerve is in danger from intraoral and extraoral approaches.[21-24]

After looping under Wharton's duct, the LN passes upward onto the genioglossus muscle and enters the oral tongue muscular substance. With the exception of one or two large branches that proceed towards the tip of the tongue, all other branches to the tongue and adjacent lingual mucosa and gingiva are of small diameter (generally not >1 to 2 mm). The larger branches provide the greatest density of innervation to the tip and medial portions of the anterior two thirds of the tongue, compared with the posterolateral, dorsal, and ventral portions; this difference is developmentally driven.[25]

Buccal Nerve

The buccal nerve (BN) is a branch of the mandibular division of the trigeminal nerve that carries sensory fibers to the lower buccal, gingival, buccal sulcus, and cheek mucosa and may contribute to the cutaneous supply of the cheek. The nerve travels between the two portions of the lateral pterygoid muscle, beneath or through the lower part of the temporalis muscle deep to the mandible, and then crosses over the external oblique ridge at the anterior border of the masseter muscle and merges with the same branch of the facial nerve. This is where the nerve crosses over the external oblique ridge that places it at risk for injury, most commonly from third molar surgery. A rare variation that supports the IAN as the origin of the BN has been described by Turner, Singh, and Jablonski et al, who identified the nerve exiting the mandible through a small foramen at the retromolar fossa.[26-29] As the nerve reaches the surface of the buccinator muscle, it divides into a superior division that supplies the muscle and an inferior division that provides the sensory innervation of the mucosa, as noted earlier.

TABLE 25-3 Distances and Relationship of Lingual Nerve to Mandible at Third Molar Region

Study	No. of Nerves	MEASUREMENT (MM)		% of LN Above Alveolar Crest	% of LN in Contact With Lingual Plate
		Vertical (mm)	Horizontal (mm)		
Kiesselbach and Chamberlain	34 cadaveric and 256 in vivo nerves	2.28 ± 1.96	0.58 ± 0.9	17.6	62
Pogrel et al	40 cadaveric nerves	8.32 ± 4.05	3.45 ± 1.48	15	0
Behnia et al	669 cadaveric nerves	3.01 ±0.42	2.06 ±1.10	14.05	23.27
Holzle and Wollfe	68 cadaveric nerves	7.83 ± 1.65	0.86 ± 1.00	8.82	57.4
Karakas et al	21 cadaveric nerves	9.56 ± 5.28	4.19 ± 1.99	4.7	0
Miloro et al	20 in vivo nerves (evaluated by HR-MRI)	2.75 ± 1.00	2.53 ± 0.67	10	25

Hendy et al,[28] in their study of the course and relationships of the BN in 20 cadaveric specimens, noted the following:

- The nerve was found to have four to eight major branches, with a mean number of three branches where it crosses the external oblique ridge.
- The nerve trunk was found to cross the ridge as close as 3 mm from its deepest concavity of the external oblique ridge in 70% of specimens(14 of 20).
- In the remaining cases, the distance ranged between 7 to 12 mm below that point.

The authors postulated that the nerve is at risk from incisions in this region, even if these are placed as far away as 12 mm from the external oblique ridge. The variation of the branching patterns of the nerve, in addition to the high mechanosensory threshold noted on the buccal mucosa, may result in a small area of damage or in the injury not being noticeable by the patient.[30,31]

Infraorbital Nerve

The infraorbital nerve (ION) is a major branch and is the termination of the second division of the trigeminal nerve that emerges on the face via the infraorbital foramen. The foramen is on average 2.7 cm from the facial midline and 6 to 8 mm from the inferior orbital rim. In males, the foramen is larger than in females, with some differences in the distances from the midline and infraorbital rim identified in anatomic studies.[4,32] In the study by Kazkayasi et al, a single foramen was found in 90% of cases, two canals in 5% and three canals in the remaining 5%. The shape of the canal was found to be oval in 30%, round in 40%, and semilunar in 30% of cases.[33] The ION enters the orbit via the inferior orbital fissure and transverses the orbital floor in the infraorbital canal (IOC) for 22 mm. The canal may not be complete because the cortical thickness of its roof ranges from 0.1 to 6.0 mm; thus, the nerve may be exposed, without a discrete superior bony wall. The combination of a groove and canal is found in 50% of cases and the remainder demonstrate a complete canal.[33] Racial and gender variations have been reported in the location and shape of the foramen and in the length of the canal.[32,34,35] After it exits the infraorbital foramen, four branches of the ION have been identified and correspond to the areas of the midface, nose, upper lip, and lower lid innervated by the nerve[36] The ION is at risk for injury during approaches for reconstruction of the orbitozygomaticomaxillary complex following traumatic events and during maxillary orthognathic surgery. Orthognathic-related injuries are typically transient and reversible, but sensory changes from entrapment of the nerve encountered in midface trauma may cause more problematic, long-term neurosensory changes.

MICROANATOMY

The microanatomy of all peripheral nerves is the same, with sensory and motor nerves containing myelinated and unmyelinated fibers in a ratio of 1:4. The difference between the two types of fibers is the number of Schwann cells that surround each fiber with nodes of Ranvier that permit rapid saltatory nerve conduction (Fig. 25-3). In

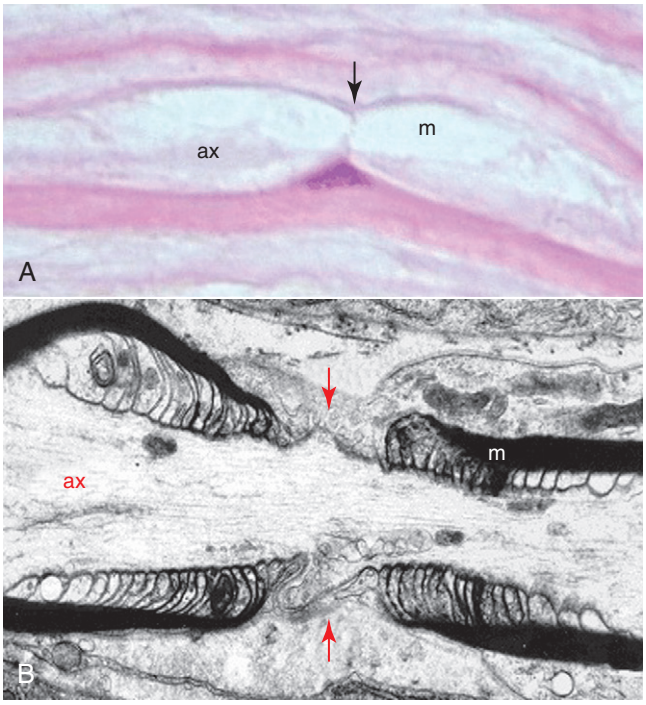


FIGURE 25-3 Hematoxylin and eosin **(A)** and silver stain **(B)** demonstrating the organization of a peripheral myelinated nerve in longitudinal section in the area of a node of Ranvier *(arrow). Ax,* axon; *m,* myelin.

the myelinated fiber, the ratio of nerve axons and Schwann cells is 1:1, whereas in the unmyelinated fiber the Schwann cell envelops several axons. The membrane, or basal lamina, created by the Schwann cell as it wraps around the axon and runs the entire length of the axon, is called a band of Büngner (laminar myelin sheath) and is crucial for the process of nerve regeneration. Although myelin may be destroyed during nerve injury, Schwann cells survive and play a major supportive role in nerve recovery and repair. The length of the axon surrounded by a single Schwann cell is known as the internode and the small area (0.3 to 2.0 μm) between the internodes, where the axon is not myelinated, is known as a node of Ranvier. In each node of Ranvier, certain ions diffuse, which causes nerve depolarization and repolarization and allows for the conduction of nerve impulses along the nerve fiber. This event—of the conduction occurring only between the nodes of Ranvier (saltatory conduction)—is responsible for the rapid impulse transmission noted in myelinated nerve fibers (up to 150 m/sec) compared with nonmyelinated nerve fibers (2 to 2.5 m/sec). Typically, unmyelinated axons are also smaller in diameter, with diameters ranging from 0.15 to 2.0 μm.

Collagen provides the framework that surrounds the nerve and creates the structural architecture within the nerve (Fig. 25-4). The endoneurium is the first organization of fine collagen fibers around each nerve fiber axon. In the pre–electron microscopy era, this organization was known as the Plenk-Laidlaw sheath or sheath of Key and Retzius.[37,38] Several bundles of endoneurial groups, known as fascicles, are grouped together and surrounded by a second layer of collagen fibers (and mesothelial

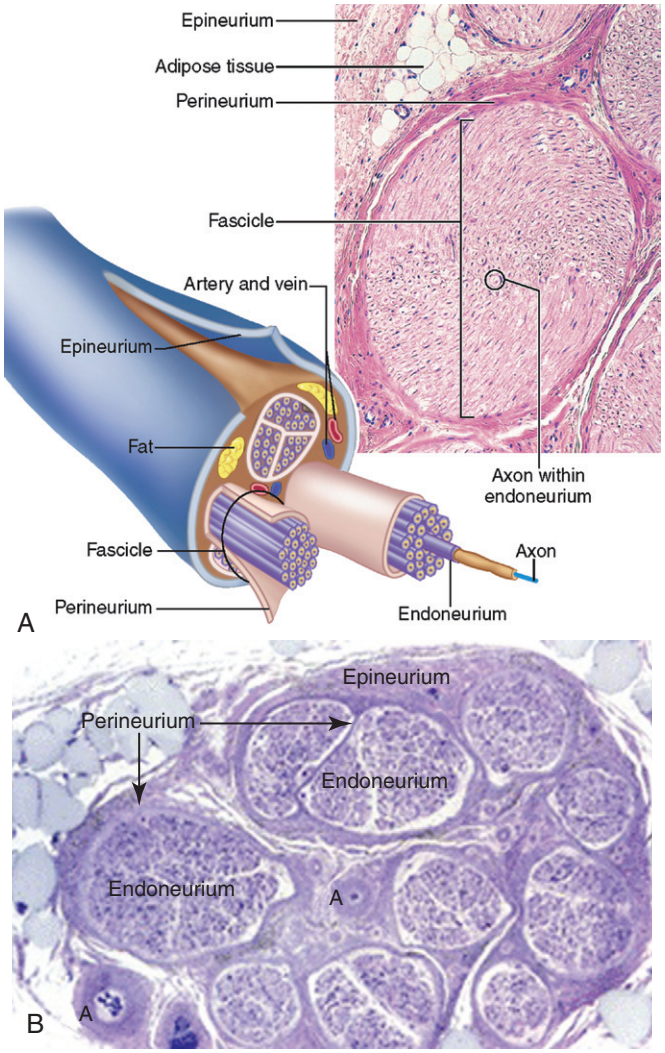


FIGURE 25-4 **A,** Schematic representation of nerve organization. **B,** Low-power cross section of polyfascicular nerve showing the organization of the nerve, with individual nerve fibers surrounded by endoneurium, fascicles surrounded by perineurium, and the investing epineurium. The mesoneurium is visible surrounding the entire nerve sheath at the periphery, with adipose tissue *(upper left). A*, perineurial and endoneurial blood vessels. (**A** from Patton K, Thibodeau G: *Anatomy and physiology*, ed 7, St. Louis, 2010, Mosby.)

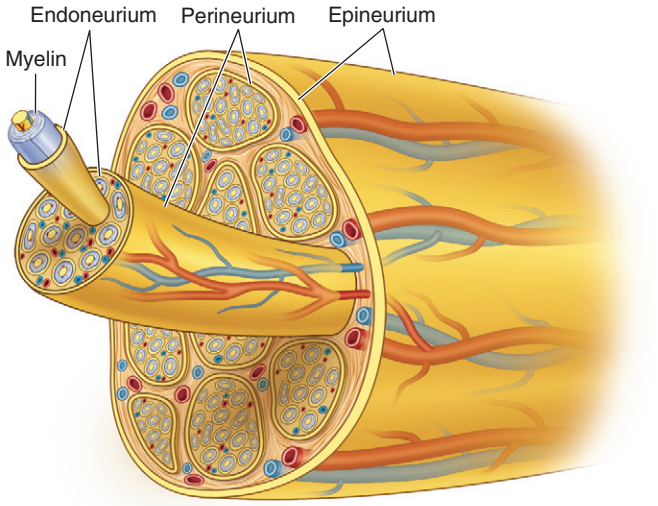


FIGURE 25-5 Schematic representation of vascular supply between the layers of the connective tissue surrounding the axon (red, arteries; blue, veins).

TABLE 25-4 Number of Fascicles and Diameter of Branches of Trigeminal Nerve

Nerve	No. of Fascicles (mean)	Cross-Sectional Area (mean)
IAN	9.4 to 21 ± 7.05 (third molar region)	1.64 ± 0.27 mm^2 (third molar region)
LN	8.5 to 10 ± 4.0 (third molar region)	1.87 ± 0.38 mm^2 (third molar region)
MN	10.9 to 12 ± 3.58	1.18 ± 0.27 mm^2
ION	5 to 6 (in the IOC)	2 mm^2 (in the IOC) 0.4 mm^2 (after it exits the IOF)

cells), called the perineurium. The outer layer of connective tissue supporting the nerve, along with some elastic fibers, creates the external or extrafascicular epineurium. Another layer of epineurium that invests the fascicles is known as internal epineurium. The epineurium makes up 50% of the cross-sectional area of the nerve and provides some protection against compression. The final layer of connective tissue that is continuous with the epineurium and surrounding tissue bed and is responsible for allowing some freedom of movement of the nerve is known as the mesoneurium, or adventitia of the nerve.[37]

Peripheral nerves are often distinguished based on their fascicular pattern, which may be of one of the following three patterns—monofascicular pattern with only one large fascicle (e.g., the chorda tympani), oligofascicular pattern, containing only some (two to ten) fascicles (e.g., the labial branch of the mental nerve), and polyfascicular, with more than 10 fascicles of different sizes (e.g., IAN, lingual, and buccal nerves). The mean number of fascicles and cross-sectional area for the trigeminal nerve branches decrease from the proximal to the distal nerve.[39-42] Table 25-4 provides the reported observations regarding fascicular number and diameter for each branch of the trigeminal nerve.

A rich interconnecting vascular network of extrinsic and intrinsic systems provides the blood supply for each neuron. The vessels reach the extrafascicular epineurium via the vasa nervosum after entering the mesoneurium and create a plexus that runs longitudinally at this level. The vessels continue their course toward the axon by passing in an oblique angle through the subsequent connective tissue layers and forming a longitudinally running plexus during their course at each level. This configuration allows for the individual fascicles to have an independent and rich blood supply, analogous to an individual nerve fiber[43,44] (Fig. 25-5). This passage of the vasculature, between the layers of connective tissue, may become an area of compromise within the intrafascicular layers; there are no associated draining lymphatics running with the vascular plexuses.

TRIGEMINAL NERVE IMAGING

Objective, noninvasive, radiologic imaging modalities for the preoperative assessment of the patient at risk for nerve injury, as well as monitoring for the postinjury and postrepair phases of neurosensory recovery, are highly desirable.[45] Radiologic assessment should be categorized with regard to the timing of the imaging period—that is, preinjury, postinjury, and postrepair phases. Preinjury assessment refers to the documentation of the in situ position of a nerve prior to any surgical intervention that places that nerve at risk for iatrogenic injury (e.g., third molar removal). Intraoperative monitoring of nerve function during a surgical procedure (e.g., sagittal split mandibular osteotomy) that involves a specific nerve may also be used, usually by a functional study of nerve conduction and electrophysiologic status, such as somatosensory evoked potentials (SSEPs).

Postinjury imaging is divided into a primary phase (i.e., following nerve injury and allowing for spontaneous neurosensory recovery without microneurosurgical intervention) and secondary phase (i.e., following surgical nerve exploration and repair). Primary postinjury imaging may be clinically significant if it can correlate objective radiologic findings with subjective, clinical examination findings and thereby guide the need for surgical intervention and possibly aid in treatment planning (e.g., the need for an interpositional nerve graft).

PREOPERATIVE RISK ASSESSMENT

Panoramic Radiography

The preoperative assessment of the position of the inferior alveolar nerve during patient consultation has been routinely performed with the use of a panoramic radiograph. Obviously, the information gained from this study is extremely limited because of the two-dimensional nature of the image, variable magnification of the bony anatomy (for the inferior alveolar nerve), and complete inability to visualize the position of the lingual nerve. Valuable information can be obtained from the panoramic radiograph as a stand-alone imaging modality in regard to the relationship of the IAN in the vertical plane, but not in the horizontal dimension. The most useful aspect of the panoramic radiograph is for assessing the increased potential for inferior alveolar nerve injury during third molar extraction based on the presence of several radiographic predictors[46,47] (Fig. 25-6).

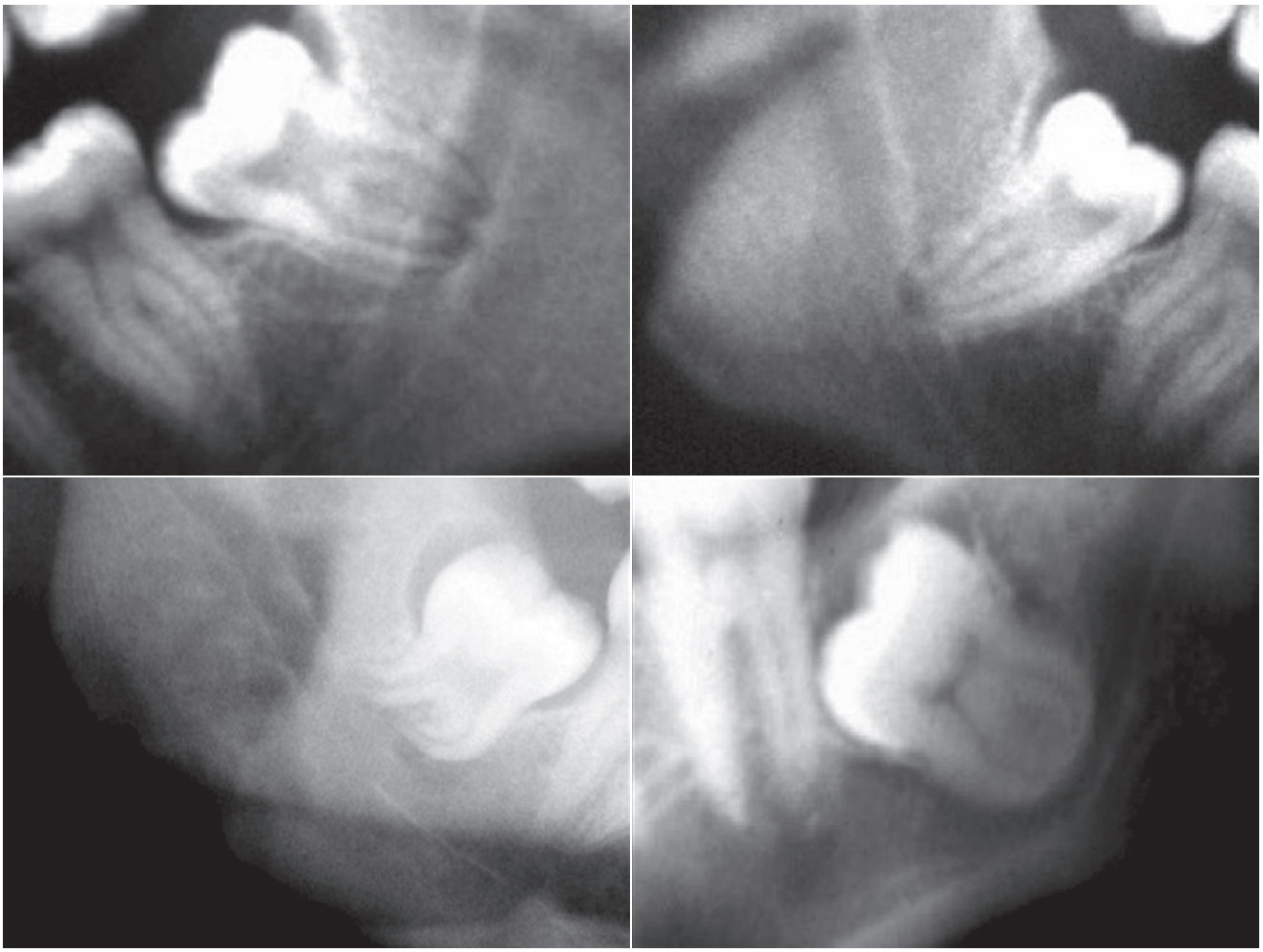


FIGURE 25-6 Panoramic radiographs showing examples of Rood radiographic predictors of root proximity to the inferior alveolar canal, including root darkening, root narrowing, root deflection, loss of the superior white line of the canal, and diversion of the canal.

Other types of plain radiographs, such as periapical or anteroposterior films and lateral cephalograms, are not routinely used for accurate preoperative routine risk assessment for IAN injury. Superimposition and variations of magnification of the structures, based on their location, do not allow for reliable and reproducible information to be obtained with plain films. Furthermore, even if the IAN can be visualized in the third molar region, only a rough outline of tooth and root anatomy can be obtained, making these images of limited value for nerve injury appraisal.

Computed Tomography

The use of CT for the assessment of nerve injuries is limited. An evaluation of bone window attenuation images may indicate violation of the cortical outline of the inferior alveolar canal, either from implant placement or following facial trauma involving the posterior mandible fracture (Fig. 25-7), but yields little information regarding the condition of the nerve or neurovascular bundle.[48] The use of soft tissue window images for the lingual or inferior alveolar nerves is compromised by poor-detail resolution, which precludes its routine application for neural assessment. Furthermore, dental artifacts often pose severe limitations in obtaining accurate information regarding the position of the LN to the lingual cortex of the mandible in critical areas, even in the soft tissue windows, and despite current high-resolution image acquisition.

In 1998, CT cone beam (CBCT) technology, until then used only in angiography, was used in the United States as a potential imaging modality for the maxillomandibular complex. The presurgical evaluation of the impacted mandibular third molar relationship to the IAN has gained popularity over CT scans and plain panoramic radiographs among surgeons[49-53] (Fig. 25-8). The need for accurate imaging with the lowest possible dose of radiation (following the ALARA rule—***a***s ***l***ow ***a***s ***r***easonably ***a***chievable) seems to be well satisfied with this technology. CBCT provides the desired three-dimensional representation of the anatomic location of interest with minimal distortion compared to traditional plain films and by simpler acquisition compared to CT systems.[54,55] Similar to panoramic radiography, CBCT can be used for preoperative risk assessment in various dentoalveolar procedures (e.g., third molar surgery or dental implants and preprosthetic surgery).[56-58] A major limitation remains the inability to visualize the lingual nerve, because no accurate soft tissue information can be obtained with CBCT.

High-Resolution Magnetic Resonance Imaging

MRI is the method of choice for visualization of all cranial nerves (CNs) and each nerve segment can be seen and examined in detail with specific sequences. Because of the complexity of the course and



FIGURE 25-7 Coronal CT scan through a mandibular body fracture showing canal involvement.

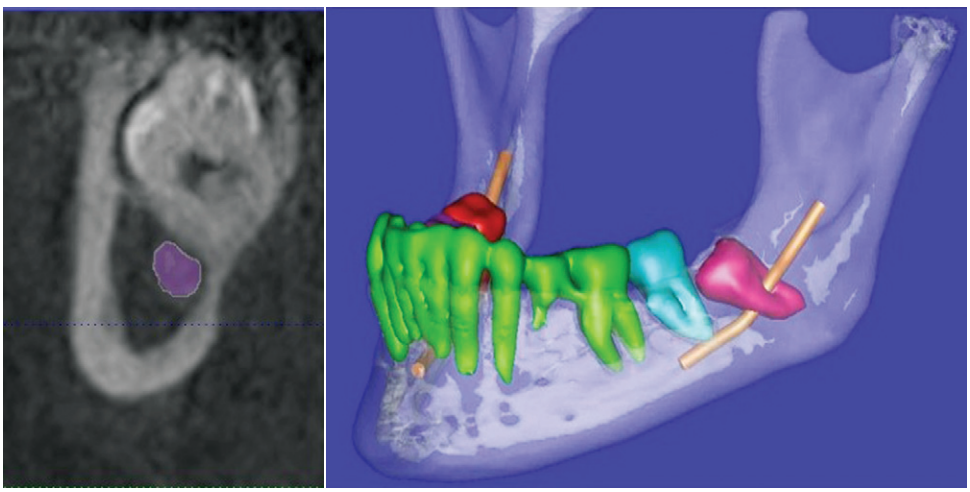


FIGURE 25-8 Cone beam CT scan showing the proximity of the inferior alveolar canal to an impacted third molar, and three-dimensional reconstruction of the scan to highlight the canal passing between the roots of the impacted third molar tooth.

surrounding anatomic structures, detailed examination of the CNs is made possible only with careful planning and MRI technique selection. The imaging plane, coil selection, slice selection, in-plane resolution, and use of special techniques can be tailored based on the CN and segment of interest so that the highest quality image possible is obtained. The trigeminal nuclei (intra-axial), the cisternal (preganglionic), and Meckel's cave (intradural) segments, contain the motor and sensory components of the trigeminal nerve and can be visualized with high-resolution T1- or T2-weighted MR images. At the anterior aspect of the gasserian ganglion, the sensory root divides into the ophthalmic, maxillary, and mandibular divisions, and each can be followed and examined separately based on their known course. The course of the lingual and inferior alveolar branches of the mandibular division, after it exits from the oval foramen, can be followed with high-resolution, contrast-enhanced, T1-weighted (T1W) or T1W three-dimensional fast field echo (T1W 3D-FFE) sequences in the axial, coronal, and sagittal or parasagittal planes.[59-63] Although detailed information can be obtained with the use of MRI, routine presurgical evaluation of the route and integrity of the LN and/or IAN is not undertaken. Instead, MRI is used as the preferred imaging modality for examination of the status of the CNs in the presence of a disease process or injury.[60,64-67]

In an attempt to document the in situ position of the lingual nerve in the third molar region directly, without surgical manipulation or tissue distortion artifact, high-resolution MRI (HR-MRI) has been studied. In one study, 10 patients (20 sides) of healthy adult volunteers without prior dental surgery were imaged using an HR-MRI sequence (PETRA [***p***hase-***e***ncoded ***t***ime ***r***eduction ***a***cquisition]) that enabled visualization of the lingual nerve (Fig. 25-9). This study documented that the lingual nerve position, although variable, is found to be superior to the lingual crest in 10% of cases and in direct contact with the lingual plate in 25% of cases.[18]

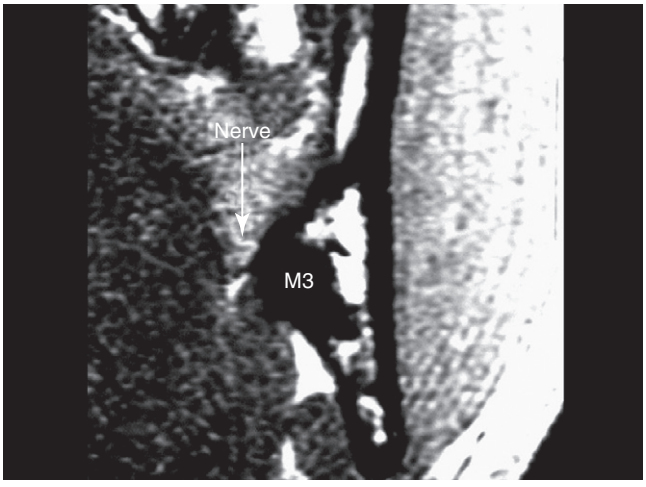


FIGURE 25-9 High-resolution MRI showing a kidney-bean shaped lingual nerve (Nerve) superior to the lingual crest, and in contact with the lingual plate in the third molar (M3) region.

Ultrasonography

Several reports have described the use of ultrasound (US) and high-resolution US technology, mainly for assessment of peripheral nerve lesions. This real-time advanced technology, with the currently available high-resolution probes, can offer compound imaging without radiation and in a relatively inexpensive manner. Although US has not been used nor investigated as a potential preoperative risk assessment tool for the trigeminal nerve, it has been demonstrated to be valuable in identification and safe advancement of the needle in brachial plexus and sciatic nerve blocks.[68] It would be reasonable to anticipate limitations with the use of US in the examination of the IAN in the third molar region because of the presence of bone and teeth. Visualization and documentation of the course and integrity of the LN, on the other hand, should be relatively easy, requiring training and familiarity of the operator with the regional anatomy.

POSTINJURY ASSESSMENT

Perhaps most clinical interest is in documenting the postinjury condition of nerves by objective means, because the information gathered by clinical and radiologic examination could be used to aid in staging the degree of neural injury. With increased image resolution, the precise degree of architectural disruption of the nerve could be visualized and surgical intervention could be planned. Also, this information could be used to document the position of the injury prior to surgical exploration for repair. For example, this documentation could help avoid surgical nerve exploration in the third molar region if the injury occurred in the pterygomandibular space as a result of a mandibular block injection injury. Finally, radiologic techniques could be used to monitor neurosensory progression objectively in conjunction with the clinical examination, after nerve injury or in the postrepair phase.

Panoramic Radiography

The postsurgery assessment of the nerve-injured patient usually includes a panoramic radiograph that may demonstrate clinically significant findings. The presence of a foreign body in the region of one or both nerves must be ruled out; these may include metallic foreign bodies from rotary instruments or amalgam particles from neighboring teeth, as well as retained tooth or root fragments following third molar surgery. The presence of an iatrogenic surgical disturbance of the nerves may be indicated by evidence of bone removal in proximity to the inferior alveolar neurovascular bundle or LN (Fig. 25-10). However, a panoramic radiograph or any other plain film is rarely used to monitor progression following injury or repair.

Computed Tomography

Postoperative investigation of the surgical site for examination of the integrity of the IAN canal or the presence of material, such as tooth or root fragments within the canal, could be superior and more reliable with CT or CBCT imaging than traditional panoramic imaging. Direct investigation of the integrity of the LN cannot be

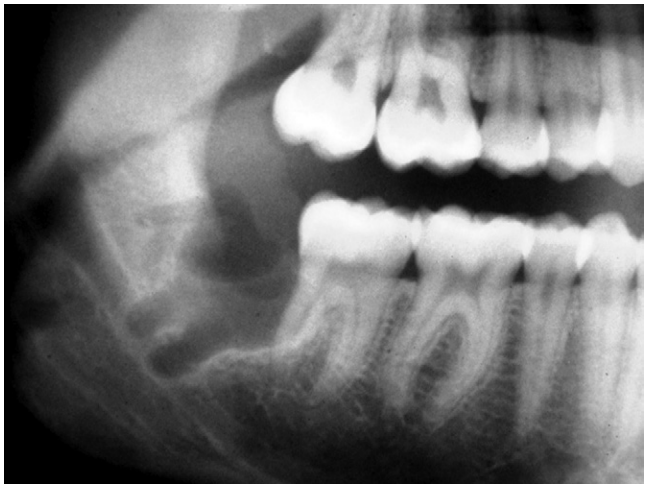


FIGURE 25-10 Panorex following third molar removal showing the presence of preoperative radiographic predictors of root proximity to the canal, and evidence of the use of a drill distal to the crown of the extraction site, in a patient with right lingual nerve injury.

reliably examined with either modality because of the limitations discussed earlier in the preoperative risk assessment section. Disruption of the lingual cortex of the mandible at the third molar region that may be noted in a postoperative panoramic radiograph and may imply trauma to the LN in the region can be reviewed in more detail with CT or CBCT. Direct comparison of pre- and postimages can be made, adding to the information gathered from the clinical examination and potentially assisting in the decision for the need of exploration in the region.

Magnetic Source Imaging

One of the few objective radiologic studies that documents inferior alveolar nerve injuries involves the use of magnetic source imaging (MSI), which combines magnetoencephalography (MEG) and HR-MRI. MEG technology uses magnetic fields to measure electrical brain activity; it is influenced less by intervening soft tissues than electroencephalography (EEG) and therefore produces a more detailed image, with higher resolution. Similar to SSEPs, a stimulus is applied peripherally and a signal is recorded centrally; this enables measurement of signal latency and amplitude. The information obtained from MEG is combined with HR-MRI images to produce a structural and functional magnetic source image of a particular region of the brain. McDonald et al[69] used MSI on six patients with unilateral inferior alveolar nerve injury and have shown that MSI technology may be able to differentiate various grades of nerve injury. The findings on clinical and MSI examinations were correlated with surgical findings; neural continuity defects were identified as different radiographically from intact nerves. Despite some limitations of the study design (small *n*, lack of blinded examiners and surgeons, and other study designs flaws), there is potential for MSI to be applied in the postinjury and postrepair phases to monitor the progression of neurosensory recovery.

High-Resolution Magnetic Resonance Imaging

The application of the HR-MRI modality to the assessment of the nerve postinjury is in its early trial phases. Presumably, with improved resolution, a variety of anatomic changes in the nerve may be visualized. First, a change in nerve diameter may be visualized in cases of nerve injury with Wallerian degeneration of the nerve segment distal to the site of injury with an acute or gradual decrease in nerve diameter. Second, an acute change in nerve position may be seen—for example, where the lingual nerve is retracted into the region of the third molar socket, with the formation of a lateral adhesive or exophytic neuromatous segment. Third, a change in nerve shape may be seen—for example in a case of a fusiform neuroma-in-continuity, where a change in the shape of the nerve for a certain distance with return to normal shape distal to the neuroma may be able to be visualized.[70]

The application of HR-MRI to postinjury neural assessment is hindered by several factors. The degree of image resolution and magnification significantly limits precise anatomic examination of the neural elements. The ability to image the internal architecture of neural anatomy will require dramatic improvements in resolution from currently available techniques. Also, although the lingual nerve lies within soft tissue and its course is unaccompanied, the inferior alveolar nerve lies within a cortical bony conduit and is joined by an artery and a vein throughout its course. Preliminary studies with HR-MRI have allowed gross visualization of the lingual nerve because it is the sole structure in the area, but examination of the inferior alveolar nerve has been complicated by the presence of the vessels, although attempts to attenuate the image signal can overcome this problem (see earlier section on magnetic resonance neurography). Depending on the plane of image section, HR-MRI may miss several anatomic indicators of nerve injury. A transverse (or coronal, in the case of the lingual nerve in the third molar region) section of the nerve may not visualize a short discontinuity or abrupt alteration in the course of the nerve, depending on the distance between images. This may be avoided with the use of a sagittal or longitudinal image along the course of the nerve. However, this is difficult because the position of the nerve varies normally in the uninjured patient and may change significantly in the injured patient, thereby requiring patient repositioning or redirection of the imaging plane.[71]

The use of noninvasive, lack of radiation exposure HR-MRI for the nerve-injured patient would provide the advantage of correlating the results of clinical neurosensory testing and subjective patient evaluation with an objective assessment of the anatomy of the injured nerve site. Although it may seem that a frank transection injury (Sunderland grade V; see later) might be visualized easily with HR-MRI, the less severe injuries (Sunderland III and IV) could be extremely difficult to discern radiographically. Future study designs with HR-MRI include an experimental group of postinjury patients who will undergo clinical neurosensory testing and HR-MRI and then microneurosurgical nerve exploration and repair, if indicated. This would allow correlation of postinjury radiologic results and findings at the time of surgery to

determine the ability of HR-MRI to predict the actual degree of anatomic nerve injury accurately. HR-MRI might also prove useful in monitoring the progression of anatomic neurosensory recovery (correlated with physiologic clinical signs and subjective symptoms) following nerve injury and/or microneurosurgical repair.[45]

Magnetic Resonance Neurography

Following the application of MRI technology to blood vessels (magnetic resonance angiography [MRA]), direct imaging of nerves with magnetic resonance neurography (MRN) was a logical progression. The MRN images are obtained using axial, coronal, and longitudinal T1 and T2 image acquisition, with customized phased array coils and imaging protocols. The application of MRN relies on its ability to distinguish nerves from surrounding structures such as blood vessels, lymph nodes, ligaments, adipose tissue, and ducts. This would allow isolation of the IAN from the neighboring artery and vein contained within the inferior alveolar bony canal. MRN studies to date have documented the ability to distinguish intraneural from perineural masses, demonstrate nerve continuity versus discontinuity at the fascicular level, and localize extraneural nerve compression prior to nerve exploration.

Most research has focused on larger, peripheral motor nerves, including the brachial plexus, sciatic, peroneal, and femoral nerves.[61,65,72-75] One report has documented nerve compression and signal hyperintensity of an IAN in a patient with a lymphoma of the pterygomandibular space. MRN has been able to document increased diameter of injured nerves, increased signal intensity, and longitudinal variations associated with nerve injury and recovery. There does not seem to be any correlation between the amount of hyperintensity and degree of neural injury, and its significance has not yet been clearly defined. The finding of signal hyperintensity has been demonstrated for a transient period following neural anastomosis and distal to a nerve graft site. The remarkable ability of MRN to depict fascicular architecture is based on the difference in fluid composition of the neural elements. The fascicles contain a preponderance of endoneurial fluid and axoplasmic water, whereas the interfascicular space is largely composed of fibrofatty connective tissue. In a sense, these images may be able to define radiographically the histologic characteristics of different grades of nerve injuries set forth by Seddon and Sunderland. Similarly, sequential images could be used to monitor nerve recovery at the fascicular level. One of the most advantageous characteristics of MRN images is the ability to image the nerve in a longitudinal plane. In a technique similar to that of MRA to image the anatomy of an abdominal aortic aneurysm, these MRN images can easily be assessed for variations in nerve anatomy, diameter, location, discontinuity, and signal intensity, which may indicate areas of nerve injury and thereby guide surgical intervention, as well as monitor neurosensory recovery.

Ultrasonography

Recently, some promising findings were reported with the use of US for visualization of the lingual trauma in the pig cadaver head. In the study by Olsen et al, the iatrogenic injuries created were successfully categorized in 17 of 27 attempts once the examiners became familiar with visualization of the LN. The major remaining limiting factor in the use of US for this application is the lack of training and familiarity with US technology and imaging among surgeons. The possibility of incorporating US for investigation of the integrity of LN postoperatively, along with clinical evaluation, seems promising. The potential for US examination in several subsequent visits in a noninvasive manner, without the need for radiation, additional cost, or discomfort with the ability to document findings of every examination for comparison and evaluation of progression, make this modality reasonably valuable.

POSTINJURY FUNCTIONAL ASSESSMENT

Among the imaging modalities available, it should be evident that the only studies that could potentially contribute to the functional assessment of the post–nerve repair are SSEPs, MSI, high-resolution functional MRI, MRN, and US technology. Success or failure of nerve repair grafting or direct anastomosis can be assessed only after several months have elapsed and is typically based on neurosensory examination, not imaging. The use of MRN has proven to be valuable to evaluate the repair site for neuroma formation or problems with the sutures when there is no recovery following repair, and this imaging may indicate the need for early surgical intervention. The few limitations posed by the presence of a hematoma in the early phases of nerve repair that were initially discussed in the literature are no longer an issue with the current advances in MRN imaging. Finally, nerve continuity after direct repair or interpositional grafting may be examined with US, but more details are possible with the use of MRN. Also, the major limitations with the use of US are the lack of training among surgeons and lack of familiarity with the acquired images for appropriate interpretation.

The current advances in MR imaging technology with high-resolution, functional, or metabolic-based images (BOLD [***b***lood ***o***xygenation ***l***evel–***d***ependent]) certainly allow for detailed examination of the neural structures, as well as associated pathology and nerve injury patterns.[76] Perhaps the main potential limitations with the routine use of these advanced imaging modalities for the investigation of IAN and LN injury and recovery are the cost associated with these studies and the lack of expertise of neuroradiologists and surgeons regarding their interpretation and clinical significance.[60,75,77-80]

CLASSIFICATION OF TRIGEMINAL NERVE INJURIES

Several complex cellular events occurring in and around the nerve structure are responsible for the response to nerve injury and are required for the regeneration and restoration of neurosensory function. Two nerve injury classification schemes, Seddon and Sunderland, are described here. These provide for a correlation between clinical symptoms and histologic changes observed within

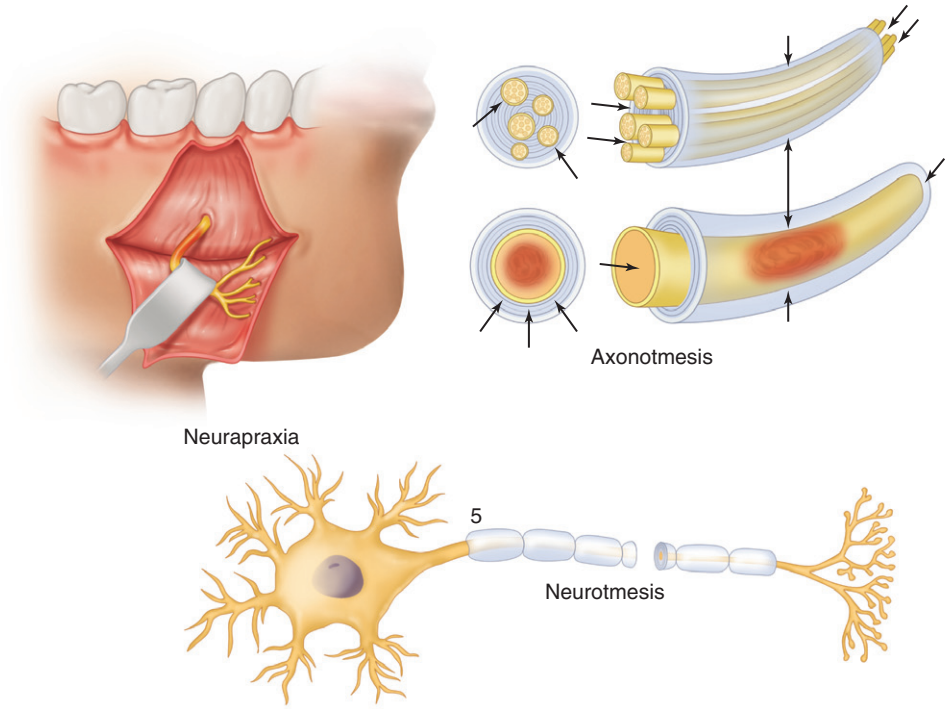


FIGURE 25-11 Seddon classification system including examples of neurapraxia, with nerve stretching, axonotmesis, with variable degrees of axonal injury, and neurotmesis, with nerve transection.

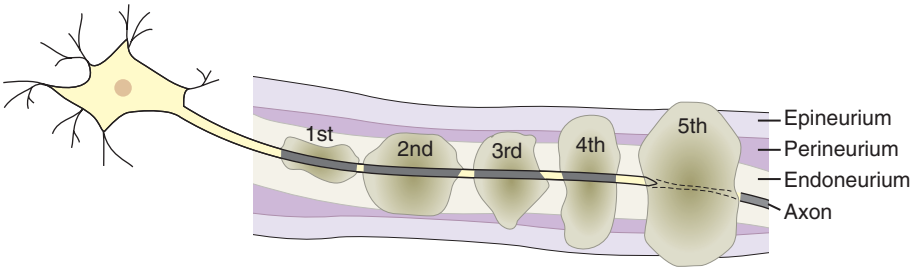


FIGURE 25-12 Sunderland classification system showing the 5 degrees of nerve injury with progressive injury beginning with damage confined within the endoneurium (first-degree injury) and extending outward to the perineurium (second degree), then through the perineurium (third degree) and to the epineurium (fourth degree), and finally through the epineurium with discontinuity (fifth degree).

the nerve structure at the time of injury. Seddon's classification is based on the time between injury and recovery and degree of recovery, whereas Sunderland places an emphasis on the specific histologic degree of damaged neural structures.

SEDDON CLASSIFICATION SYSTEM

The Seddon classification system includes three levels of nerve injury (Fig. 25-11)—neuropraxia, axonotmesis, and neurotmesis.[81] In neuropraxic injuries, the integrity of the axon is maintained, and the injury indicates a local conduction block from a transient anoxic event caused by acute vascular interruption of the epineurial or endoneurial vasculature. Neuropraxia is usually the result of a mild nerve manipulation, traction, or compression injury; it is characterized by a reversible conduction block with a favorable outcome, with rapid and complete recovery within days to a few weeks of the event. No axonal degeneration occurs in neurapraxic injuries and damage is confined to the endoneurium only. Axonotmesis involves axonal damage, corresponds to Sunderland injuries degrees II to IV; variable degrees of demyelination and axonal injury and therefore spontaneous recovery vary significantly in this category of injuries. More details are provided in the following discussion of Sunderland's classification of nerve injuries. Finally, neurotmesis implies complete or near-complete nerve transection that includes epineurial discontinuity. Spontaneous recovery is unlikely, whereas neuroma formation may occur more commonly.

SUNDERLAND CLASSIFICATION SYSTEM

Sunderland revised and further subclassified nerve injuries in 1951 and described a five-degree classification system (Sunderland's I to V degrees of nerve injury) based on histologic findings of the degree of nerve involvement[82] (Fig. 25-12). A first-degree Sunderland injury is the same as Seddon's neuropraxia described earlier, and was further divided by Sunderland into three types based on severity and recovery time. First-degree

TABLE 25-5 Classification of Nerve Injuries and Associated Findings

Injury type	Histologic Findings	Response	Recovery Pattern	Recovery Rate	Treatment
First-degree (Seddon neuropraxia)	Transient ischemia, anoxia, ± segmental demyelination, intrafascicular edema	Conduction block	Complete	Fast (hours to weeks)	None indicated
Second-degree (Seddon axontmesis)	Axon and myelin interruption, (intact endoneurium, perineurium and epineurium)	Wallerian degeneration distal to injury	Complete	Slow (weeks)	None indicated
Third-degree	Injury involves endoneurium (intact perineurium and epineurium)	Wallerian degeneration and some degree of neuron cell death	Variable	Slow (weeks to months)	Nerve exploration maybe considered
Fourth-degree	Injury involves endoneurium and perineurium	Wallerian degeneration, neuron cell death, neuroma formation, intrafascicular fibrosis	None	Spontaneous recovery not likely	Microneurosurgery
Fifth-degree (Seddon neurotmesis)	Complete nerve transection, continuity disruption	Wallerian degeneration, cell death neuroma, fibrosis	None	No spontaneous recovery possible	Microneurosurgery

type I injury is the result of nerve manipulation that causes transient blood supply interruption that once restored, allows for full sensory recovery after only a few hours. First-degree type II injury involves intrafascicular edema formation, from fluid exudate or transudate, resulting from moderate traction or compression on the nerve. Sensory recovery follows edema resolution within a few days. First-degree type III injury results from more aggressive nerve manipulation and may involve segmental demyelination; recovery requires a few days to weeks.[83]

Second-degree injuries involve axonal and myelin interruption but the intact endoneurium, epineurium, and perineurium allow for axonal regeneration. Recovery is a relatively slow process and may take up to 1 year for complete return to normal sensation, but is possible because of the intact supporting connective tissue structures. These injuries are caused by traction or compression on the nerve.

In third-degree injuries, the endoneurium is involved in the injury but the epineurium and perineurium remain intact. The usual causes are the same as for first- and second-degree injuries, and some spontaneous recovery is expected. However, complete recovery is not likely and surgical exploration may be considered.

Fourth-degree injuries caused by traction, compression, needle injection, or chemical injury lead to disruption of the endoneurium and perineurium; the epineurium remains intact. These injuries are associated with a poor potential for spontaneous recovery and a high probability of neuroma formation and intraneural fibrosis. Surgical intervention is required for these injuries, especially if there is no improvement within 3 months following injury.

The fifth-degree injury in Sunderland's classification system corresponds to Seddon's neurotmesis, which implies a nerve transection, avulsion, or laceration of the nerve trunk, with complete disruption of all components of the nerve organization; there is little to no chance of spontaneous recovery. Extensive fibrosis, neuroma formation, and/or neuropathic changes occur in these serious clinical situations and microneurosurgery is indicated. In cases of witnessed nerve transection, immediate surgical intervention is required. Table 25-5 summarizes the types of injuries, corresponding histologic changes, responses, recovery pattern, rate of recovery, and recommended treatment.

AXONAL AND CELLULAR RESPONSE TO INJURY

Nerve injury involves several events and variable responses, but the basic process of nerve healing remains the same and involves a sequence of degeneration followed by regeneration.[84,85] Within hours after axonal injury, a series of events occurs at the primary site of injury, as well as at the distal and proximal portions of the axon and cell body. The changes within the cell body and the nerve fiber proximally depend on the proximity of the injury to the cell body, as well as the severity of the injury. Within 6 hours of injury, the metabolic rate of the cell body increases significantly, resulting in edema and migration of the nucleus toward the periphery of the cell body and upregulation of the rough endoplasmic reticulum (Nissl substance), with increased protein synthesis that will be exported from the cell body toward the site of injury. These events are collectively termed *chromatolysis* and represent the histologic correlation of RNA and protein synthesis, a programmed event intended to

ensure cell survival that peaks within 14 to 21 days following acute injury.[86-94] Chromatolysis signals the proliferation of perineural glial cells that along with macrophages, phagocytes, and Schwann cells, begin the clean-up process of cellular debris at the injury site.[84,85,95]

When the injury involves the axonal fibers (Sunderland grades II to V grade injuries), a series of molecular and cellular events occur, simultaneously or consecutively, and collectively result in Wallerian degeneration.[96] These events occur throughout the distal nerve trunk and the small portion of the proximal end close to the injury site. The distal portion of the axon becomes edematous so that its ends are sealed, whereas the myelin that surrounds the axon breaks down into droplets.[97] The debris is phagocytosed by Schwann cells, which demonstrate enhanced proliferation and activity; the recruited macrophages also participate in this débridement process. These events are a prerequisite for nerve regeneration to occur, initiated within hours of injury and completed within 1 week following the nerve injury.[98-104] Neural degeneration occurs in the distal and proximal nerve ends so the final result is empty endoneurial tubes that act as conduits for nerve regeneration; these tubes are present up to the site of the injury.[97,101,105-107]

Proximally, the transected axon forms sprouts, or axonoplasmic extensions, that mimic the axon growth cones observed during embryonic neural development. Several substances produced by neural and supportive tissues are required for these events to occur, which ultimately lead to the growth and regeneration of the axon toward its receptor target.[100,108-111] Each axon may have as many as 50 collateral axonal sprouts. The high proliferative activity of the Schwann cells noted during these phases is thought to be required for the production of new myelin that will host the new axon. This theory has been recently challenged by Yang et al,[99] who demonstrated that Schwann cell proliferation may not be an absolute requirement for nerve regeneration but that this is achieved via existing Schwann cells that survive the insult, but do not divide. The regeneration is axon-driven and axon-dependent, as opposed to a predominant Schwann cell influence.

In any case, if these processes occur in the appropriate chronologic order, spontaneous nerve regeneration will be achieved. In cases in which there is interruption or unfavorable sequencing of these events, the outcome will be failure of reinnervation of target receptors and possible neuroma formation. Neuromas represent a disorganized mass of collagen fibers and nerve sprouts that are randomly oriented.[112-116] Neuromas can be of the following types: amputation (stump neuroma); neuroma-in-continuity (central or fusiform neuroma, Fig. 25-13); or lateral (lateral exophytic, or lateral adhesive neuroma, Fig. 25-14). Neuromas may be painful or asymptomatic and histologic differentiation between the two clinical scenarios is not possible. What appears to distinguish painful and nonpainful neuromas appears to be their neurophysiologic properties, based on studies that have shown that neuromas may be sensitive to both mechanical stimuli as well as activation of the sympathetic system by chemical or physiologic manipulation.[117-119] Finally, consideration must be given to the mechanism of injury and the resultant nerve injury. For clean lacerations of the nerve, primary repair is usually indicated but for avulsive-type injuries, a delayed primary repair is preferred. A delayed primary repair is typically performed at 21 days following injury, which allows time for the area of injury to become defined macroscopically. At 3 weeks' time, the levels of neurotrophic and neurotropic factors are at their highest and the nerve is most amenable to repair.

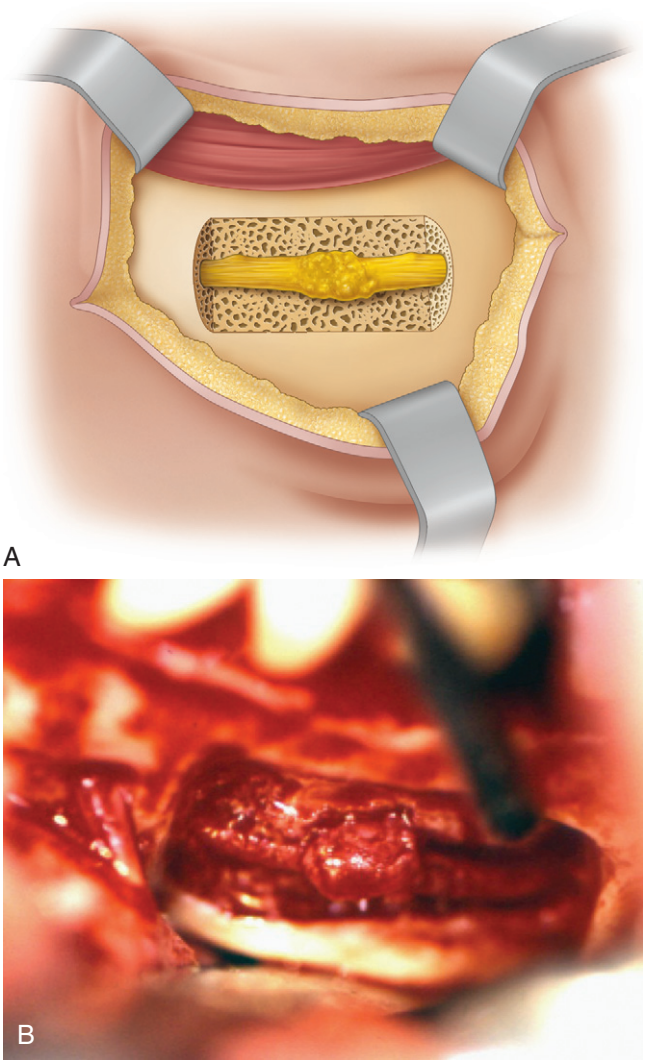


FIGURE 25-13 **A,** Diagram of a fusiform neuroma-in-continuity. **B,** Clinical example of an inferior alveolar nerve neuroma-in-continuity at the apex of a mandibular first molar following endodontic therapy with nerve injury.

MECHANISMS OF INJURY TO THE TRIGEMINAL NERVE

Several commonly performed procedures in the maxillofacial region are associated with potential injury to the terminal branches of the trigeminal nerve.

LOCAL ANESTHETIC INJECTION

Injury to the IAN and LN is possible during the administration of mandibular block local anesthesia. Interestingly, prolonged sensory alterations are rare, considering the number of blocks administered daily by dental professionals. The ratio of injuries to injections has been estimated as between 1:26,762 and 1:160,571 although this may not represent the actual incidence, because not all cases of sensory disturbances are reported.[120,121] Harn and Durham, in 1990, reported an incidence of transient sensory disturbance of 3.62%, with only a 1.8% incidence of long-term changes (>1 year duration).[122] There are a number of proposed mechanisms that result in this injury and the exact cause remains unclear; in addition, there is no reliable way of avoiding these types of injuries. Direct penetration of the nerve by the needle, hematoma formation from vessel laceration, direct laceration of the nerve from a barb on the tip of the needle after repeated injections, needle contact with cortical bone, and chemical injury from intraneural injection are reasonable possibilities.[122-124] Direct toxicity of the local anesthetic solution (specifically, 4% articaine and 4% prilocaine), especially when deposited within the confines of the epineurium, has been associated with sensory changes after mandibular blocks.[125-128] Box 25-1 presents the conclusions that can be made based on the current understanding of injection-related nerve injuries. These cases are usually associated with only a limited distribution of neurosensory alteration, reflecting the small number of fascicles insulted or involved in this type of injury.[129]

THIRD MOLAR SURGERY

Complications associated with third molar removal are the most common cause of trigeminal nerve injury reported and can occur during any stage of the actual procedure (Fig. 25-15), as well as during postoperative care.[130-134] The overall incidence of IAN injury during third molar surgery is estimated between 0.41% and 7.5%, resulting in temporary sensory changes in 2% to 6% and permanent disturbances in 0.5% to 2% of cases. Stretching or manipulation of the lingual nerve from tissue retraction during third molar surgery is reported to cause temporary sensory disturbances in rates from 6.4% to as high as 15%, but less than 1% result in permanent changes.[129,135-141] Several risk factors associated with an increased risk of IAN injury during this procedure have been discussed in the literature. These include

BOX 25-1 Clinically Based Conclusions About Local Anesthesia–Related Nerve Injuries

1. Not predictable, possibly preventable
2. Painful response on injection (electric shock sensation) does not always accompany an injury
3. More common in female patients
4. More commonly result in dysesthesia than hypoesthesia
5. May follow a nonanatomic distribution (involvement of more than one division of the trigeminal nerve) because of segmental demyelination
6. More common with higher concentration anesthetic solutions (e.g., 4% articaine)
7. Surgical exploration not indicated because of nature and location of injury
8. Lingual nerve is more commonly affected than inferior alveolar nerve
9. Spontaneous recovery usually occurs within 8 wk of injury
10. Less than one third of cases lasting longer than 8 wk recover fully
11. Pharmacologic management indicated for unpleasant sensations (dysesthesia)

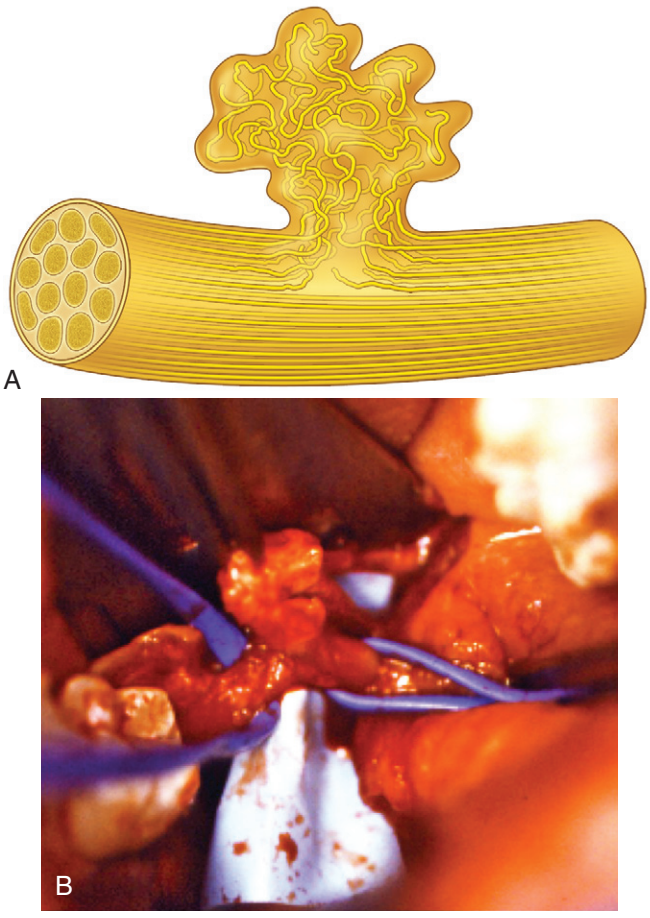


FIGURE 25-14 **A,** Diagram of a lateral exophytic neuroma. **B,** Clinical example of a lateral exophytic neuroma of the lingual nerve because of third molar removal.

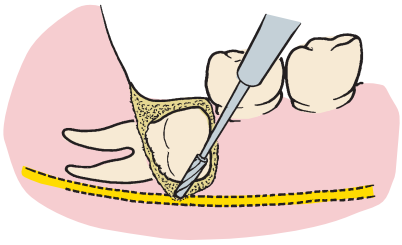


FIGURE 25-15 Diagram showing possible iatrogenic injury of the inferior alveolar nerve during extraction of a deeply impacted third molar. (From Hupp JR, Ellis E, Tucker MR: *Contemporary oral and maxillofacial surgery*, ed 5, St. Louis, 2008, Mosby.)

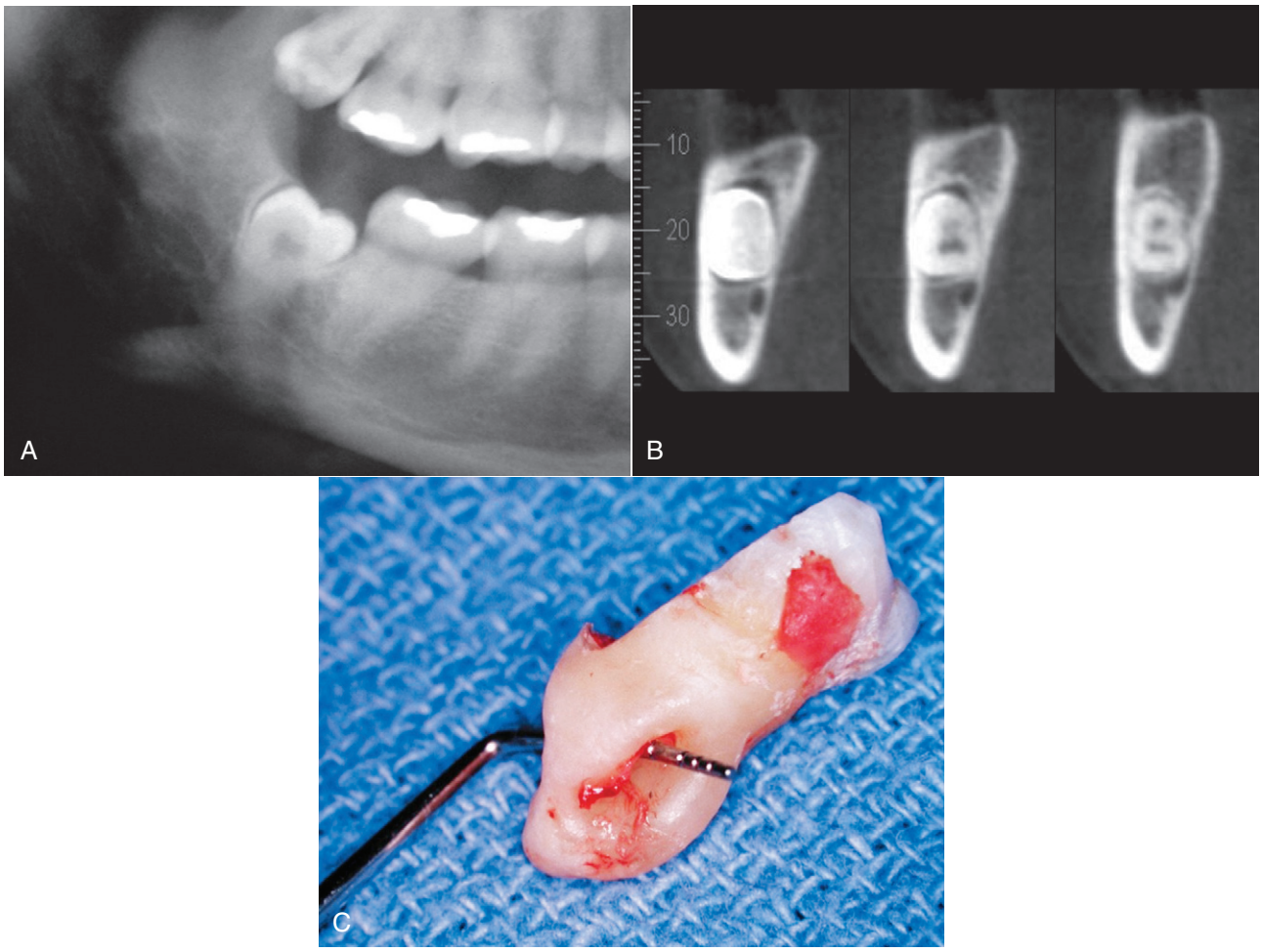


FIGURE 25-16 **A,** Panorex showing root proximity to the inferior alveolar canal. **B,** Cone beam CT showing root proximity to the inferior alveolar canal. **C,** Third molar following removal with a periodontal probe showing the location of the inferior alveolar neurovascular bundle. Despite an attempt to section the tooth to avoid nerve injury, the nerve was transected during the extraction.

advanced age, female gender, tooth angulation, depth and type of impaction, integrity of the lingual cortex, complexity of the procedure, and surgeon experience.[142,143] Rood and Shehab, in 1990, described seven radiographic predictors of potential proximity of the third molar to the IAN on panoramic radiographs that may be helpful in the decision making process regarding extraction versus more conservative approaches, such as coronectomy or observation.[47] These predictors include darkening, deflection or narrowing of the tooth root, diversion, narrowing or interruption of the white line of the IAN canal, and a dark or bifid root apex. The most significant radiographic findings of potential nerve injury included root darkening, root deflection, and interruption of the white line of the canal. It is important to note that subsequent studies have reported controversial findings, both in support of and opposition to the value of these radiographic predictors in nerve injury[144,145] (Fig. 25-16).

DENTAL IMPLANT SURGERY

With the wide use of dental implants for dental rehabilitation worldwide, injury to the IAN is a fairly common occurrence, with a 0% to 40% reported incidence of nerve injury caused by implant placement, depending on location (Fig. 25-17), and patient management can be problematic.[146,147] The IAN, in particular, is at risk in the molar region and anterior to the mental foramen, especially in edentulous patients, because of the associated alveolar ridge resorption, in addition to the anterior extension, or genu, of the neurovascular bundle beyond the foramen. These injuries can result from surgical exposure, during preparation of the osteotomy site, or at the time of implant placement. Mechanical compression of the neurovascular bundle can result from overextension and canal violation during the osteotomy that causes infracture of the cortex of the canal at the site and direct pressure on the IAN (Fig. 25-18). Alternatively, bleeding within the canal from a laceration of the inferior alveolar vein or artery with an implant drill can cause pressure similar to that of a compartment syndrome. Finally, direct compression on the neurovascular bundle following restoration of the implant with mastication, or because of prior clot formation and subsequent ectopic calcification, may occur. Although extensive literature is available regarding the use of modern imaging modalities for

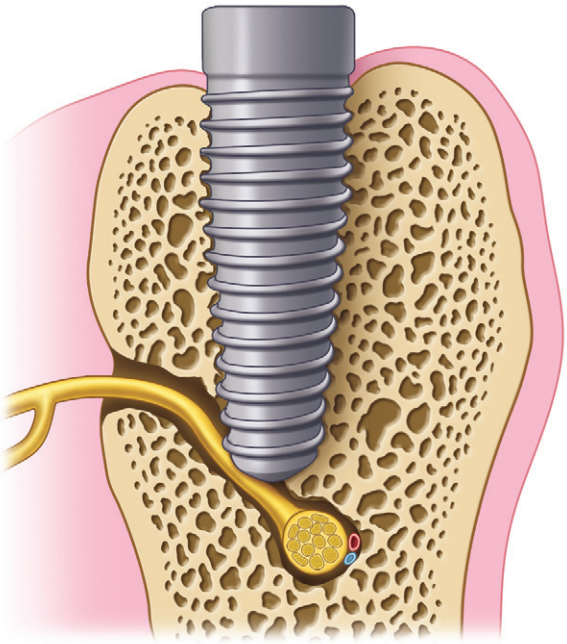


FIGURE 25-17 Diagram showing implant-related nerve injury that can occur when the implant is in proximity to the closed canal with possible bleeding, edema, and development of a compartment syndrome that causes deleterious effects on the nerve, even in the short term.

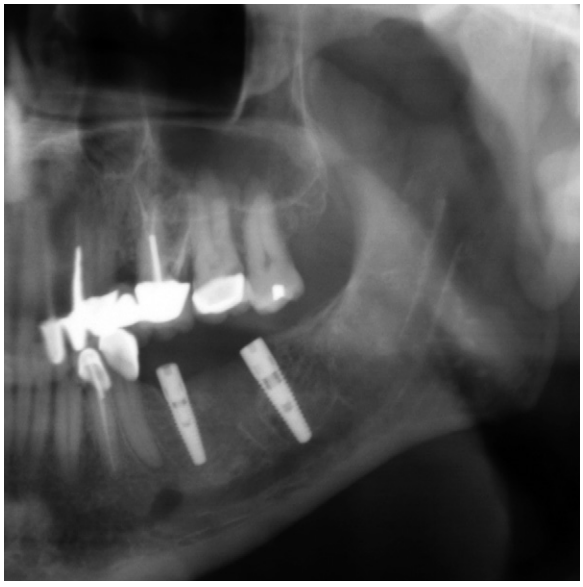


FIGURE 25-18 Panorex showing the distal implant placed within the confines of the inferior alveolar canal in a patient with inferior alveolar nerve paresthesia.

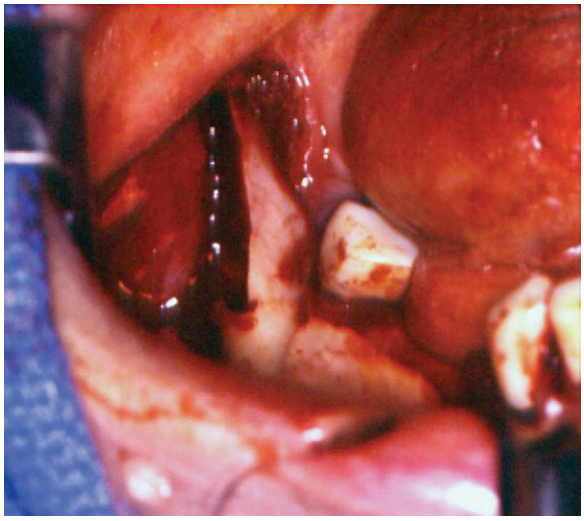


FIGURE 25-19 Ramus graft procedure that places the inferior alveolar nerve at risk for injury because of its lateral location in this region of the mandible.

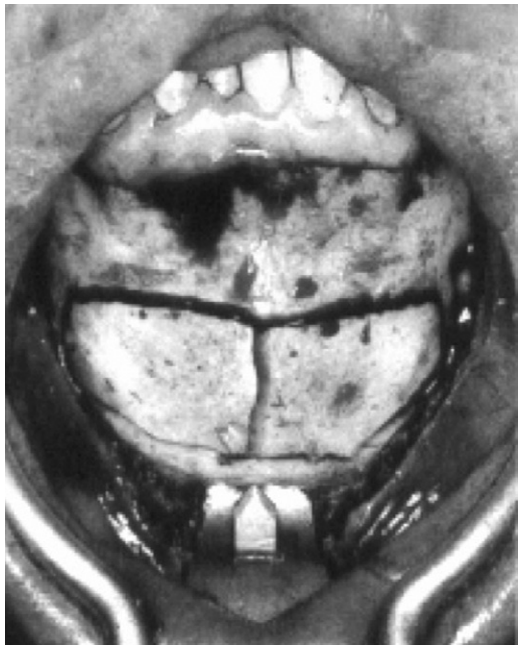


FIGURE 25-20 Genial bone graft harvest that may sacrifice the incisive branch of the inferior alveolar nerve and result in paresthesia to the anterior mandible dentition.

treatment planning in implant dentistry, data are lacking on the most appropriate modality for nerve injury risk assessment, as discussed earlier.[3,147-152]

Management of these injuries is tailored individually based on the mechanism of injury and may involve implant removal as soon as the injury is recognized, or the use of pharmacologic therapy, if indicated for dysesthesia. Immediate implant replacement with a shorter implant, may be considered but is dependent on the exact cause of the trauma and the need or lack thereof for nerve canal exploration, as well as the timing and recognition of the occurrence.[129,146,153-155]

Procedures aimed at augmenting the alveolar bone height in the posterior mandible or improving the site to facilitate implant placement, such as bone grafting, distraction osteogenesis (DO), or IAN lateralization, carry their own risks for nerve injury. During bone harvesting from the mandibular ramus (Fig. 25-19) or chin region (Fig. 25-20), the IAN or mental nerves could be inadvertently injured, especially when a large area requires augmentation. When small, single, tooth-sized grafts are harvested, and appropriate preoperative

radiographic examination of the area is undertaken, this risk appears to be minimal.[156] The same branches of the trigeminal nerve are at risk during securing autogenous or allogenic bone graft material at the recipient site, either from direct compression of the nerve from the graft or from hardware (e.g., plates and screws) used to secure the graft in place. DO, another method used for alveolar ridge height augmentation, may lead to nerve injury, again at the time of the osteotomy or directly from to the device used for the distraction. The actual response of the IAN to the DO process has been studied in animal models. It has been shown that several events occur at the cellular level, including activation and proliferation of Schwann cells, and a limited degree of axonal degeneration and regeneration. The result is minor nerve injury that is reversible and has no long-term sequelae.[157-159]

Nerve lateralization for the facilitation of implant placement is a viable option in select cases. With this approach, the lateral cortex of the mandible is removed and the IAN is exposed and lateralized so that implants of adequate length can be placed (Fig. 25-21). The procedure inevitably causes some neural trauma, but the injury is "controlled" while the risk of compartment syndrome is eliminated, along with the risk of direct injury to the neurovascular bundle from the implant placement itself. The procedure is associated with a high incidence of neurosensory changes, with some permanent changes, but with excellent results in implant stability and success and high patient satisfaction.[160-166] If the nerve lateralization procedure requires release of the mental nerve from the mental foramen, the incidence and degree of neurosensory dysfunction is increased over a transposition of the nerve posterior to, and not involving, the mental foramen. Recovery after nerve lateralization should be expected within 3 to 6 months, although up to 30% to 40% of patients may experience long-term neurosensory dysfunction, especially older individuals.[166] The use of piezosurgery in a cadaveric study (10 sheep mandibles with 20 lateralizations) has found no disturbance of structures beyond the epineurium; the overall degree of injury was found to be less than when the procedure was performed with conventional burrs.[167]

MAXILLOFACIAL TRAUMA

Maxillofacial trauma and surgery for the correction of facial injuries may result in disturbances of the peripheral branches of the trigeminal nerve in the vicinity of the traumatic injury. The overall incidence of neurosensory impairment has been reported to be 70.9%. In cases of nondisplaced fractures, the incidence was found to be 54.4%, whereas a significant increase was noted in displaced fractures of 88.2%. As expected, cases of direct injury had 100% neurologic impairment.[168] Fractures of the mandible may result in neurosensory alterations caused by laceration, traction, or compression of the IAN from bony segment displacement (Fig. 25-22) or treatment of the fracture with iatrogenic injury to the nerve canal (Fig. 25-23). Similarly, midface fractures or soft tissue trauma, lacerations, or avulsions may violate the infraorbital canal and damage the ION, leading to transient or permanent sensory alterations[168-171] (Fig. 25-24). In the study by Kloss et al, hypoesthesia of the ION was found in 10% of single and 16% of multiple midface fractures.[172]

Reduction of fractures with alignment of segments and removal of loose bony segments that impinge on the nerve will assist in spontaneous neurosensory recovery. Unfortunately, on occasion, the actual treatment of facial fractures may cause further injury to peripheral nerve branches of the trigeminal nerve because of nerve retraction or from direct injury caused by inadvertent placement of plates and screws.

ORTHOGNATHIC SURGERY

Neurosensory alterations are common sequelae of orthognathic surgery for correction of dentofacial deformities, especially mandibular procedures.[173] During sagittal split osteotomy (SSO), the neurovascular could be injured at several locations and during the various steps of the procedure. These include during medial dissection at the lingula region, the actual osteotomy procedure, or mobilization or fixation of the segments. If the IAN is found to be located in the proximal segment after the osteotomy, it may need to be mobilized, which may cause further injury. The location of the IAN in relationship to the inferior border of the mandible, ramus height, dentofacial deformity to be corrected, and patient age and gender have all been associated with various incidences of neurosensory disturbances.[174-177] In addition, the incidence of neurosensory alterations during mandibular orthognathic surgery has been found to increase with intraoperative complications, usually unfavorable splits.[178] Furthermore, IAN sensory alterations appear to occur more frequently with additional procedures e.g., genioplasty in addition to SSO), but are transient; spontaneous full recovery is usually reported within 6 to 12 months.[173,179,180] Interestingly, a substantial difference has been demonstrated between subjective and objective findings regarding sensory alterations after SSO, with normal sensation reported in 73.7% of osteotomy sites; objective testing revealed normal sensation in only 34.2% of cases.[181]

During maxillary or midface procedures, the ION is at risk for injury, usually because of soft tissue flap retraction rather than direct injury that results in sensory changes of the upper lip, cheeks, lateral aspect of nose, and infraorbital region. Based on the nature of the injury (e.g., traction, compression, or pressure), full recovery is expected and usually achieved without intervention.[180] Segment fixation with plates and screws could theoretically cause direct injury to the ION or LN from bicortical screw overpenetration (Fig. 25-25), but this is easily avoidable with adequate exposure, visualization, and protection of the nerve.

MAXILLOFACIAL PATHOLOGY

Several odontogenic and nonodontogenic benign cysts and tumors may be found in the nerve-bearing segment of the mandible in intimate relationship with the IAN, often causing displacement of the canal; in some cases, the canal may be located within the pathologic entity (Fig. 25-26). This also applies to tumors of the maxilla in the vicinity of the IOC. Benign pathology usually is not associated with neurosensory disturbances, with the

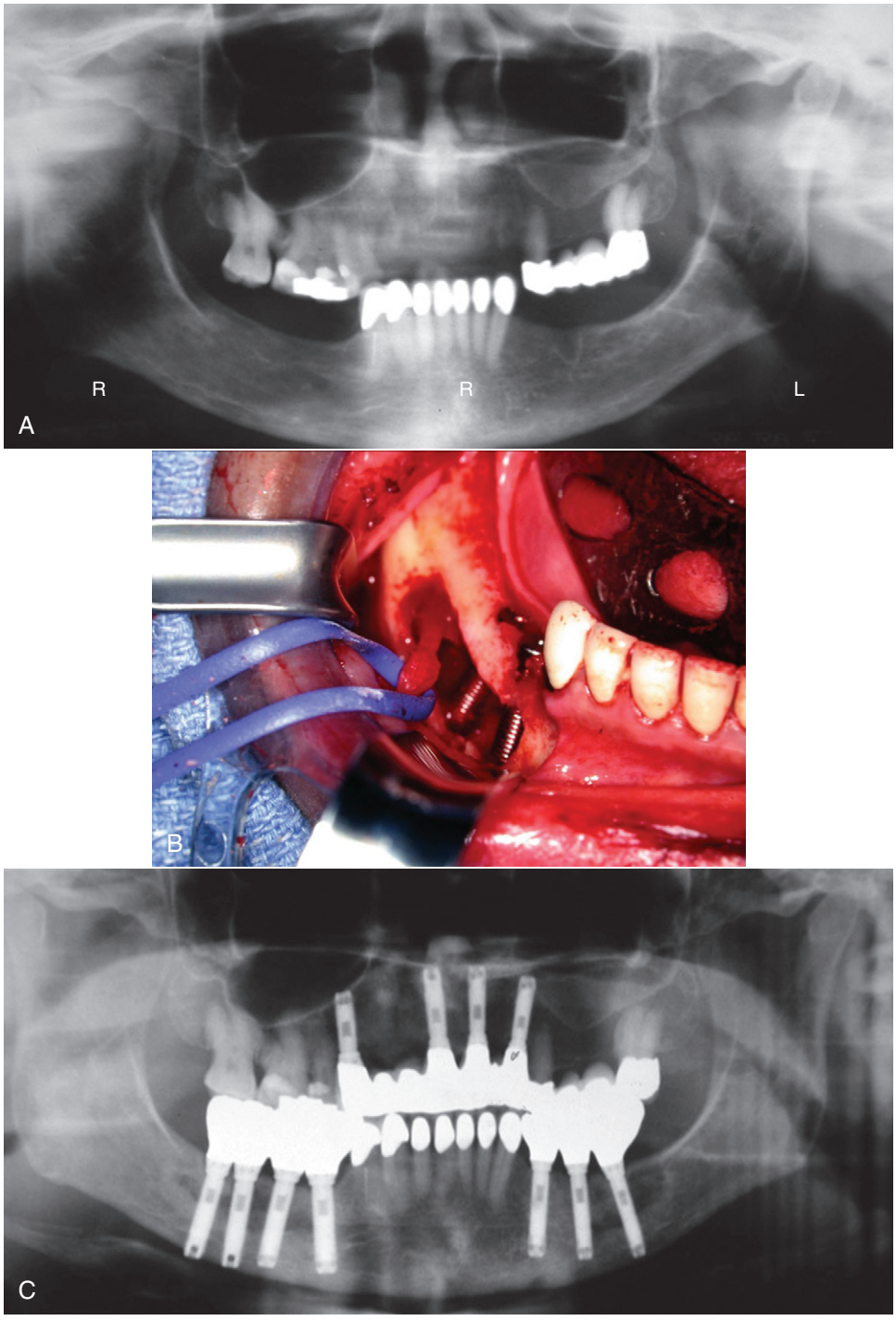


FIGURE 25-21 **A,** Panorex showing posterior mandibular edentulism with insufficient bone height above the canal and inadequate interocclusal clearance for implant placement. **B,** Nerve repositioning procedure results in a neurapraxic injury in a controlled fashion during implant placement. **C,** Panorex showing bilateral inferior alveolar nerve repositioning with restored implants that engage the superior and inferior borders of the posterior mandible for stability.



FIGURE 25-22 Panorex showing an open, displaced, unfavorable left mandible angle fracture. Note the displacement of the inferior alveolar canal.



FIGURE 25-23 Panorex showing fixation of a right mandibular body fracture with screws that penetrate the inferior alveolar canal in a patient with right inferior alveolar nerve dysesthesia.

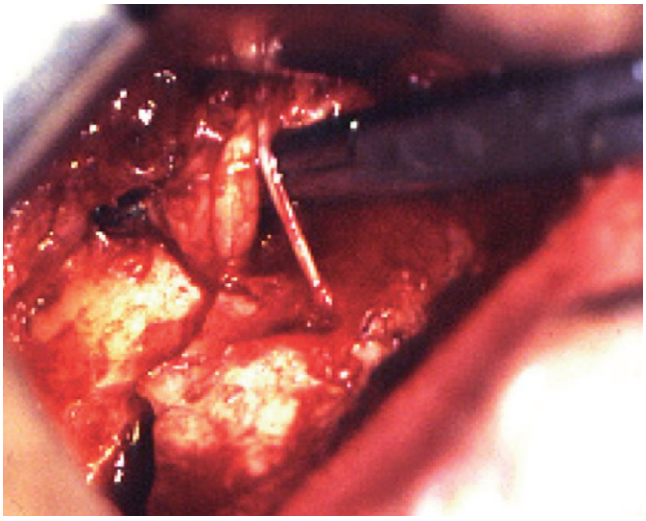


FIGURE 25-24 Intraoperative view compression of the terminal branches of the right infraorbital nerve exiting from the infraorbital formen in a patient with a displaced right zygomaticomaxillary complex fracture.

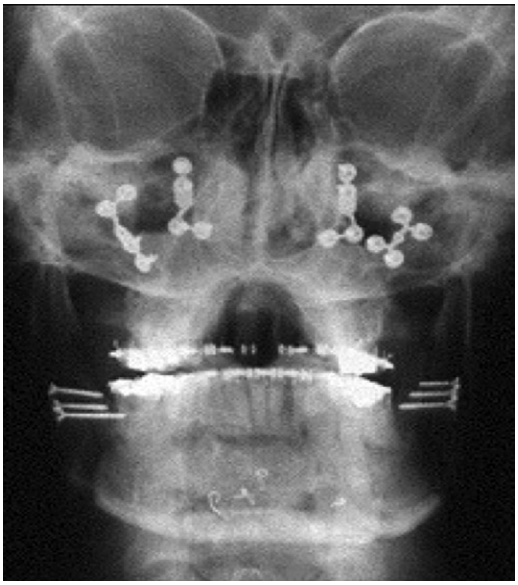


FIGURE 25-25 Posteroanterior skull film showing overpenetration of the bicortical screws used to stabilize the sagittal split osteotomies. This places the lingual nerve at risk for iatrogenic injury during orthognathic surgery.

exception of neural origin or vascular tumors originating from the involved neurovascular bundle (Fig. 25-27). On the contrary, malignant lesions usually will be associated with sensory changes early, as is the case with specific pathology (e.g., adenoid cystic carcinoma with neurotropism), in cases of tumors with aggressive behavior, when the IAC is invaded by the tumor, or the tumor has caused a pathologic mandible fracture (Fig. 25-28). Infectious processes are similarly associated with neurosensory changes if they involve nerve-bearing areas of the jaws; these usually resolve spontaneous with resolution of the infection (Fig. 25-29).

Treatment of maxillomandibular pathology often involves extensive bone and soft tissue resection that may include the IAN, mental nerve (MN), or ION, resulting in permanent anesthesia in the distribution of the corresponding resected nerve. On occasion, nerve pull-through procedures or use of adjuncts, such as enucleation and curettage with cryotherapy, have been advocated for some tumors in an effort to minimize morbidity associated with more aggressive treatment and permanent neurosensory disturbances. These approaches remain somewhat controversial with regard to the associated risk of recurrence, in addition to a conflict with the basic oncologic principles for management of these tumors.[182-190] Acceptable outcomes, with complete neurosensory recovery after treatment of benign odontogenic pathology with simple enucleation followed by cryotherapy, have been reported in the literature and may be a viable option in select cases.[191-194]

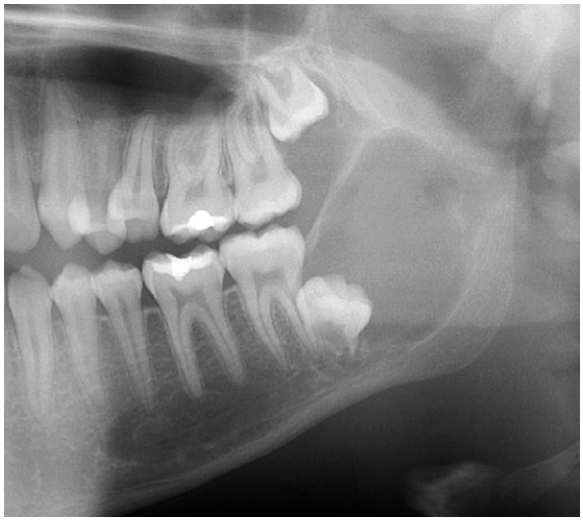


FIGURE 25-26 Panorex of a keratocystic odontogenic tumor causing displacement of the left inferior alveolar canal, and without paresthesia.

ENDODONTIC AND CHEMICAL INJURY

Endodontic therapy and periapical root surgery may place the IAN at risk for injury. The nerve may be injured by physical disruption with a file by overinstrumentation of the canal(s), root resection, or apicoectomy procedures, or by physical compression from extrusion of the filling materials used (Fig. 25-30). The other mechanism of injury is chemical neurotoxicity, which may result from the use of certain root canal medications to treat the canal(s). These types of IAN injuries may be caused by mechanical or chemical factors, or both, and may result in a foreign body reaction or granuloma formation in the canal that could impede spontaneous neurosensory recovery. Unfortunately, these injuries are difficult to manage, with variable success following microneurosurgery, and a large proportion of these injuries may manifest clinically as dysesthesia. For extrusion of potentially neurotoxic root canal filling materials into the IAC, prompt exploration, decompression, and débridement of the foreign material may be beneficial in preventing long-term sequelae.

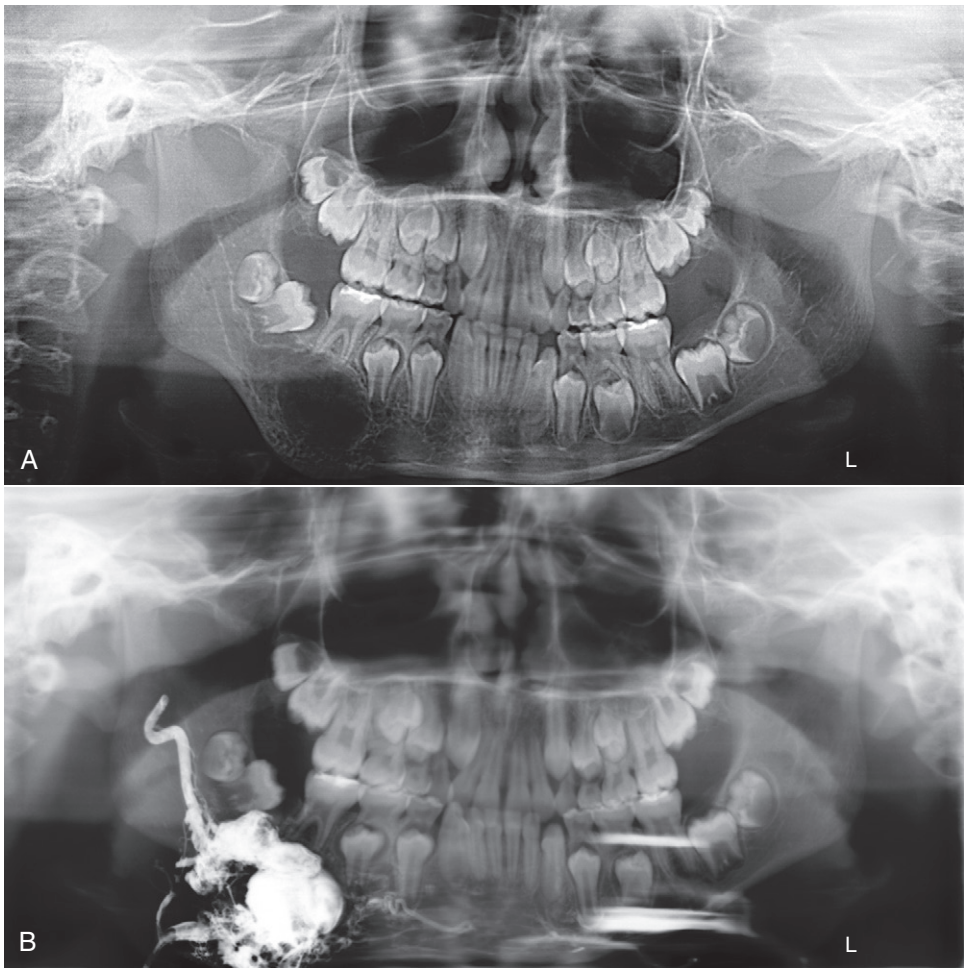


FIGURE 25-27 **A,** Panorex of an arteriovenous malformation of the right mandible involving the inferior alveolar canal. **B,** Panorex of the same patient following arteriography with embolization.

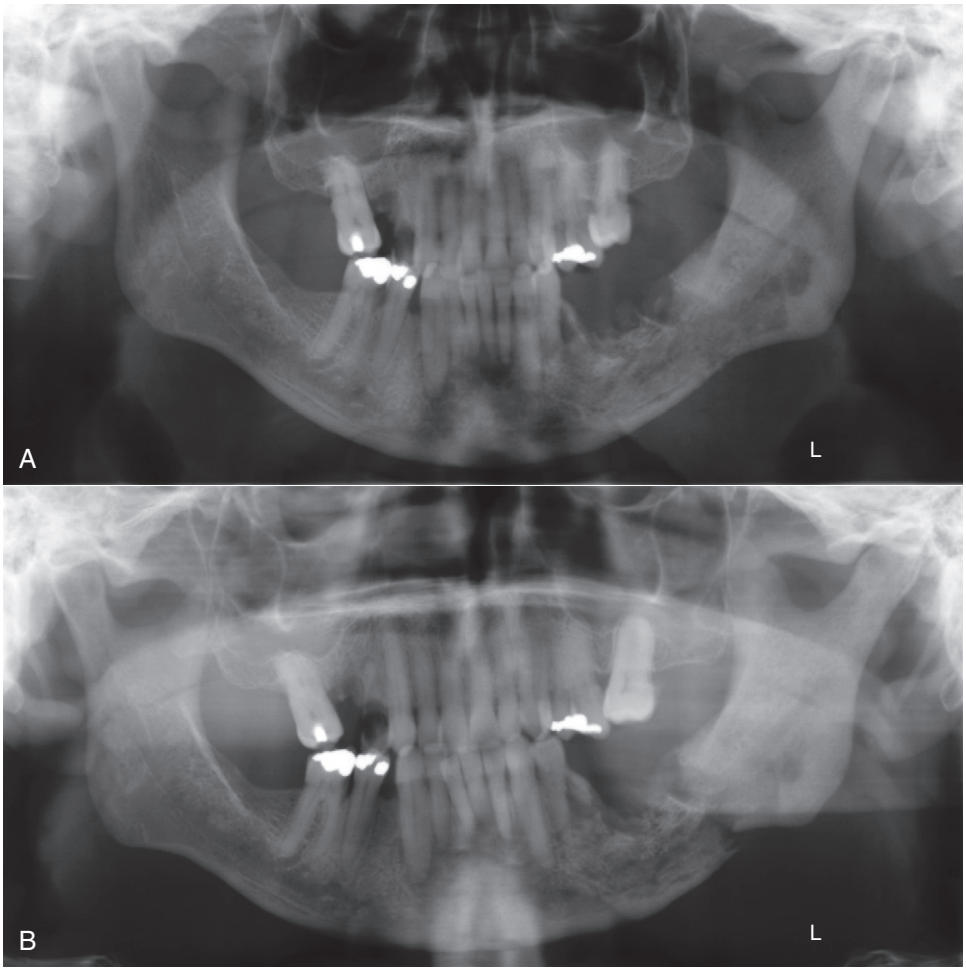


FIGURE 25-28 **A,** Panorex of multiple myeloma involvement of the left mandibular body. **B,** Panorex of the same patient with progression of the disease with a displaced unfavorable pathologic fracture of the left mandibular body, with resultant inferior alveolar nerve paresthesia.

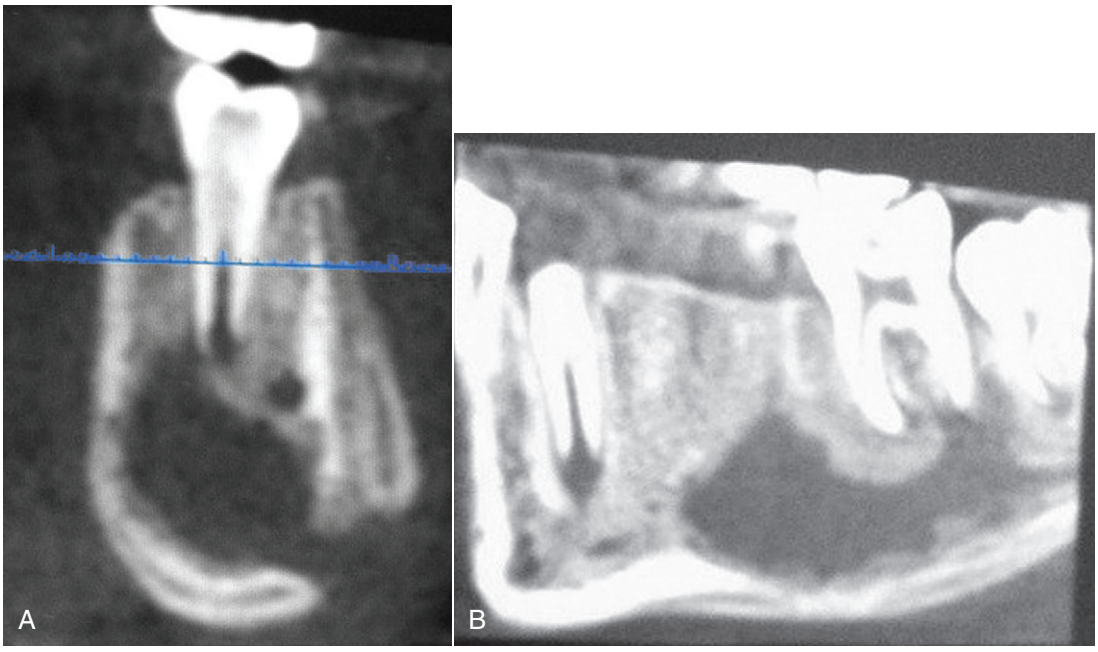


FIGURE 25-29 **A,** Coronal cone beam CT showing Garre's osteomyelitis of the mandible with buccal cortical perforation and proliferative periostitis in a young patient involving the inferior alveolar canal with paresthesia. **B,** Sagittal cone beam CT of the same patient showing the extent of involvement of the Garre's osteomyelitis throughout the marrow involving the inferior alveolar canal.

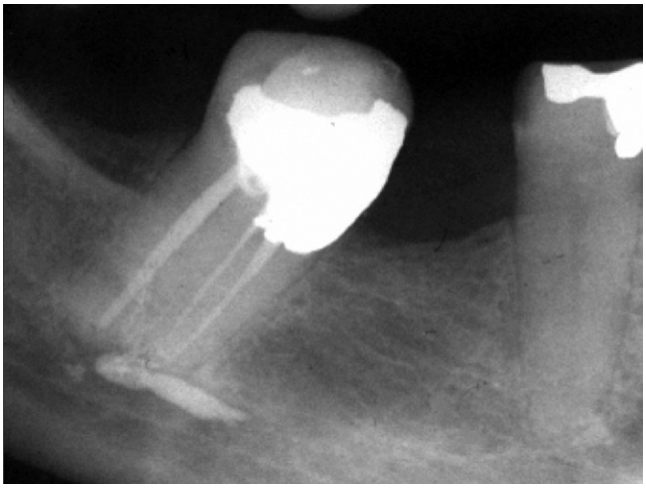


FIGURE 25-30 Periapical film showing root canal filling material within the inferior alveolar canal in a patient with inferior alveolar nerve dysesthesia.

In addition to root canal medications, other chemical agents are potentially neurotoxic, such as tetracycline, surgical (oxidized cellulose), and prolonged exposure of the nerve to Carnoy's solution (a solution of ferric chloride, glacial acetic acid, chloroform, and ethanol).

CLINICAL NEUROSENSORY TESTING

A detailed history and clinical examination, along with objective testing that includes the use of specific neurosensory tests (NSTs), pain questionnaires, and visual analogue scales, are crucial in the assessment of the patient who has sustained trauma to the trigeminal nerve. Attention to detail regarding the onset of symptoms, events associated with the current complaints, and any changes, including worsening or improvement, require careful documentation and play a critical role in the treatment decision making process.[129,170,195,196] Descriptive terms used by the patient to qualify his or her symptoms need to be documented in the chart exactly as stated by the patient.

In a critical evaluation of the accuracy of NSTs to diagnose trigeminal nerve injuries accurately, Zuniga et al have demonstrated that for LN assessment, NSTs have superior sensitivity and specificity compared with examination of the IAN. In addition, IAN findings were associated with a high incidence of false-positive and false-negative results, 23% and 40%, respectively.[197] This was subsequently supported in a recently published comparison between clinical NSTs and current perception threshold.[198] NSTs should be performed for patients with neurosensory changes to determine the degree of sensory impairment, monitor recovery, and assist in decision making in regard to the need for surgical intervention. The clinical NST protocol involves mechanoreceptive fiber testing performed first (two-point discrimination, static light touch, directional discrimination, and vibratory sense), followed by nociceptive fiber testing (pain stimuli and thermal discrimination). Testing should be performed in the same manner and sequence on each occasion, in a quiet, calm, comfortable environment, with the patient in a semireclined position. It is important that the clinician explain in details the tests that will be performed and assure the patient that there will be no pain involved. An uninvolved normal site should be used to establish a baseline control and to demonstrate the tests about to be performed. Marking can be done directly on the patient's face with erasable markers, on photographs obtained prior to testing, or on drawings on standard recording forms, if available. If the marking is done directly on the face, the patient needs to be photographed; all photographs, drawings, and forms must be kept in the records for documentation purposes and for future reference and comparison.

The clinical neurosensory testing protocol (Fig. 25-31) begins with outlining the area of sensory disturbance or mapping, using brush stroke directional discrimination to differentiate between the normal and affected areas.

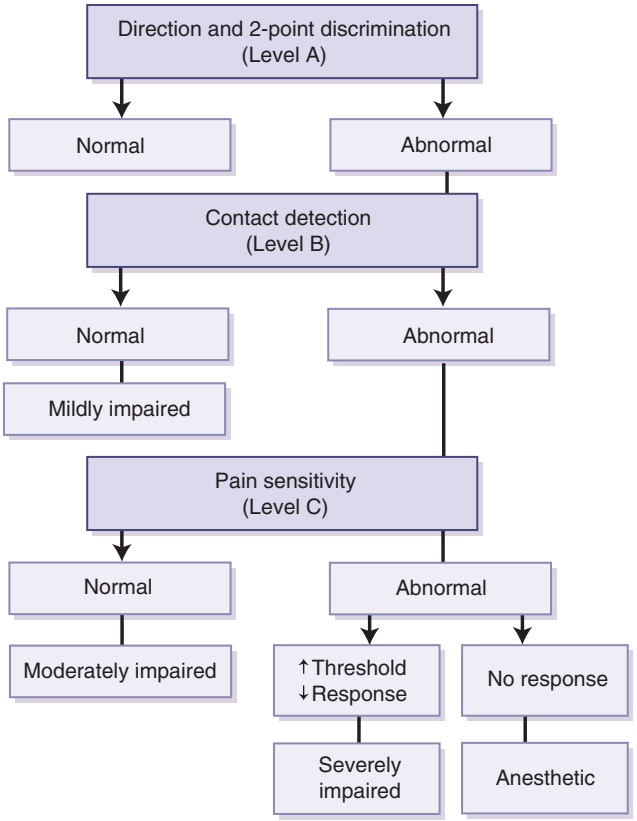


FIGURE 25-31 Clinical neurosensory testing algorithm. Level A testing (brush stroke direction and two-point discrimination) is done first and, if normal, the examination is normal (Sunderland first-degree injury). If level A testing is abnormal, level B testing with contact detection is performed and, if normal, the examination indicates mild impairment (Sunderland second degree injury). If abnormal, level C testing (pinprick and thermal discrimination) is done and, if normal, the examination indicates moderate impairment (Sunderland third degree). If Level C is abnormal, then the patient is either severely impaired (Sunderland fourth degree), or with no response to testing, is considered anesthetic (Sunderland fifth degree).

Brush stroke directional discrimination is performed with a camel hair or fine sable brush that is stroked across the affected area at a constant rate and the patient is asked to indicate the direction of movement. The correct number of responses out of 10 is recorded. Static two-point discrimination is performed next, using any device that allows for measurement between two points; these tips should be blunt, not sharp, which would stimulate the nociceptive fibers. The closest distance that the patient can discriminate two points versus one point is recorded. If the findings at this level of testing, brush stroke and two-point discrimination (level A testing) are normal responses, no further testing is required. The patient NST is considered normal and the injury can be classified as Sunderland first-degree injury. However, if the findings are abnormal, testing proceeds to the next level. Level B testing involves contact detection and is performed with von Frey hairs or Semmes-Weinstein monofilaments of several diameters that reflect the stiffness of the filament and the force required to deflect it on contact. The narrowest filament consistently detected by the patient that requires the least force to deflect is recorded for the control and involved sites. Both levels A and B testing examine the integrity of the large myelinated A alpha and A beta fiber types. If level B testing is normal, no further testing is required and the patient is considered mildly impaired, representing a Sunderland second-degree injury. If findings are abnormal at level B, then testing proceeds to the next level, which involves pinprick nociception and thermal discrimination and evaluates smaller myelinated A delta fibers and unmyelinated C fibers. A 30-gauge needle, in the absence of a pressure-sensitive device, can be used for pinprick testing, with care not to cause injury to the skin or mucosa, and an all or none response may be recorded. For thermal discrimination, specialized Minnesota thermal discs are available, or simpler options such as ice or hot water on a cotton swab can be used for suprathreshold thermal testing and has been shown to be valuable for assessment of iatrogenic injuries to the IAN and LN.[199] Normal responses at level C testing imply a moderate impairment (Sunderland third-degree injury). Abnormal findings may be consistent with severe impairment, with an increased thermal threshold or decreased nociceptive response (Sunderland fourth-degree injury), or lack of response to pinprick or temperature with complete anesthesia (Sunderland fifth-degree injury).[129]

Pain as a result of nerve injury can be assessed specifically with a simple visual analogue scale or with a more complex McGill pain questionnaire that can also be used for the evaluation of altered sensation; it is a useful tool for continuous monitoring of progress. Direct or indirect injury to the LN may cause taste alteration, which can be a significant component of the patient's subjective complaints. Taste requires special testing that may involve the entire mouth or be localized, but it is generally difficult to assess taste alterations; there is poor correlation between the degree of injury and these changes, as well as response to surgical intervention.[129,200-203] Taste can be evaluated with 5% saccharose (sweet), 5% saline (salt), 5% citric acid (sour), and 0.5% chinin hydrochloride (bitter).[202]

MICRONEUROSURGERY

INDICATIONS

Careful patient assessment and collection of information from neurosensory testing are crucial in the decision making process for treatment recommendations, and specifically the need for surgical innervation (Box 25-2). With the exception of an observed nerve transection, which requires immediate surgical repair, successful treatment of all other injuries requires a balance of timing to allow for potential spontaneous recovery, but without delaying timely microsurgical intervention. The complex events surrounding nerve injury, with degeneration and regeneration at various levels—distally, proximally, and centrally—make the timing of surgical intervention critical when it is indicated.[204-207]

Clinical neurosensory testing, along with subjective findings, should guide and dictate appropriate treatment. When improvement is documented during periodic nerve testing, surgery is not indicated. Microneurosurgery is also not indicated in cases of central neuropathic pain or most cases of dysesthesia or unpleasant sensations, and consideration should be given to pharmacologic management of these difficult clinical scenarios. If persistent paresthesia is documented in subsequent visits, referral to a surgeon with expertise in microneurosurgery is prudent. Although there are several timing guidelines regarding surgical intervention, the scientific evidence to support them is limited.[208] It is generally accepted that prolonged delay may decrease the success of microneurosurgical repair. Regardless of the exact timing, indications for microneurosurgery include a witnessed nerve transection, no improvement of significant hypoesthesia (>50% compared to control site), the presence of a foreign body, neuroma formation, progressive worsening of hypoesthesia or dysesthesia, and intolerable subjective hypoesthesia.[209] Intervention for LN paresthesia should be considered earlier (1 to 3 months postinjury) than for the IAN (3 to 6 months postinjury), because the inferior alveolar canal provides

BOX 25-2 Indications and Contraindications to Microneurosurgery

INDICATIONS

Complete anesthesia
<50% residual sensation (Sunderalnd III, IV, V)
No improvement within 3 mo after injury
Observed nerve transection
Early dysesthesia (neuroma formation)
Intolerable subjective paresthesia

CONTRAINDICATIONS

Continuing improvement in sensation
Late dysesthesia (especially IAN)
Central neuropathic pain
Complex regional pain syndrome
Trigeminal neuralgia
Atypical facial pain
Anesthesia dolorosa

BOX 25-3 Recommended Time Frame for Microneurosurgery (When Indicated)

Lingual nerve: 1-3 mo after injury
Inferior alveolar nerve: 3-6 mo after injury

a conduit to spontaneous nerve regeneration, whereas the LN lies in soft tissue. This may explain why IAN injuries have a higher rate of spontaneous recovery than LN injuries (Box 25-3). Some studies have indicated that no surgical intervention should be considered longer than 1 year postinjury because distal end-organ neurotization is less likely. However, others have suggested that timing is less important and improvement can be achieved when microneurosurgery is performed after 12 months.[129,209]

MICRONEUROSURGERY FOR TRIGEMINAL NERVE INJURIES

Microneurosurgical procedures are best performed in the operating room under loop magnification or with an operative microscope. Surgical exposure to assess the IAN, LN, MN, and ION can be easily performed via intraoral approaches and, in these cases, surgical loop magnification may be more practical than a microscope. There is a clear aesthetic benefit from intraoral approaches that avoid incisions on the face and associated potential scars or injury to other vital structures, but may be more challenging based on the site of the injury.[129] The more proximal the injury, the more challenging is access; for example, an LN injury at the third molar region is more challenging to repair than an IAN at the mental foramen area. The individual patient's anatomy, surgeon's experience, mechanism of injury, and location of the injury are further considerations when deciding the most appropriate surgical approach.

The LN is approached intraorally via a paralingual or lingual gingival sulcus incision (Fig. 25-32). The paralingual incision allows for a small incision and direct visualization of the nerve, but may be more challenging to identify the nerve stumps after transection because of retraction into the soft tissues. The latter approach involves a longer incision with anterior and posterior releases, but there is less risk of retraction of the nerve endings during dissection.[210] For the IAN (and MN), a vestibular incision with identification of the MN and lateral decortication to expose a portion of the IAN are usually adequate to mobilize the mental nerve for repair. In cases of restricted mouth opening, an extraoral approach may be required. Once the mandible is accessed, the IAN is approached via lateral decortication techniques (Fig. 25-33).[211] Access to the ION can be accomplished transorally via a maxillary vestibular incision at the time of maxillary or zygomaticomaxillary complex fracture repair. Alternatively, a lower lid, transconjuctival, or subciliary incision may be more suitable and an osteotomy around the infraorbital canal may be required for nerve isolation.[39,210]

Dissection of the nerve from the surrounding tissue bed for inspection and further manipulation is the next

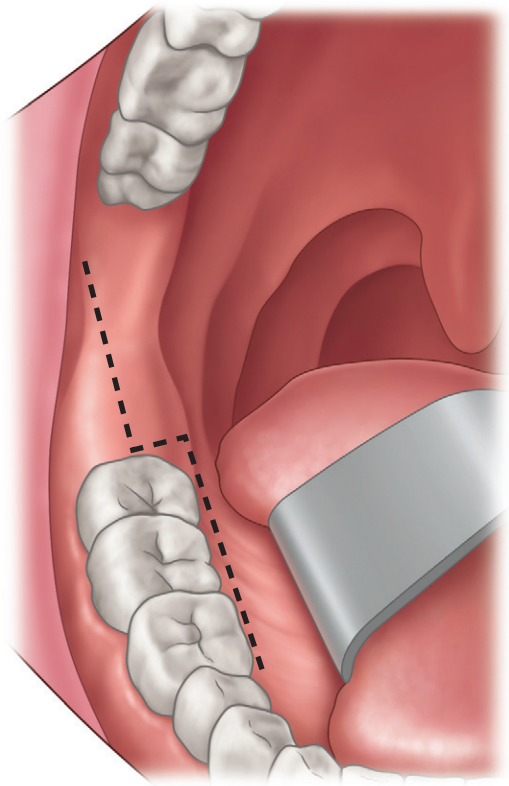


FIGURE 25-32 Diagram showing surgical access for lingual nerve repair using a lingual gingival sulcus approach with posterolateral extension.

step and is termed *external neurolysis* (EN). For the LN, this may involve microdissection of the neurovascular bundle from surrounding scar tissue, whereas for the IAN it may imply the need for decortication of the IAC and lateralization of the IAN. Separation of the nerve from scar tissue that may be responsible for restriction or compression leading to a conduction block and prevention of spontaneous recovery may be all that is required. In cases of short duration of symptoms (<6 months), or if nerve compression from scarring is less than 25% of the nerve diameter without neuroma formation, EN may provide definitive treatment.[129,209,212,213] EN is performed under loop magnification and, once completed, an examination of the nerve is performed and the need for addition intervention is determined. If foreign material or bone or tooth fragments are identified, they should be removed at this stage.

Several injuries may cause intraneural scarring that results in neurologic deficits while nerve continuity is maintained.[214] In such cases, internal neurolysis (IN) is indicated, which requires opening of the epineurium for fascicular examination. Because of the sparse amount of epineurium in the trigeminal nerve branches, this may lead to further fibrosis and iatrogenic scarring. Caution should be exercised when making the decision to proceed with IN because it may cause worsening of the condition.[129] Several types of IN are described based on the exact tissue manipulation performed: epifascicular epineurotomy, epifascicular, epineurectomy, and

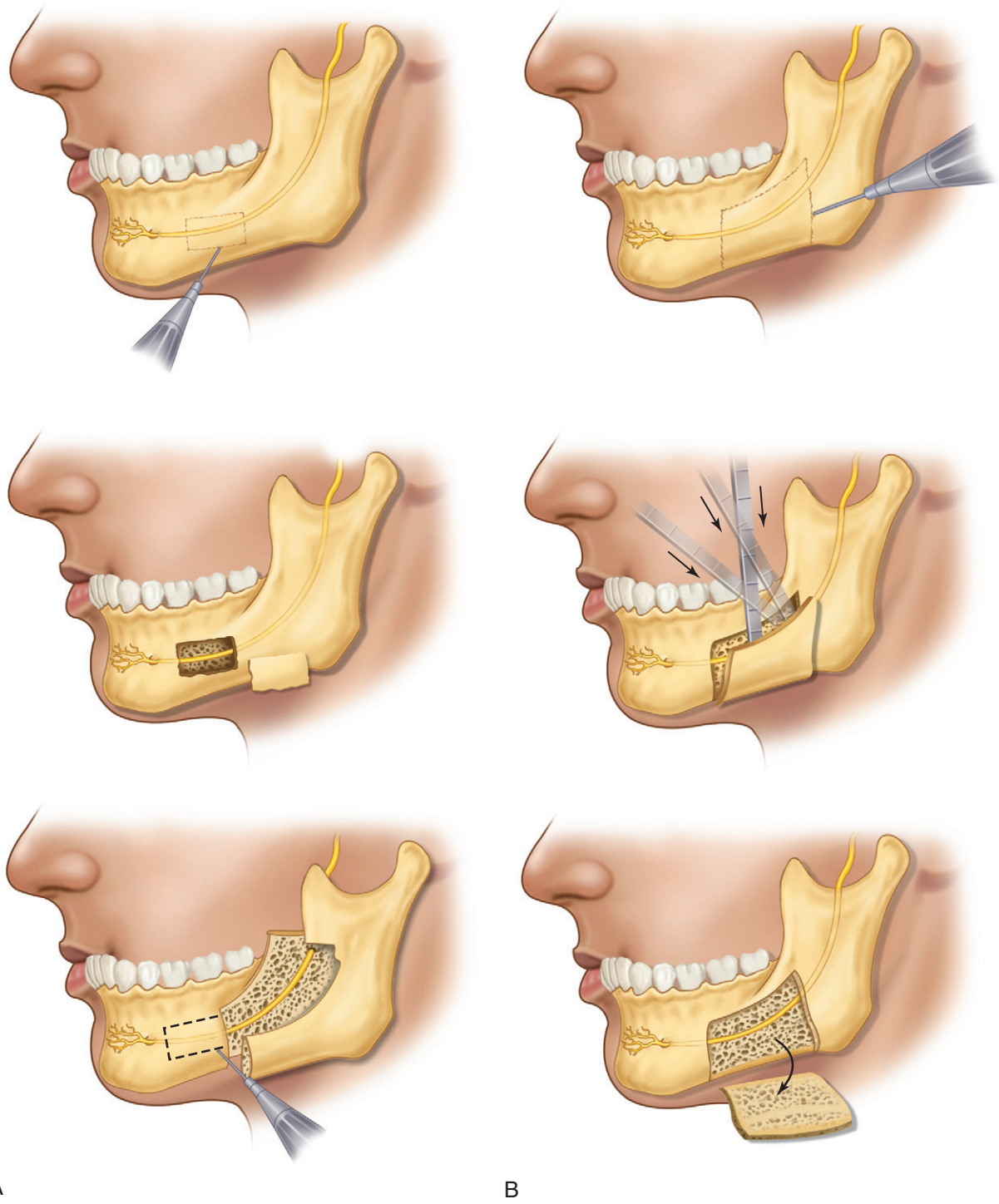


FIGURE 25-33 **A,** Diagram of techniques for inferior alveolar nerve access, including isolated decortication or a sagittal split osteotomy, with extension to the mental foramen if necessary. **B,** Diagram of technique described by Miloro in 1995 for wide access to the inferior alveolar nerve via a complete lateral decortication window that may be replaced to protect the nerve repair site.

intrafascicular epineurectomy that are of progressive complexity, involve tissue removal, and are of questionable benefit for the trigeminal nerve system.[208,209]

In cases of neuroma formation, excision followed by restoration of nerve continuity is required. Careful examination under magnification of the nerve stumps to be anastomosed is crucial so that healthy nerve tissues are repaired; scarred and necrotic nerve stumps are completely removed at 1.0-mm resection increments until normal tissue is encountered (Fig. 25-34). For a direct neurorrhaphy, epineurial suturing with three or four 7.0 or 8.0 nonreactive sutures (nylon) is adequate for the trigeminal nerve (Fig. 25-35).[215] Coaptation is the alignment of individual fascicles during direct repair, but this

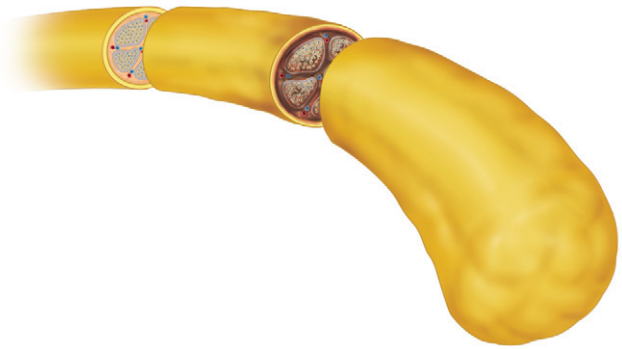


FIGURE 25-34 Diagram showing preparation of the nerve stump with serial 1.0-mm resections to remove scar tissue (neuroma) and ensure that normal healthy neural tissue is encountered prior to the neurorrhaphy procedure. Failure to débride the nerve stumps adequately will result in failure of neurosensory recovery.

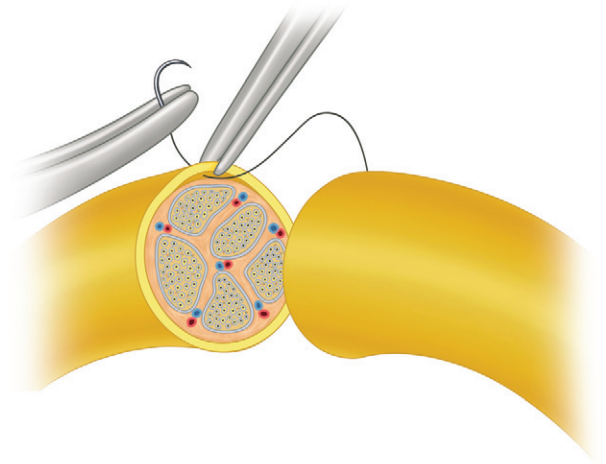


FIGURE 25-35 Diagram showing direct neurorrhaphy with epineurial sutures.

is difficult or impossible with a polyfascicular nerve, such as the trigeminal.[216,217] Nerve mobilization for tension-free closure is critical and may require dissection in proximal and distal directions (Fig. 25-36) or, additionally, in the case of the IAN, sacrifice of the terminal incisive branch to allow further release and mobilization of the IAN.[216]

Cases of nerve injury that result in nerve continuity defects, or prevent primary tension-free repair, will require indirect repair, with interpositional grafting (Fig. 25-37). Traumatic avulsive injuries, pathologic conditions involving the nerve, or extensive neuroma resection at the time of nerve repair are common reasons for these continuity defects of the nerve. When nerve mobilization is not adequate to allow tension-free primary repair, interpositional grafts may be required. The donor site selection for a nerve graft depends on several factors, including the diameter and fascicular pattern, correlation of neural function, and donor site morbidity. Usual donor sites for trigeminal nerve repair include the greater auricular and sural nerves because of ease of access and relatively low donor site morbidity, and anesthetic areas relatively tolerable.* In fact, the sural nerve may be the preferred autogenous nerve graft because it matches most closely with the diameter and fascicular pattern of the trigeminal nerve system and results in an area of minor donor site paresthesia (Fig. 25-38).[218] The greater auricular nerve diameter is almost 50% of that of the trigeminal nerve and therefore its usefulness may be limited to cable grafting (multiple grafts) for the trigeminal nerve. Also, the harvest would result in a facial scar as well as an area of deficit in the facial region that is less tolerable to patients than the donor site deficit of the lateral foot that results from sural nerve harvest.

*References 42, 129, 209, 210, and 217.

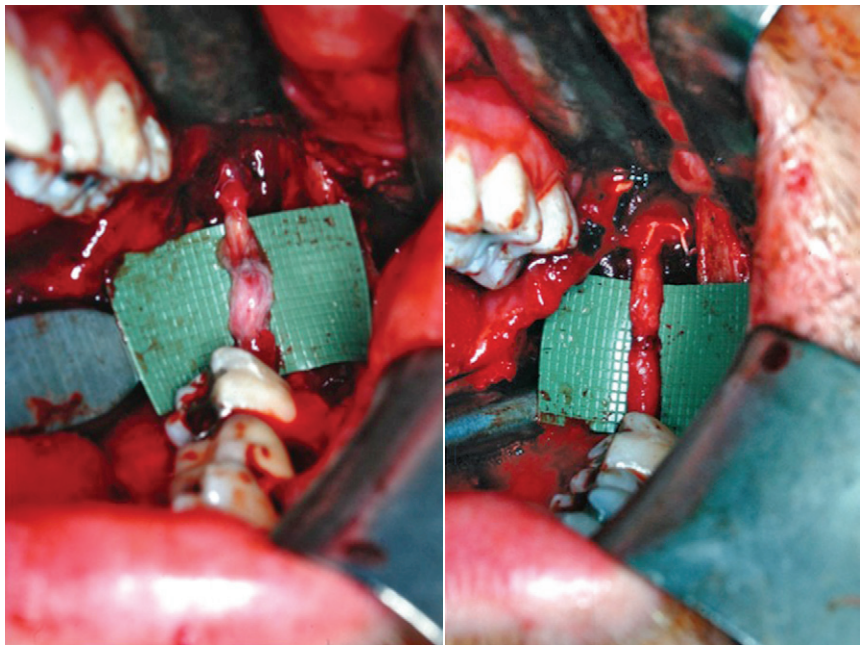


FIGURE 25-36 Clinical example of a left lingual nerve neuroma **(A)** and following resection of the neuroma and direct repair with epineurial sutures **(B).**

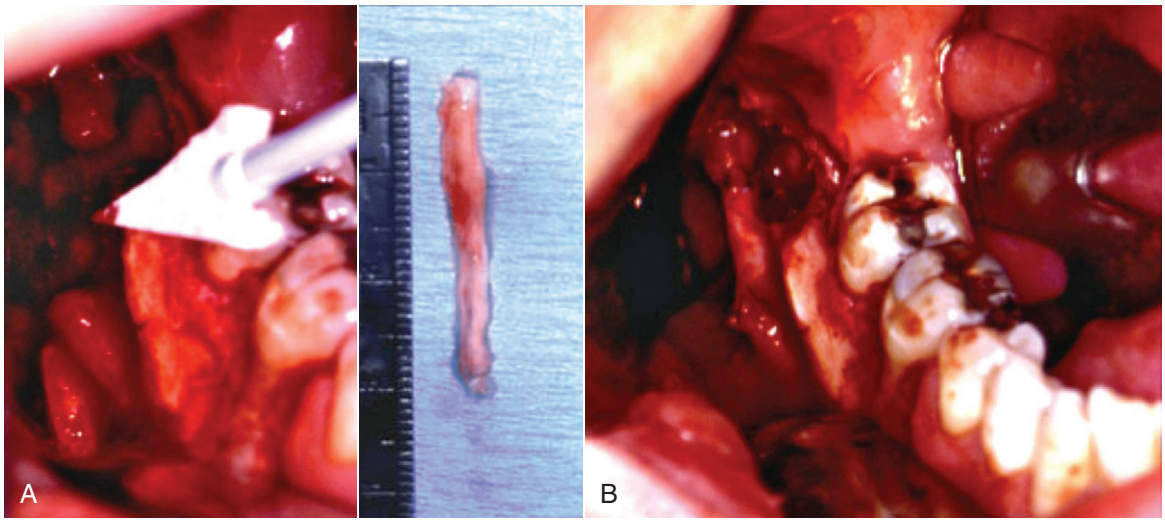


FIGURE 25-37 **A,** Clinical example of a discontinuity of the right inferior alveolar nerve following resection of a neuroma because of a third molar injury (arrowhead wexel sponge; *left*). Autogenous sural nerve graft, 3 cm *(right).* **B,** Indirect graft repair of the right inferior alveolar nerve continuity defect using the autogenous sural nerve graft with epineurial sutures.

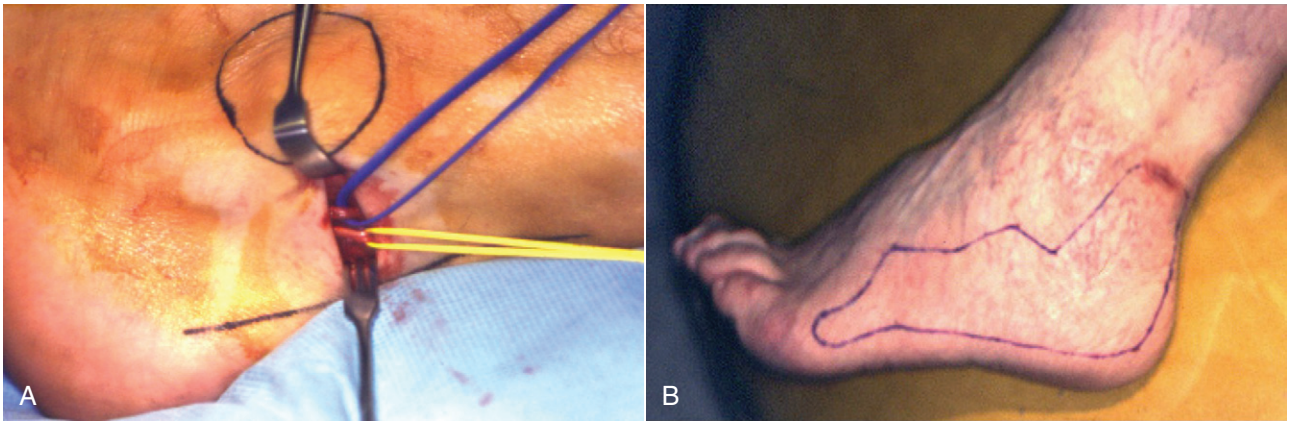


FIGURE 25-38 **A,** Clinical example of sural nerve harvest site posterior and superior to the lateral malleolus with identification of the lesser saphenous vein (anterior) and the sural nerve (posterior). **B,** Donor site deficit mapped with marker 1 week following sural nerve harvest. This area of paresthesia decreases significantly over time.

Alternatively, several other materials may be used for indirect neurorrhaphy via entubulation techniques, including alloplastic tubes (Silastic, expanded polytetrafluoroethylene, polyester, polyglycolic acid polymer), vein grafts,[219] and allogeneic nerve grafts (Fig. 25-39). The use of alloplastic tubes is attractive because it eliminates donor site morbidity. Unfortunately, there has been no consistency in the reported clinical outcomes with these entubulation techniques, and therefore this is not a standard technique for the management of trigeminal nerve injuries with continuity defects. Vein grafting, on the contrary, has been successfully used for this purpose, with results comparable to primary repairs, especially in defects smaller than 5 mm.[220-225] Following surgical intervention, periodic follow-up with repeat NSTs to evaluate the response to treatment is required, and sensory reeducation exercises may be used to augment spontaneous neurosensory recovery.

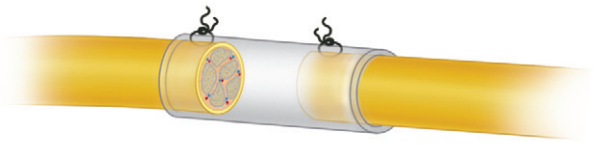


FIGURE 25-39 Diagram showing the principle of entubulation using a conduit to guide neural regeneration in cases of nerve discontinuity.

OUTCOMES OF TRIGEMINAL NERVE INJURY AND SURGICAL INTERVENTION

Injuries to the peripheral branches of the trigeminal nerve may be associated with impairment of speech, taste, mastication, and impact on quality of life. Long-term effects of these injuries are not well documented in

the literature. In a recent effort to elucidate on this subject, Pogrel et al[204] attempted to obtain information from patients several years following nerve injury via a telephone survey. Despite the poor 20% response rate, patients did report long-term difficulties with jobs, relationships, depression issues, speech, and eating problems. It was reported, however, that general improvement was noted by patients over time, with 8% describing complete recovery. It is unclear from this study if this represents true neurosensory recovery or adaptation to the altered status of the nerve, but the authors were in support of surgical intervention when indicated.

Based on the literature in this area, the success of microneurosurgical reconstruction of trigeminal nerve injuries could be estimated to be between 30% and 50% for all degrees of injury and types of reconstruction, with higher success rates achieved when primary repair is performed.[129,226-228] On the contrary, gap repair with an autogenous sural nerve graft may provide a better outcome because the graft itself provides a rich source of Schwann cells and neurotropic and neurotrophic factors to an area of injury that can assist in the degeneration and regeneration process of neural recovery.[218] Factors contributing to the variability of reported success rates relate to inconsistencies in documentation, standardization, and heterogeneity with regard to the cause of the injury, specific medical or surgical intervention, experience of the surgeon, timing of intervention, length of follow-up, NST methods used, method of reporting results, and criteria used to evaluate and define a successful outcome. In a retrospective review of outcomes in 51 patients who underwent microneurosurgical intervention, the following observations were reported: (1) repairs performed in less than 10 weeks (early repairs) from the injury appeared to do better than late ones; (2) improved sensation for IAN and LN can be achieved with microneurosurgery in select cases; and (3) carefully selected patients benefit from trigeminal nerve microneurosurgery.[226] There is a consensus that a need exists for more data in this area based on prospective randomized, multicenter clinical trials, with standardization in the diagnosis and management of trigeminal nerve injuries.

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PART FOUR

Special Considerations in the Management of Traumatic Injuries

CHAPTER 26

Anesthetic Considerations in the Acutely Injured Patient

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OUTLINE

Importance of the Airway in Maxillofacial Trauma
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Initial Assessment
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Mechanical Ventilation

Few situations are more challenging than the management of the acutely traumatized patient. It is the leading cause of death in individuals younger than 45 years.[1,2] Up to 75% of these deaths occur within hours of injury. Effective management requires cooperation and communication between a team of physicians.

The initial survey of the patient should occur immediately on arrival to the health care facility and must assess if the patient is in an imminent life-threatening condition. A systematic approach should be followed. The Advanced Trauma Life Support (ATLS) protocol describes a process of assessment and resuscitation that involves the evaluation of airway patency, breathing, circulation, and neurologic function. For the critically injured patient, the primary anesthetic responsibilities focus on assessing, maintaining, or obtaining an airway and providing respiratory support. If the injury warrants, surgical intervention will occur in the operating room. Anesthetic management in this phase of the patient's care will focus on drug selection to facilitate the surgical procedure. The anesthetic objective will be to achieve anxiolysis, analgesia, amnesia, and hemodynamic stability. The selection of drugs may be modified secondary to the injury and by the fact that resuscitation of the patient may be ongoing.

This chapter will focus on anesthetic and airway considerations in the management of the patient with oral and maxillofacial injuries. Many of the topics relevant to anesthetic trauma management are presented in the chapters pertaining to the assessment and evaluation of neurologic, thoracic, and abdominal injuries. This chapter will emphasize aspects that have significant anesthetic implications while minimizing repetition of the material presented elsewhere in this text.

IMPORTANCE OF THE AIRWAY IN MAXILLOFACIAL TRAUMA

The patient presenting with maxillofacial trauma presents a potentially complex and emergent scenario. Of significant concern is the susceptibility of the airway to injury in association with trauma to the head and neck. Airway narrowing or obstruction can result from edema, vascular congestion, or hemorrhage into the soft tissues or fascial spaces. These processes are dynamic and minimal; even an absence of early findings in the patient with significant trauma does not ensure that such problems will not occur or not worsen. Fracture of the skeletal complex may also result in airway narrowing and/or

distortion. This may be a direct result associated with the displacement of bony fragments or may be secondary to the instability of bony segments with an unfavorable effect on normal muscle function. Airway irritability may also occur secondary to hemorrhage within upper airway. This could contribute to airway obstruction, laryngospasm, or aspiration.

Maxillofacial trauma may also impede the ability to secure the airway. Trismus may occur secondary to edema, skeletal mechanical obstruction, or inflammation of the muscles of mastication. The cervical spine is susceptible to injury and, until an injury is ruled out, modifications in airway management will be dictated.

AIRWAY EXAMINATION AND MANAGEMENT

INITIAL ASSESSMENT

The initial airway survey occurs immediately and consists of making a gross observation. Is the patient breathing spontaneously? Does the patient have adequate exchange? A negative response to either of these questions will necessitate emergent intervention. The initial assessment should also determine the patient's adequacy of oxygenation. Observation for cyanosis may not be evident secondary to significant blood loss. Use of a pulse oximeter to determine oxygen saturation may be difficult for numerous reasons, including cold extremities, decreased perfusion, burns, and smoke inhalation. Centrally positioned probes should decrease the incidence of artifact associated with cold extremities and decreased peripheral perfusion. An oxygen saturation above 92% on room air confers some degree of safety, but this has to be interpreted based on the specific situation. For example, the patient with a decreased hematocrit may reflect a satisfactory oxygen saturation but in reality have a compromised oxygen content because of the diminished blood volume. Rapidly obtained initial laboratory studies, such as arterial blood gas (ABG) or venous blood gas (VBG), can provide data useful for evaluating the adequacy of ventilation and oxygenation. The astute clinician will also look for subtle changes, such as irritability and agitation, that may be early signs of hypoxia. Apprehension and restlessness may be suggestive of hypoxia as opposed to fear or anxiety caused by the ongoing events. As the hypoxia worsens, this may progress to deteriorating levels of altered consciousness, ultimately progressing to unconsciousness. Maxillofacial trauma patients who demonstrate an altered mental status, whether it be apprehension or confusion, must also be considered in the differential diagnosis to have a cerebral injury. The emergent need to manage the airway will preclude the ability to evaluate the patient fully and assess the extent of the head and neck injuries. Management of the airway will thus necessitate the assumption of concomitant injuries and dictate appropriate care.

If emergent intervention is not required, the examination must then identify injuries that may complicate or impede airway management. The examination must identify injuries that although not initially of an emergent matter, may potentially worsen and result in adverse respiratory consequences at some future point. The practitioner must also ensure that the examination focuses beyond the maxillofacial injury, determining whether the patient has other injuries that increase the risk of respiratory complications or would benefit from ventilatory assistance.

The complexity of managing the airway in the traumatized patient is that the decision to intubate may not be clearly defined. There are patients who, on admission, will appear to be ventilating and oxygenating adequately, but who will have less morbidity if the airway is controlled. Conversely, there are also patients in whom aggressive airway intervention on retrospect was contributory to an adverse event.

HISTORY

To provide optimal care, all patients should have a complete medical history and physical examination. If the patient is in acute distress, there frequently is inadequate time to perform such a complete history and physical examination. Emergent resuscitation and surgery may need to proceed with little or no background information. Whatever information is available should be obtained expeditiously. Information about the past medical history or the events surrounding the injury may be ascertained from the patient, family members, friends, or bystanders.

The emergency response personnel may also provide information about the events surrounding the traumatic event. Information about the mechanism of the injury may facilitate the diagnosis of associated clinical conditions. Information such as the death of another occupant of the vehicle may alert the practitioner to the potential for severe injuries that might otherwise go undetected. The report of the use of shoulder belts may alert the practitioner to blunt cervical injuries, such as a hematoma, that may displace the airway and produce pharyngeal and laryngeal congestion. A history of fire or smoke in a closed space will raise concern about the potential for airway injury.

Many trauma patients have major systemic diseases in addition to the injuries that currently brought them to the hospital. These systemic diseases may directly affect the patient's care. Cardiovascular, pulmonary, hepatic, or renal disease may decrease the patient's reserve and increase the patient's risk. Trauma patients frequently have histories of alcohol and illicit drug use. These drugs can cause various physiologic changes and interactions with anesthetic drugs. Their actions could affect preoperative, intraoperative, and postoperative care.

The trauma patient may also be pregnant. Pregnancy in the traumatized patient presents special problems. Changes in cardiovascular and respiratory parameters can alter the patient's response to anesthetic drugs. Additionally, many anesthetic drugs cross the placenta, having adverse effects on the fetus, and thus may be contraindicated.

An additional factor that needs to be considered during the intake visit is patient maturity. The child or mentally impaired patient poses a unique challenge. These individuals may lack the ability to cooperate. Techniques that may be selected for the typical adult patient may need to be foregone for safety purposes because they require a degree of patient cooperation.

PHYSICAL EXAMINATION

INJURIES AFFECTING THE AIRWAY

The anesthesiologist first inspects for injuries that decrease airway patency and impair the ability to ventilate and intubate the patient. Stridor, dyspnea, dysphonia, dysphagia, odynophagia, or gurgling may be suggestive of obstruction or swelling of the airway. Coughing or hemoptysis may be associated with significant hemorrhage and an inability to maintain a patent airway. A muffled voice, hoarseness, or inability to speak may be suggestive of a laryngeal fracture. Subcutaneous emphysema may be suggestive of a disruption of the airway. The physical examination of the maxillofacial region must identify congenital abnormalities in addition to the following: (1) displacement of teeth or oral appliances; (2) hemorrhage; (3) soft tissue edema; (4) maxillofacial fractures; (5) limited interincisal opening; (6) cervical spine injury, instability, or hypomobility; and (7) burns and smoke inhalation. An extended examination must assess for neurologic, ophthalmologic, thoracic, abdominal, and orthopedic injuries, all of which may affect the anesthetic decisions that are made.

The initial examination may be limited. Injuries may be missed during the primary assessment, both in the emergency department and operating room. Undiagnosed cervical spine injuries, pneumothorax, or thoracoabdominal vascular injuries may cause significant intraoperative or postoperative morbidity or mortality. A secondary assessment or reexamination of the patient, including a review of the radiographs and laboratory results before surgery if the patient is not immediately operated on, or postoperatively, has been shown to diagnose 90% of significant injuries missed during the initial examination.[3]

Maxillofacial Injury

Although unusual, teeth and/or associated structures may be aspirated. These foreign objects may become lodged in the supraglottic, glottic, or laryngeal area, or below the carina in the bronchi. A foreign object in the supraglottic, glottic, or laryngeal area may cause partial or complete obstruction. Immediate removal is indicated. General anesthesia should be considered for the patient with a partial obstruction, because anything that causes agitation of the patient could result in movement, resulting in a total obstruction. Alternatively, a foreign object that is aspirated into a bronchus is an urgent situation as opposed to an emergent situation. Maneuvers such as coughing should be avoided because they could dislodge the object so that it becomes wedged above the carina, causing a complete as opposed to a partial obstruction. The surgeon must also be cognizant that aspirated foreign objects are not always symptomatic. They frequently are not considered and may be only incidentally detected during laryngoscopy or evaluation of a chest radiograph, which should always be inspected for foreign material.

Maxillofacial injuries usually do not result in significant hemorrhage. However, in the traumatized patient, hemorrhage into the airway may increase airway irritability or cause laryngospasm, both of which impair ventilation. Impaired reflexes in the obtunded patient or significant hemorrhage may also lead to aspiration of blood. Patients are actually at more risk for aspirating blood than gastric contents. Vascular injury does not have to produce evident hemorrhage. A hematoma may cause a narrowing of the pharynx or superior-posterior displacement of the tongue, which will lead to partial or complete obstruction of the airway. The hematoma or edema may also cause vascular congestion that compounds the narrowing of the airway, contributing to respiratory distress. The edema or hematoma formation may not necessarily be noted extraorally. The swelling may also take several hours to become clinically significant and occlude the airway. An injury to the tongue represents an example of this, in which the extent of swelling may be unimpressive on initial examination, with delayed edema causing airway obstruction. Hemorrhage within soft tissue is more common with penetrating head and neck injuries as opposed to blunt injuries. Another consideration with soft tissue injuries is the potential distortion of the anatomy so that a mask fit cannot be satisfactorily achieved.

Certain fractures of the maxillofacial complex have the potential to compromise the airway. Mandibular fractures (e.g., bilateral parasymphyseal or condylar fractures) can result in posterior displacement of the tongue and paraglottic soft tissue, resulting in partial or complete respiratory obstruction. Establishment of a patent airway can usually be achieved by traction on the anterior mandibular segment and/or tongue, pulling either or both forward, away from the posterior pharyngeal wall. One method used to maintain anterior traction on the tongue is to place a heavy suture or towel clamp through the anterior midline of the tongue. The placement of the suture through the midline avoids injuring a vessel. Reducing the fracture using maxillomandibular fixation or interdental wires may also reduce the posterior displacement of the tongue and paraglottic soft tissue, alleviating the airway obstruction. The placement of a nasopharyngeal or oropharyngeal airway may also assist in establishing a patent airway when the tongue is displaced posteriorly.

Le Fort fractures produce a disruption of the pterygoid plates that may result in the posterior displacement of the maxilla. At the Le Fort I level, this may result in an anterior open bite but generally does not produce airway obstruction secondary to the skeletal displacement. The severity of the open bite, however, may cause difficulty in achieving a satisfactory mask fit. Posterior and downward displacement of a Le Fort III fracture may result in obstruction of the nasopharynx. The disruption of the skeletal stability, with its associated effect on the soft tissue of the pharynx, may contribute to obstruction during negative pressure inspiration.[4] Nasal fractures may also cause nasopharyngeal obstruction. The hemorrhage associated with a nasal fracture may be sufficient to result in airway irritability and aspiration of blood.

Aspects of the examination seek to identify injuries that may necessitate modifications in airway management. A restricted interincisal opening may be seen with mandibular and midface trauma. The restricted

movement may be secondary to soft tissue edema, muscle splinting, or mechanical bony obstruction. Frequently, it is secondary to pain and muscle splinting and should be alleviated on induction of anesthesia. An actual mechanical limitation may exist. A condylar or high subcondylar fracture of the mandible, the displacement of the zygomatic buttress or zygomatic arch so that either impinges on the path of the coronoid process of the mandible, or significant edema may limit mandibular opening and is not relieved with the induction of anesthesia.

Le Fort II and III fractures, along with naso-orbital-ethmoidal (NOE) fractures, may involve a fracture of the cribriform plate of the ethmoid bone or the medial wall of the orbit. The fracture of these bones creates the risk for the endotracheal tube to enter the cranium or orbit with nasal intubation. This is a relative contraindication to nasotracheal intubation until a cranial base fracture has been ruled out. There is also a potential risk for bacteria or air to be forced into the cranial cavity with positive-pressure ventilation.[5]

Cervical Spine Injury

Cervical spine injury must be suspected in any patient with a maxillofacial injury. The incidence of cervical injury in the presence of facial fractures has been reported to be 1% to 6%.[6-8] The higher percentage is among patients who were involved in a motor vehicle accident (MVA). The increased incidence probably is secondary to the forces sustained in the MVA and the associated hyperextension, hyperflexion, compression, and rotation of the cervical spine.[9] However, no definitive relationship has been found between the mechanism of injury and a specific maxillofacial or cervical spine injury.

In the conscious patient, cervical spine injury is unlikely if the patient subjectively is without pain or paresthesia and objectively is without deformity or tenderness to palpation in a neutral position and during flexion and extension.[10] If there is any positive finding, or if the patient is obtunded or not fully alert and able to concentrate, further investigation is warranted. The pediatric patient may be unable to provide reliable information and a lower threshold for further investigation is warranted. In the unconscious patient, flaccid areflexia, loss of rectal sphincter tone, diaphragmatic breathing, and bradycardia are suggestive of a cervical spine injury.

If further investigation is required, the diagnosis of a cervical injury is made radiographically. The examination must be able to detect fractures and ligamentous injuries. The examination must visualize all seven cervical vertebrae and the first thoracic vertebrae to be able to rule out a cervical injury. Fractures of the cervical spine are more reliably detected with a computed tomography (CT) scan compared with plain films (90% versus 58%), but ligamentous injuries are more reliably detected with plain films compared with CT scans (93% versus 54%).[11] Immobilization of the neck in a neutral position is required until a definitive diagnosis is established, with the understanding that the urgency of the resuscitation may preclude the ability to obtain a definitive diagnosis for several hours to days. The best method of immobilization is achieved with a combination of a rigid cervical collar, a cervical-head immobilizer, and a backboard.[12] Rigid cervical collars by themselves limit rotation and lateral movement to only 50% of normal and flexion and extension to only 30% of normal and do not provide adequate stabilization. An anesthetized or paralyzed patient should not remain on a backboard for a period exceeding 1 hour because of the risk for decubitus ulcers.

Respiratory complications are common with cervical spine injuries. The extent of the respiratory derangement is associated with the level of the injury to the cervical spine. Diaphragmatic paralysis occurs with injuries at C4 or above. This will necessitate ventilatory support. Although injuries at C5 or below spare the phrenic nerve and therefore diaphragmatic breathing, expiratory reserve may be adversely affected secondary to accessory muscle paresis. This may be further compounded by the respiratory depressant effects of alcohol and/or illicit drugs and any concomitant injuries to the brain, chest, or abdomen. Respiratory function may also be impaired because of pulmonary edema. This is secondary to pulmonary capillary damage and left ventricular dysfunction associated with the acute transient hypertension that frequently follows a spinal cord injury.

Cervical Airway Injury

Injuries to the cervical airway may result from blunt or penetrating trauma. The incidence of these injuries is low. Blunt trauma to the airway from a direct blow to the cervical airway or from severe flexion-extension injuries may result in a thyroid, cricoid, or laryngeal cartilage fracture, or laryngotracheal separation.[13] A fracture of the thyroid cartilage is frequently associated with edema. Stridor, dyspnea, dysphagia, odynophagia, or gurgling may be suggestive of swelling of the airway. A muffled voice, hoarseness, or inability to speak may also be suggestive of a laryngeal fracture. A fracture of the cricoid cartilage is less common. It is frequently associated with an injury to the recurrent laryngeal nerve. Injury to this nerve results in vocal cord paralysis. Vocal cord paralysis impairs the ability to protect the airway, which can lead to pulmonary aspiration. Although the incidence of a cricoid cartilage fracture is rarer, there is a mortality of 43% associated with this injury compared with 11% seen with a thyroid fracture.[14,15] Up to 70% of patients sustaining blunt airway trauma have a concurrent cervical spine injury.[16]

Thermal and Inhalation Injury

Thermal and/or inhalation injury to the airway must be suspected in any patient presenting with a history of exposure to fire or smoke. Injury to the respiratory system may extend from the mouth to the alveoli. The heat and noxious chemicals from exposure to fire or smoke can produce upper airway edema, resulting in airway obstruction, periglottic edema, which potentially impairs laryngeal function, resulting in compromised protection of the lower airway, chemical injury, resulting in impaired pulmonary gas exchange, and carbon monoxide and cyanide toxicity. Patients with facial burns or a history of being in an enclosed space with a fire or smoke have a high risk of developing airway damage.

When the patient presents with a risk of thermal or inhalation injury, the examination must include looking for signs such as facial burns, singed facial hair, and/or carbonaceous debris in the nasal or oral secretions. These may be the only indications of potential respiratory problems because some patients may not demonstrate respiratory dysfunction on initial presentation. However, approximately 15% to 30% of burn patients develop some degree of respiratory dysfunction.[17,18] Other early physical findings include wheezing, cough, dysphonia, and hoarseness. Upper airway edema, which may appear unremarkable initially, is an early contributing factor to respiratory distress. Within 2 hours, it may become notable, progressively worsening and necessitating intubation within 4 to 8 hours.[19] The pediatric patient's airway, with its relatively smaller diameter, is more susceptible to airway obstruction because any edema will have a more profound effect on airway patency. Late complications, such as parenchymal lung damage, may take several days to develop.

Periodic examination over the first 12 to 24 hours should be done to assess the severity of the injury. Fiber optic laryngoscopy and bronchoscopy will provide direct visualization of the periglottic region and lower airways, respectively. Serial ABGs and pulmonary function tests may also aid in assessing and differentiating upper from lower airway obstruction and pulmonary injury.

Carbon monoxide and cyanide are products of combustion; both cause tissue hypoxia. Hypoxia secondary to carbon monoxide toxicity occurs because carbon monoxide has a 250 times greater affinity for hemoglobin compared with oxygen. This displaces oxygen from its hemoglobin binding site and results in a lower oxygen-carrying capacity and lower blood oxygen content. The oxyhemoglobin dissociation curve is also shifted to the left. The leftward shift of the oxyhemoglobin dissociation curve results in less oxygen being released to the peripheral tissue.[20]

Monitoring oxygen saturation by pulse oximetry is inaccurate because these devices do not differentiate between oxyhemoglobin and carboxyhemoglobin.[21,22] Serum carboxyhemoglobin levels should be obtained to establish the actual level of carbon monoxide present. Carboxyhemoglobin levels less than 20% cause headache and possible confusion. Carboxyhemoglobin levels between 20% and 40% present symptoms that include nausea, vomiting, disorientation, and visual impairment. Levels between 40% and 60% result in agitation, hallucinations, and coma, whereas levels greater than 60% are fatal.[23] The half-life of carboxyhemoglobin is 4 hours. Oxygen therapy is indicated. The administration of 100% oxygen can shorten the half-life of carboxyhemoglobin to less than 1 hour. Hyperbaric oxygen (HBO) therapy has been recommended for carboxyhemoglobin levels of 30% or higher.[24] Patients with neurologic symptoms should be considered for HBO regardless of their carboxyhemoglobin level.

Cyanide interferes with mitochondrial cytochrome function, resulting in tissue hypoxia. Nonspecific neurologic findings, including agitation and coma, are seen with cyanide toxicity. Lactic acidosis is also found with cyanide toxicity because it occurs with carbon monoxide toxicity. However, elevated levels in the patient without a significant burn injury and with ventilatory correction are more suggestive of cyanide toxicity.[25] The mixed venous partial pressure of oxygen is also elevated in these patients. Cardiac rhythm disturbances are also not uncommon in these patients. Cyanide levels above 0.2 mg/liter are toxic and above 1 mg/liter are fatal.[26]

Cyanide is hepatically metabolized, with a half-life of less than 1 hour. Oxygen therapy, possibly with mechanical ventilation, is indicated and effective. HBO therapy may have its indications but, considering the relatively rapid elimination of cyanide, its practicality may be limited. Specific pharmacologic therapies for cyanide toxicity include sodium nitrate and thiosulfate. Thiosulfate increases the liver's ability to metabolize cyanide; sodium nitrate promotes the formation of cyanomethemoglobinemia.

INJURIES AFFECTING BREATHING

The traumatized patient may sustain multiple injuries. Of specific concern to the anesthesiologist are those injuries that affect respiratory function. Thoracic trauma resulting in rib fractures, pneumothorax, and pulmonary contusion potentially can result in respiratory dysfunction.

Thoracic Trauma Injury

Rib fractures may be single or multiple. Ventilation may be impaired because of splinting secondary to intense pain. Fractures of three adjacent ribs are referred to as a flail chest. This results in chest wall instability and is associated with paradoxical chest wall movement, contributing to respiratory insufficiency. Therapy entails pain relief with analgesic administration. Continuous epidural analgesia directed to the thoracic segments has been shown to be more beneficial than parenteral analgesics.[27] Respiratory insufficiency, despite analgesia, however, may require intubation and mechanical ventilation. Rib fractures may also produce pleural and abdominal injuries. Pleural injuries include a closed, open, and tension pneumothorax.

A small closed pneumothorax, in which the patient is not to be anesthetized with positive-pressure ventilation, may be treated with observation. Nitrous oxide, which is more soluble than oxygen and nitrogen, should not be administered because it will diffuse into the pleural cavity and significantly expand the pneumothorax. For the patient who will be intubated and ventilated with positive pressure during a surgical procedure, thoracostomy drainage should be performed. If not, the closed pneumothorax may be converted to a tension pneumothorax as air is forced into the pleural space. A tension pneumothorax presents an immediate threat to the patient's life. Classic signs of a tension pneumothorax include hypotension, neck vein distention, tracheal deviation, and diminished breath sounds on the affected side. In the acutely symptomatic patient, treatment involves the insertion of a 14-gauge catheter through the second intercostal space at the midclavicular line.

An open pneumothorax also is an immediate threat to the patient's life. The chest wound allows air to enter

the pleural cavity through the chest wall opening instead of through the airways during spontaneous ventilation. Lung expansion and ventilation are poor, with resultant hypoxia and hypercapnia. Treatment in the spontaneously breathing individual entails placing an occlusive dressing, which is taped on three sides, over the wound. This prevents air entry into the pleural cavity during inspiration but allows air egress during expiration. An occlusive dressing over the wound and a chest tube at another site should be used when positive-pressure ventilation is desired.

ANESTHETIC STRATEGIES

TRIAGE

The goal of triage is to classify patients into various categories, identifying the urgency and immediacy of treatment. In regard to airway management, traumatized patients are divided into two categories: (1) those who will require airway control as part of the resuscitation; and (2) those who will require semielective airway control at some point during hospitalization for surgical management of the injury. Airway management as part of resuscitation can be further categorized as emergent or urgent.

The patient categorized as requiring emergent airway control is apneic or has total or near-total airway obstruction. This category of patient should be identified during the primary survey and requires immediate establishment of a patent airway. The quickest technique to secure the airway artificially is required. The possibility of a cervical spine injury must be considered and appropriate interventions taken to prevent a new neurologic injury or an exacerbation of a preexisting neurologic injury. The initial intervention is to give positive-pressure ventilation by mask with 100% oxygen. The mouth is inspected and suctioned, with any foreign material removed before placing the mask. If ventilation is unsuccessful, a jaw thrust may be performed or an artificial airway may be inserted, which may facilitate mask ventilation. Placement of an airway is not without potential risk. An oropharyngeal airway may stimulate the pharynx and precipitate gagging and coughing, which may cause an increase in intracranial or intraocular pressure. The upper body movement that may be associated with the gagging and coughing may cause further insult to a cervical spine injury. Insertion of a nasopharyngeal airway must also be done cautiously in a patient suspected of having a base of skull fracture. It may also cause an epistaxis, which may further impair the airway. Distortion of the soft tissue and unstable mandibular or maxillary fractures may impair mask fit and compromise the ability to provide positive-pressure ventilation. The clinician should remain cognizant of the urgency of the situation. Brain damage begins to occur as soon as 4 to 6 minutes in the complete absence of inspired oxygen. If the attempt to ventilate the patient fails, laryngoscopy and intubation under direct vision can be tried. As the anesthesiologist is attempting the laryngoscopy and intubation, the trauma team should be preparing for a surgical airway. A laryngeal mask airway (LMA) may also be considered in the "cannot ventilate, cannot intubate" patient in select situations. Although contraindicated in a patient with a full stomach, because it does not protect the trachea against gastric regurgitation and pulmonary aspiration, it may provide a temporary airway and facilitate intubation. Importantly, the LMA protects the lower airway against aspiration of pharyngeal blood and secretions. Patients with maxillofacial injuries are more likely to aspirate blood associated with the traumatic injury than gastric contents.[28] Also, a large-gauge IV catheter (e.g., 12 or 14 gauge) can be used to provide a temporary airway by performing a transtracheal jet ventilation. The easiest site for needle insertion is the cricothyroid membrane, which may be difficult to locate depending on the nature of the maxillofacial trauma. This technique is not without risk and does require higher pressures to insufflate the lungs. However, this may provide the patient with sufficient oxygen until a more definitive airway can be established.

Patients who are in acute respiratory distress will also require emergent airway intervention. These patients may present with stridor, labored breathing, intercostal retractions, and tracheal tug. Despite being alert, these patients are fatigued. Also included are patients who are unconscious, even though they may have adequate ventilation. A primary purpose in intubating the unconscious and obtunded patient is to protect the airway, in addition to providing supplemental oxygen and supporting ventilation.

Patients requiring urgent airway control are those who present with conditions such as the following: airway narrowing secondary to hemorrhage, edema, or maxillofacial skeletal instability; airway irritability secondary to hemorrhage; or thermal or inhalational airway injury. These situations, although urgent, allow the practitioner to proceed in an organized and deliberate but nonemergent manner. Supplemental oxygen should be administered. A more complete medical history and physical examination may be obtained, including appropriate cervical spine radiographs. Alternative intubating techniques may be considered. Appropriate medications may be administered, such as those to minimize the risk of gastric aspiration. The extent of injury may necessitate CT imaging. Monitoring and accessibility of the patient may be less in these remote environments, with the potential for an adverse event. Consideration should be made to intubate a patient electively with occult, impending respiratory impairment before sending the patient for the CT scan.

BASIC PRINCIPLES OF MANAGEMENT

PATIENT POSITION

The first principle in any emergency protocol is maintaining or obtaining airway patency. Maxillofacial trauma may result in a notable disruption of soft and hard tissue, which may compromise the patency of the airway. Conscious patients will seek out a position that optimizes airway patency. When appropriate (e.g., the patient is not at risk for a cervical spine injury), allow the patient to remain in that position.

SEDATION AND ANALGESIA TECHNIQUES

Parenteral conscious sedation is capable of providing analgesia, anxiolysis, and amnesia. Although this can be done safely, overly deep sedation may allow relaxation of the upper airway musculature, leading to airway obstruction, depression of the hypoxic or hypercapnic respiratory drive, and obtundation of the protective airway reflexes. Maxillofacial soft and hard tissue injuries may increase the susceptibility to airway obstruction. The administration of analgesic and sedative medication to these patients may result in muscular relaxation so that the patient no longer compensates and airway patency is compromised.

Induction of Anesthesia

Pharmacologic management is frequently required in all but the severely obtunded or comatose patient. If the patient is hemodynamically stable, anesthesia may be safely induced in any number of ways (e.g., inhalational mask, benzodiazepine, or barbiturate), but propofol is frequently used. IV opioids and/or lidocaine are also advantageous in attenuating the stimulation that accompanies instrumentation of the airway and intubation. Preventing exaggerated hypertensive responses is important for numerous reasons, such as preventing dislodgment of a hemostatic clot, possibly resulting in a hematoma leading to airway narrowing, and increasing intracranial pressure. In the presence of hemodynamic instability, etomidate, benzodiazepine, or ketamine should be considered. Alternatively, smaller boluses of propofol given intermittently can also be used. The advantage of etomidate is that it has minimal cardiovascular depressant effects, specifically with regard to causing the least vasodilatory effects. The advantage of ketamine is that it increases sympathetic activity, which produces a modest increase in blood pressure and heart rate. However, in the traumatized patient with high sympathetic tone, ketamine may produce hypotension secondary to its direct myocardial depressant effect. Ketamine is contraindicated in the presence of head injury because it increases intracranial pressure.

A neuromuscular blocking agent is commonly administered in conjunction with the induction agents. This agent paralyzes the patient, enhancing the success and safety of intubation by decreasing patient movement and adrenergic stimulation. In the severely hypovolemic or obtunded patient, a neuromuscular blocking agent may be indicated without the administration of the sedative or induction agent. Neuromuscular blockade should not be used if there is a concern that the patient may not be able to be ventilated or intubated. If the anesthesiologist's concern is that of ventilation, mask ventilation should be attempted after sedative administration before administration of the neuromuscular blocking agent. If the anesthesiologist's concern is that of intubation, a partial laryngoscopy may be performed to determine whether intubation may be accomplished after paralysis.

Awake Endotracheal Intubation

Before using a general anesthetic, the practitioner must have the ability to control and protect the airway. Ideal objectives are that the patient: (1) be NPO; (2) have a normal mandibular opening; (3) have a stable skeletal framework; and (4) have minimal soft tissue edema and distortion. A negative answer to any of these factors will necessitate a modification from standard induction and intubation.

Intubation may be attempted in an awake patient who is not in respiratory distress. The awake patient has the ability to protect his or her airway against pulmonary aspiration and maintain spontaneous ventilations. Securing the airway while the patient is awake may be considered the safest technique when the initial evaluation of the patient suggests that ventilation or intubation may be extremely difficult or impossible once anesthesia has been induced. Awake intubation may also be considered as an alternative to rapid sequence intubation in the patient with a full stomach. The alternative to an awake intubation in this situation is to establish a surgical airway. The airway can be secured surgically under local anesthesia only or in combination with minimal or moderate sedation.

Success of an awake intubation is dependent on patient comfort and cooperation. This includes explaining the procedure to the patient. Sedation will improve patient comfort and minimize potentially detrimental hemodynamic fluctuations. Benzodiazepines and opioids are an appropriate combination for sedation and analgesia. Benzodiazepines provide anxiolysis, sedation, and amnesia. Opioids provide analgesia. Opioids should be used with caution because they have the greatest degree of respiratory depression for the amount of sedation gained. Also, when combined with benzodiazepines, the respiratory depressant effect of opioids is potentiated. Too deep a sedation may compromise the patient's ability to maintain airway patency, satisfactory ventilation, and oxygenation and protect the airway against pulmonary aspiration. The safety of this technique is that benzodiazepines and opioids have specific antagonists. Opting to administer benzodiazepines as the sole sedative agent can provide satisfactory operating conditions without the overt risk of opioid-induced respiratory depression.

Intubation in the awake individual can be a stimulating process. Sympathetic stimulation can result in increased central venous pressure, which may result in an elevation in intracranial and/or intraocular pressure. Furthermore, inadequate local anesthesia of the upper airway can drastically impair the ability to secure the airway safely. Anesthetizing the upper airway can improve patient comfort and lessen the stimulation associated with the procedure. The gag reflex can be effectively controlled with a nerve block of the lingual branch of the glossopharyngeal nerve and/or the superior laryngeal nerve.[29-31] Both these nerve blocks are relatively simple techniques. The glossopharyngeal nerve block requires the bilateral deposition of 1 to 2 mL of local anesthetic agent into the caudal portion of the tonsillar pillar. The superior laryngeal nerve block is accomplished by deposition of 1 to 2 mL of local anesthetic agent into the hyothyroid membrane. Local anesthetic agents can also be topically administered orally (as a swish) via a nebulizer or atomizer, or injected transtracheally. Transtracheal administration anesthetizes the mucosa of the trachea and upper airway. Anesthetizing

the airway while optimizing intubating conditions blunts the glottic and cough reflexes, increasing the patient's susceptibility to aspiration. However, by combining several local anesthetic administration techniques with moderate procedural sedation with a benzodiazepine alone, the airway can be safely and comfortably secured.

FACTORS AFFECTING ANESTHETIC STRATEGIES

Full Stomach

Traumatic injuries are not planned. Therefore, it is common for the patient to have recently consumed food. Patients with altered levels of consciousness from intracranial injuries, alcohol, or drug intoxication will have diminished protective airway reflexes. These patients may also be more prone to vomiting. Induction of anesthesia also results in the loss of protective reflexes; laryngoscopy and intubation may initiate regurgitation. Thus, the traumatized patient with a full stomach is at danger for pulmonary aspiration. The incidence of aspiration is approximately 5% for the patient with a full stomach during intubation for emergency surgery.[32]

The risk of complications due to aspiration is directly related to gastric acidity (pH < 2.5), gastric volume, and presence of particulate matter. The acidity of the gastric contents causes a chemical pneumonitis and the particulate matter can produce obstruction of the airway and inflammatory foreign body reactions. Larger volumes of aspirate can lead to further distribution of the offending material and widespread pulmonary damage. Fasting guidelines before inducing anesthesia for elective surgery diminish the risk of aspirating gastric contents. However, for the traumatized patient, injury, anxiety, and pain can delay gastric emptying time. Thus, the time interval for determining a patient's state of fasting is measured from the last time a patient ate to the time of injury. If emergent surgery is not indicated, it is most appropriate to delay surgery to facilitate gastric emptying. However, the risk of aspiration with induction of anesthesia can persist despite delaying surgery well beyond the traditional 6- to 8-hour fast, between the time of last food ingestion and induction of anesthesia.

Several adjunctive techniques and pharmacologic agents are available to decrease the risk and severity of aspiration. Insertion of a nasogastric or orogastric tube provides active evacuation of gastric contents. The gastric tube reduces gastric volume and gastric pressure but is not presumed to empty the stomach completely, especially of large particulate material. Once placed, the gastric tube can remain in place during induction of anesthesia because it is now thought that its presence neither compromises the integrity of cricoid pressure nor acts as a wick for the ascension of gastric contents into the pharynx.[33] Placement of a gastric tube stimulates the pharynx and may cause coughing and vomiting in the conscious patient. In the patient with a head or globe injury, this can increase intracranial or intraocular pressure. Other relative contraindications to the placement of a gastric tube include the following: (1) a Le Fort II or III fracture because a nasogastric tube could be placed intracranially; and (2) suspected esophageal perforation. Cricoid pressure (Sellick maneuver) consists of posterior pressure on the cricoid cartilage, causing temporary esophageal occlusion between the cricoid cartilage and cervical vertebrae. It has been shown to be effective in preventing gastric regurgitation and gastric insufflation during anesthetic induction and positive-pressure ventilation. However, it is not without risk in the traumatized patient. Cricoid pressure can theoretically displace a vertebra of the cervical spine, potentially damaging the spinal cord. The technique should be modified in the patient with a suspected cervical spine fracture by supporting the back of the neck with another hand.[34] Cricoid pressure is absolutely contraindicated in the patient with suspected laryngotracheal injury because it may produce complete airway occlusion.[35,36]

Patient position may also be of some benefit. In the event of regurgitation, the Trendelenburg position is gravitationally favorable in preventing aspiration. This position is generally inappropriate for the patient with a head injury. Alternatively, the concept of reverse Trendelenburg theorizes that the head-up position minimizes the incidence of passive regurgitation. However, active vomiting, which can occur in the traumatized patient, is not prevented and, in the event that the patient vomits, gravity promotes aspiration. This position is inappropriate for the hypovolemic or hypotensive patient.

Pharmacologic agents that may decrease the risk of aspiration are histamine-2 (H2) blocking agents, metoclopramide and sodium citrate. H2 blocking agents inhibit the secretion of gastric acid, resulting in an increase of the pH of the gastric contents. They require at least 1 hour to achieve this effect after the drugs have been administered IV. Metoclopramide promotes gastric emptying, reducing gastric volume. It also increases gastroesophageal sphincter tone. It requires at least 20 minutes, when administered IV, to demonstrate any benefit. Its effect may be diminished when opioids have been administered. Adverse effects associated with metoclopramide include hypotension and extrapyramidal effects. Sodium citrate is a monoparticulate antacid. It is effective within 15 minutes for raising intragastric pH.

Intracranial Injury

Head trauma is a contributing factor in approximately 40% of trauma-related deaths.[37] It is also a major cause of respiratory depression and long-term disability in the trauma patient. Morbidity and mortality occur as a result of primary brain injury (i.e., the damage produced to the brain by the original mechanical force) and secondary brain injury, which is usually caused by increased intracranial pressure, ischemia, acidosis, and hypoxia. Management consists of recognizing primary brain injury and preventing secondary brain injury. Prevention can be accomplished by providing oxygen delivery, supporting ventilation, and maintaining cerebral blood flow.

Emergent airway control is indicated to protect against aspiration, correct hypoxia, and support ventilation. In many communities with advanced medical services, prehospital intubation has been shown to improve the survival in patients with head injury.[38]

Cerebral blood flow (CBF) may be decreased in the head trauma patient as a result of an increase in intracranial pressure secondary to cerebral edema. Local CBF

is regulated by several factors including $Paco_2$. For every 1-mm Hg decrease in $Paco_2$, CBF decreases by 2% to 3%. This is important to understand in the management of these patients because a $Paco_2$ of 20 to 25 mm Hg will result in a CBF less than half of normal. Hyperventilation is a technique used to reduce intracranial pressure (ICP). Hyperventilation reduces ICP by constricting pial and cerebral arterioles. Most clinicians attempt to achieve a $Paco_2$ around 35 mm Hg, because excessively aggressive hyperventilation can further reduce blood flow to injured sites, causing ischemic brain injury with a worse outcome. Temporary hyperventilation to a $Paco_2$ of 30 mm Hg or below is reserved for temporary early control before intervention or in obvious signs of herniation.[39] The cerebrospinal effects of hyperventilation are relatively transient.

Barbiturates can be effective in reducing persistent ICP when other modalities fail. The mechanisms of action are related to cerebral vasoconstriction, reduction in cerebral metabolism and oxygen requirements, and reduction in free-radical damage to brain cells. Pentobarbital is administered with a loading dose of 5 to 10 mg/kg and continued as a drip of 1.5 mg/kg/hr until the ICP is below 15 to 20 mm Hg. Careful monitoring is necessary (mean arterial pressure [MAP] > 70 mm Hg, normovolemic pulmonary artery wedge pressure [PAWP] 8 to 12 mm Hg, and temperature > 32° C [90° F]) because hypothermia and barbiturate therapy can cause severe cardiac arrhythmias. Good results have been reported with lowering ICP; however, questions remain regarding improved outcomes and survival.

Although intubation is frequently indicated, the route in accomplishing it may be detrimental, because laryngoscopy and intubation can significantly increase ICP. Profound anesthesia and muscle relaxation will minimize the elevation in ICP. Opioids are beneficial in attenuating the sympathetic response to airway manipulation. Succinylcholine-induced fasciculation elevates ICP. Its rapid onset and short duration are advantageous for rapid sequence induction but its administration must be preceded by a defasciculating dose of a nondepolarizing muscle relaxant.[40] Ketamine is contraindicated because it will increase ICP.

Patients sustaining maxillofacial injuries not infrequently have a history of loss of consciousness and/or amnesia. These patients should not receive any medication that will alter their mental status, such as sedatives, analgesics, or selected antiemetics. Medications such as anticholinergics, which will induce papillary dilation, should also be avoided because they will alter the findings during a neurologic examination. In addition to a neurologic examination, these patients should undergo CT to detect abnormalities before using an anesthetic. The concern in patients with a closed head injury is their undetected decompensation while under an anesthetic. A patient with a negative head CT scan should be stable and not deteriorate while under anesthetic.[40]

TREATMENT OF SPECIFIC INJURIES

Cervical Spine Injury

The goal in the management of the patient with a cervical spine injury is to prevent a new neurologic injury or an exacerbation of a preexisting neurologic injury. Cervical hyperextension, flexion, and traction must be avoided when securing the airway. Attention must be given during all stages of airway management because basic airway maneuvers, such as the head tilt or chin lift, have been shown to produce disc space expansion.[41] Definitive airway protection is achieved with orotracheal intubation, blind nasotracheal intubation, fiberoptic intubation, or cricothyrotomy. The literature is equivocal as to which is the most optimal technique for securing the airway in the patient with a cervical spine injury.

Blind nasoendotracheal intubation has been advocated as a technique to limit neck movement. However, nasoendotracheal intubation is frequently facilitated by flexion and extension of the neck.

Pressure is also frequently applied to the anterior neck to position and stabilize the larynx. Pressure applied to the anterior neck has been shown to cause posterior subluxation at the fracture site.[41] Blind nasoendotracheal intubation is also associated with a moderate incidence of failure and can cause epistaxis. If an alternative intubation technique is required, the bleeding into the airway will decrease visualization and potentially cause laryngospasm or airway contamination.

Because of the potential for neck movement associated with blind nasoendotracheal intubation, laryngoscopy and oroendotracheal intubation with head and neck stabilization or fiber optic laryngoscopy are recommended for the conscious patient.

The success of fiberoptic laryngoscopy is dependent on the skill and experience of the anesthesiologist. Theoretically, there should be no neck movement. To improve the conditions for a successful intubation, the intensity of the room lights should be decreased. This will allow the intubating team to determine the position of the endoscope as the fiberoptic light is transilluminated through the anterior neck. Patient comfort and cooperation can also be improved by anesthetizing the upper airway. Difficulty with this technique in the traumatized patient can be secondary to distortion of normal anatomy and/or hemorrhage. Hemorrhage into the airway frequently contributes to intubation difficulty, despite the ability to irrigate and suction with the endoscope. An advantage with an awake intubation is that neurologic function can be demonstrated after the procedure.

When urgent airway intubation is mandated or an awake intubation is contraindicated, the recommended technique is oroendotracheal intubation with direct laryngoscopy.[42] The anterior portion of a rigid cervical collar impedes mouth opening and the application of cricoid pressure and therefore is frequently removed. Immobilization is accomplished with manual in-line stabilization, which is accomplished by placing the hands on both sides of the head, holding down the occiput, and preventing rotation and extension. Glottic visualization is frequently decreased compared with intubation without manual in-line stabilization, but intubation can be accomplished.[43] Due to the likelihood of impaired visualization during the laryngoscopy, intubating stylets or semirigid bougies can be implemented to facilitate successful passage of the endotracheal tube. If difficulty exists,

there are a number of devices and techniques that can facilitate intubation.[44] One advance in airway management has been the intubating laryngeal mask. However, pressure against the cervical vertebrae from the intubating laryngeal mask can potentially aggravate a cervical spine injury.[45]

Manual in-line stabilization does not limit complete cervical movement during airway manipulation. Some studies have shown it to cause distraction of the fracture.[46-48] Manual in-line stabilization is also least effective in limiting extension of the upper cervical spine, which may occur during laryngoscopy and mask ventilation. Patients with injuries to C1 and C2 might be more vulnerable to adverse effects arising from intubation.

Airway manipulation, unless the patient is completely obtunded, will require the administration of anesthetic medications. An induction or sedative agent establishes an anesthetic state that provides sedation and amnesia and blunts the hemodynamic response to intubation. The neuromuscular agent decreases patient movement and excessive adrenergic stimulation. The presence of a cervical spine injury may modify the selection and dosing of these medications. The patient with a cervical spine injury has a loss of sympathetic stimulation. A vascular vasodilation with a lack of compensatory tachycardia results in hypotension. Left ventricular dysfunction secondary to the cervical spine injury may also contribute to the hypotension.[49] This is compounded by the vasodilatory effects of the anesthetic medication and the unopposed vagal stimulation, which causes a bradycardia secondary to pharyngeal and tracheal stimulation, during laryngoscopy and intubation. An anticholinergic agent administered before airway manipulation can blunt the vagal stimulation. Decreased dosages and slow administration of sedative or induction agent may minimize the vasodilatory effects of the anesthetic medications. The patient's hemodynamic status may dictate the complete avoidance of an induction agent. A neuromuscular blocking agent may be indicated alone during these situations.

There is no ideal neuromuscular blocking agent. For rapid sequence intubation in the trauma patient, succinylcholine has been the agent of choice. Succinylcholine, however, has been associated with severe hyperkalemia if administered to the spinal cord–injured or burn patient. The severe hyperkalemia can result in ventricular dysrhythmias and cardiac arrest. This adverse effect associated with succinylcholine develops over the first week; therefore, succinylcholine can be used within the first 24 hours of a patient sustaining a cervical spine injury. Alternatively, most anesthesiologists would prefer to use a nondepolarizing neuromuscular blocking agent (NMBA). Unfortunately, there currently is no ultra–short-acting NMBA available on the U.S. market. Rocuronium is a NMBA capable of approximating the rapid onset (≈90 seconds) similar to that of succinylcholine. However, the larger dose of rocuronium required to produce rapid paralysis necessary for a rapid sequence induction lasts far longer than succinylcholine. A rapid sequence dose of rocuronium typically produces a prolonged paralysis in excess of 30 to 45 minutes, requiring at least partial neuromuscular return before reversal can safely be instituted.

Thermal and Inhalation Injury

The maxillofacial surgeon may be involved in the management of the burn patient at the time of initial injury and subsequently several months later for reconstructive surgery. The concerns regarding airway management are specific to those periods.

Anesthetically, the initial concern is assessment of current airway edema and prediction of subsequent airway edema as a result of the thermal insult. During the first 3 to 5 days, changes in vascular integrity occur in burn patients. This results in extravasation of fluid, which also contributes to the edema. Overaggressive fluid resuscitation during this period further compounds the airway edema. Careful and repetitive observation is required so that intervention is timely and appropriate. Because of the potential for periglottic edema, consideration should be given to early airway control and the use of a smaller than normal endotracheal tube. Added attention should be paid to securing the endotracheal tube in the patient with a facial burn. Reintubation after an unplanned extubation in the patient with massive facial and airway edema may be impossible and ultimately prove fatal. Carbon monoxide toxicity may also be a complicating factor during initial presentation. Treatment may involve HBO therapy. There are issues related to hyperbaric therapy in the burn patient that the trauma team must address. For the patient who is intubated, the endotracheal tube balloon must be filled with saline. If the patient is wheezing, there is an increased risk of pneumothorax.

The second phase of airway management involves the care that transpires during the several days to weeks after the acute inhalation injury. Factors contributing to respiratory insufficiency include diminished alveolar compliance and intrapulmonary shunting. Aggressive and frequent pulmonary toilet and humidification are required to prevent occlusion of the airway secondary to sloughing endobronchial debris and secretions. Bronchoscopy may be necessary to assist in pulmonary toilet. Pharmacologically, nebulized heparin and acetylcysteine may have some benefit.[50] Bronchospasm may also be a complication of aerosolized irritants from the inhalation injury. Management with beta agonists is effective. Chest wall burns may restrict chest wall compliance, resulting in respiratory compromise. Escharotomy of the chest and abdomen will be necessary to manage this problem.

There are a few specific modifications in airway management in regard to the burn patient during this period. Succinylcholine can cause a massive hyperkalemia, resulting in ventricular dysrhythmias and cardiac arrest, and is contraindicated in the patient with a burn exceeding more than 10% of total body surface. However, this effect does not occur for a few days and the use of succinylcholine, although preferable to avoid, is not contraindicated in the first 24 hours.[51] Succinylcholine should then be avoided for the next 2 years until after the burned skin has healed. A nondepolarizing muscle relaxant should be used to facilitate intubation. A transient resistance to the nondepolarizing NMBAs may occur between 1 and 6 weeks.[52] Pharmacologically, the management of these patients will be dictated by the hemodynamic status of

the patient. The burn patient commonly has significant pain and appropriate analgesic therapy should be ensured. The burn patient also commonly requires repeated surgical débridements. Ketamine is an excellent drug in that it maintains spontaneous ventilations, functional residual capacity, and protective laryngeal reflexes and provides analgesia into the postoperative period. This facilitates the repeated surgeries with the avoidance of repeated intubation.

Burn patients may need reconstructive surgery several months after the initial injury. There are two primary airway management issues at this time. The patient who has had prolonged intubation or tracheostomy during the initial resuscitation may have tracheal or subglottic stenosis.[53,54] The second issue pertains to the physical disfigurement, including microstomia and neck contractures that impede ventilatory and intubation efforts. Fiberoptic endoscopic intubation may be the technique of choice.

POSTOPERATIVE AIRWAY SUPPORT

POSTOPERATIVE SEDATION AND ANALGESIA IN THE INTENSIVE CARE UNIT

Sedative and analgesic medications are frequently prescribed to the postoperative trauma patient who is mechanically ventilated. These medications are prescribed to sedate the patient and achieve an optimal level of pain control to ameliorate the detrimental stress response.

There are several agents that have been used to sedate a mechanically ventilated patient. These include propofol, benzodiazepines, etomidate, and dexmedetomidine. In selecting an agent, the practitioner seeks to achieve sedation, amnesia, and analgesia without producing physiologic instability.

Propofol

Propofol is an alkyl phenol. It has sedative, hypnotic, and amnestic properties. Propofol is administered as a rapid bolus for induction, as a weight-based dose ranging from 1 to 2.5 mg/kg. It can also be administered as a continuous infusion, usually at a rate between 50 to 200 mcg/kg/min.[54a] It has rapid onset with rapid recovery after discontinuation of the drug.[55] Its clearance rate and minimal tendency for drug accumulation make it an ideal agent for sedating the intubated patient. Assessment of the patient, with the ability of the patient to respond to verbal commands, usually can be performed within 10 minutes of discontinuing the propofol infusion. Weaning from mechanical ventilation is most favorable with propofol compared with the benzodiazepines because of its rapid recovery, which is relatively independent of the duration of the infusion.[56]

Propofol is beneficial in the traumatized patient because it decreases cerebral metabolism and has been shown to improve outcome in traumatic brain-injured patients.[57] It is also beneficial in suppressing seizure activity.[58,59]

A propofol infusion in the postoperative patient is not without potential adverse effects. Propofol is formulated as a lipid emulsion. Triglyceride levels must be monitored for patients on long-term infusion. Patients must also be observed for pancreatitis, which may be associated with hypertriglyceridemia.[60,61]

Propofol is approved for use in patients older than 3 years. Case reports of fatal metabolic acidosis and cardiac failure, termed the *propofol infusion syndrome*, have been reported in children who have had prolonged propofol infusions to assist in optimizing conditions for mechanical ventilation.[62-64] A review by the U.S. Food and Drug Administration (FDA) has concluded that propofol is not directly linked to the metabolic acidosis and pediatric deaths.[65] Practitioners, however, should be aware of the risk of this reaction in children and limit the dose and duration of propofol infusion in these situations.

Benzodiazepines

Benzodiazepines are commonly prescribed to provide anxiolysis, sedation, and amnesia in intensive care unit (ICU) patients. Of the benzodiazepines, midazolam or lorazepam are frequently administered. Midazolam has a shorter duration of action. It also has minimal cardiovascular depressant effects. It is easily titratable to achieve an appropriate sedative and anxiolytic depth to assist with mechanical ventilation. Despite its relatively shorter duration of action compared with other benzodiazepines, midazolam when compared with propofol has been associated with a prolonged weaning time. Midazolam is metabolized by hepatic microsomal oxidation. The oxidative pathway is susceptible to many factors, including hepatic disease and numerous drug interactions. Lorazepam is often chosen to provide amnesia and sedation for patients requiring mechanical ventilation. It has a prolonged duration of action that reflects a high degree of protein binding and poor water solubility. As such, lorazepam is often dosed twice daily, which can be advantageous in the ICU setting. Also, it should be noted that lorazepam has no active metabolites.

Etomidate

Etomidate is an imidazole derivative. The advantage of etomidate for induction of anesthesia is that it has minimal cardiovascular depressant effects. However, etomidate has been associated with suppression of adrenal steroid synthesis when administered both as an induction agent and an infusion. This has resulted in increased mortality in the ICU patient.

Dexmedetomidine

Dexmedetomidine is an alpha-2 agonist. It has sedative, anxiolytic, and analgesic properties. Patients sedated with dexmedetomidine maintain respiratory function.[66] It is a unique drug in that patients sedated with dexmedetomidine are readily roused, more cooperative, and interactive when stimulated.[67] The drug is also advantageous in that it attenuates the response to intubation and extubation.[68] It is devoid of amnestic properties.

Dexmedetomidine infusion may be associated with adverse effects. Hypertension can occur with rapid IV administration, which is short-lasting. Hypotension and bradycardia may subsequently develop and are attributable to the inhibition of sympathetic activity in the central nervous system.[69]

MECHANICAL VENTILATION

There are a number of traumatized patients who will require airway support after surgery. The indications for maintaining control of the airway may be motivated by local factors, such as swelling or systemic factors, including an intracranial injury or cardiopulmonary injuries. The intubated patient will often require mechanical ventilation. Mechanical ventilation can provide full ventilatory support (e.g., control mode ventilation), in which the ventilator provides the required minute ventilation or partial ventilatory support (e.g., intermittent mandatory ventilation), in which the required minute ventilation is partially supported by the ventilator and partially achieved by spontaneous unassisted ventilation.

The decision as to when to extubate the patient may prove as challenging as the decision about when to intubate the patient. It will be dictated by local factors (e.g., airway patency) and systemic factors. Systemic factors revolve around the patient's ability to assume the full work of breathing. These are demonstrated by vital capacity and negative inspiratory pressure. Adequate nutritional support to the patient who has sustained a severe injury is important in being able to assist the patient and promote a favorable outcome.

SUMMARY

Maxillofacial trauma patients, with their associated injuries, present various challenges to the trauma team members. This chapter has discussed various anesthetic management techniques demonstrating the importance of a highly skilled anesthesiologist as a member of the trauma team. In rural hospitals, oral and maxillofacial surgeons, with their anesthetic and surgical knowledge of the airway, may function in multiple roles during resuscitation of the trauma patient.

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CHAPTER

27

Maxillofacial Ballistic and Missile Injuries

David B. Powers | Robert I. Delo

OUTLINE

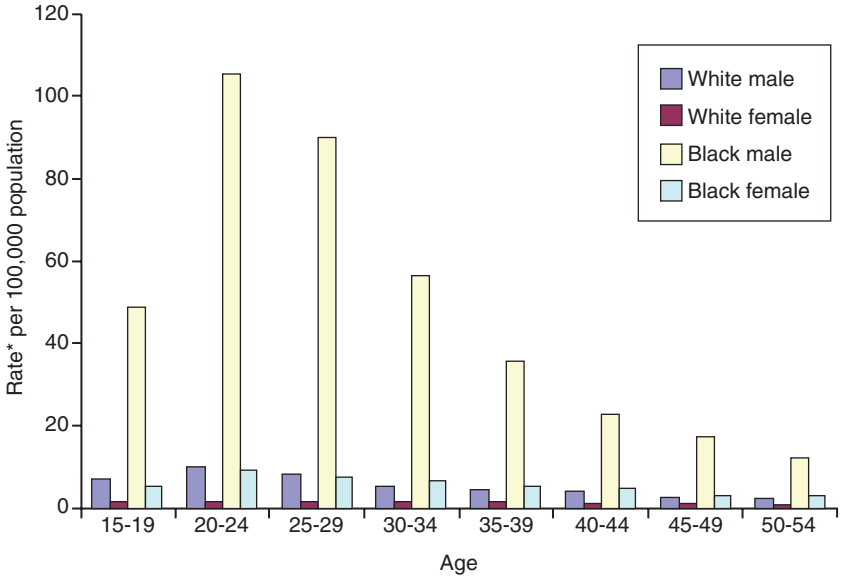
Epidemiology
Fatal and Nonfatal Firearm Injuries
Risk Factors for Firearm Injuries
Characteristics of Maxillofacial Ballistic and Missile Injuries
Categorization of Gunshot Injuries
Injury Patterns and Associated Injuries
Acute Care Considerations
Airway Management
Hemorrhage Management
Management of Gunshot Wounds to the Face
Soft and Hard Tissue Involvement
Bone and Soft Tissue Reconstruction
Postoperative Complications

Ballistic injury patterns to the maxillofacial region present a unique and challenging dilemma for the practicing craniomaxillofacial trauma surgeon. The tissue disruption associated with ballistic injury to the facial region can be daunting, and the identification of normal anatomic planes, usually embedded within a hemorrhaging mass of pulverized soft and hard tissues, can try the skills of even the most experienced facial trauma specialist. The soft tissue injuries inherent in ballistic trauma may exhibit avulsive loss, sequential necrosis over days to weeks, and compromised vascularity negating potential microvascular or pedicled soft tissue reconstruction, in addition to supplying proper nutrients to the underlying hard tissues of the facial skeleton, native bone or grafted bone, to promote healing. Due to the frequent occurrence of comminuted bony fractures, the necessity for open reduction of the hard tissue injuries further complicates the soft tissue response. A compromised soft tissue bed can lead to necrosis of free-floating bone fragments, avascular necrosis of the underlying facial skeleton, devitalization of stabilized fracture segments, and development of soft tissue infection or osteomyelitis, resulting in increased tissue loss and scarring of the facial composite. Hard tissue loss, including bone and teeth, presents the unique challenges of reconstruction, including reconstitution of the masticatory complex to support the oral intake of nutrition, reestablishment of the normal anteroposterior projection and angular shape of the facial skeleton, maintenance of lip competence, and control of salivation. Beyond the anatomic concerns of reconstruction, the presence of specialized vascular and neurosensory components in the maxillofacial region, including the great vessels of the neck, various branches of the cranial nerves compromising motor and sensory functions (e.g., sight, smell, hearing and taste) only serve further to complicate the potential for catastrophic injury and lifelong deformity that ballistic injuries cause to the craniomaxillofacial region.

Because the face is the component of the body most involved in a patient's personality and interaction with society, preservation of form, cosmesis, and functional outcome should remain the primary goals in the management of ballistic injury. A logical sequential analysis of the injury patterns to the facial complex is absolutely necessary for the treatment of craniomaxillofacial ballistic injuries. Fortunately, these skill sets should be well honed by all craniomaxillofacial surgeons through their exposure to generalized trauma, orthognathic, oncologic, and cosmetic surgery patients. Identification of injured tissues, understanding the functional limitations of these injuries, and preservation of hard and soft tissues, minimizing the need for tissue replacement, are paramount.

EPIDEMIOLOGY

The epidemiology of ballistic injuries to the maxillofacial region remains constant in the civilian community due to the historical and overriding presence of firearms in the United States. Although the presentation of catastrophic ballistic injuries typically is associated as being under the purview of the military surgeon during times of armed conflict, large metropolitan trauma centers, and many community and rural hospitals in the United States, routinely encounter patients injured during criminal acts, attempted suicide, or accidental shootings associated with recreational or hunting activities. By better understanding the causes, craniomaxillofacial surgeons are better prepared to serve as educators and advocates in the community for the elimination of these injuries.



* Age-adjusted rate per 100,000 U.S. Standard population based on year 2000 standard

FIGURE 27-1 Demographics of firearm homicides for ages 15-54 by race and gender in the United States, 2002. (Data from National Center for Injury Prevention and Control: WISQARS leading causes of death reports, 1999-2007 [http://webappa.cdc.gov/sasweb/ncipc/leadcaus10.html]).

FATAL AND NONFATAL FIREARM INJURIES

The most current comprehensive analysis of fatal and nonfatal firearm injuries in the United States was accomplished by Gotsch et al in 2001.[1] In excess of 115,000 firearm-related injuries occur annually in the United States, with firearms involved in over 58% of homicides and 57% of suicides. Approximately 30,000 of them sustained fatal wounds from these incidents, but more than twice this number survived their injury, many with significant injuries and permanent disabilities.[2] Coben, in 2003, reported the average hospital length of stay for a firearm-related wound was 6 days, with 7% of hospitalized firearm wound patients expiring from their injuries, and estimated hospital costs to be in excess of $800 million.[3] Because 29% of these patients were uninsured, and the estimated cost of firearm-related injuries per incident in the United States is in excess of $17,000 (over $2.3 billion aggregate lifetime medical costs for firearm injury survivors), the societal impact and payment implications for hospitals, local government, and taxpayers are immense.[4,5] Black males are disproportionately more likely to suffer firearm-related injuries than any other ethnic group or gender[1] (Fig. 27-1). According to Logan et al, the homicide rate for black males aged 10 to 19 years (1999 to 2002) was 17.8/100,000, a rate 10 times that of whites (1.8/100,000), three times that of Native Americans (6.0/100,000), and more than double the rate for Hispanics (8.0/100,000). [6] The Federal Bureau of Investigation (FBI) and Centers for Disease Control and Prevention (CDC) reported in 2003 that firearms were used in 66.9% of homicides in the United States, so the logical conclusion would be that the acute management of these conditions in the American health care system would be an expected event[7] (Fig. 27-2). In 2007, homicide was the sixth leading cause of death for blacks, regardless of age, while it ranked as the fifteenth leading cause for death when all races were included.[8] A 1999 report indicated that over 32,000 deaths resulted from firearm injuries in 1997, making it the second leading cause of death after motor vehicle accidents (MVAs). This article highlighted the positive efforts by the medical community to effect change in treatment protocols and the educational outreach programs by law enforcement, government, and the community to reduce the impact of firearms in the United States. There was a reduction in firearm fatalities since 1995 by 29.3% and the number of nonfatal injuries by 46.9%[9,10] (see Fig. 27-2*D* and *E*).

RISK FACTORS FOR FIREARM INJURIES

Unintended Firearm Injuries

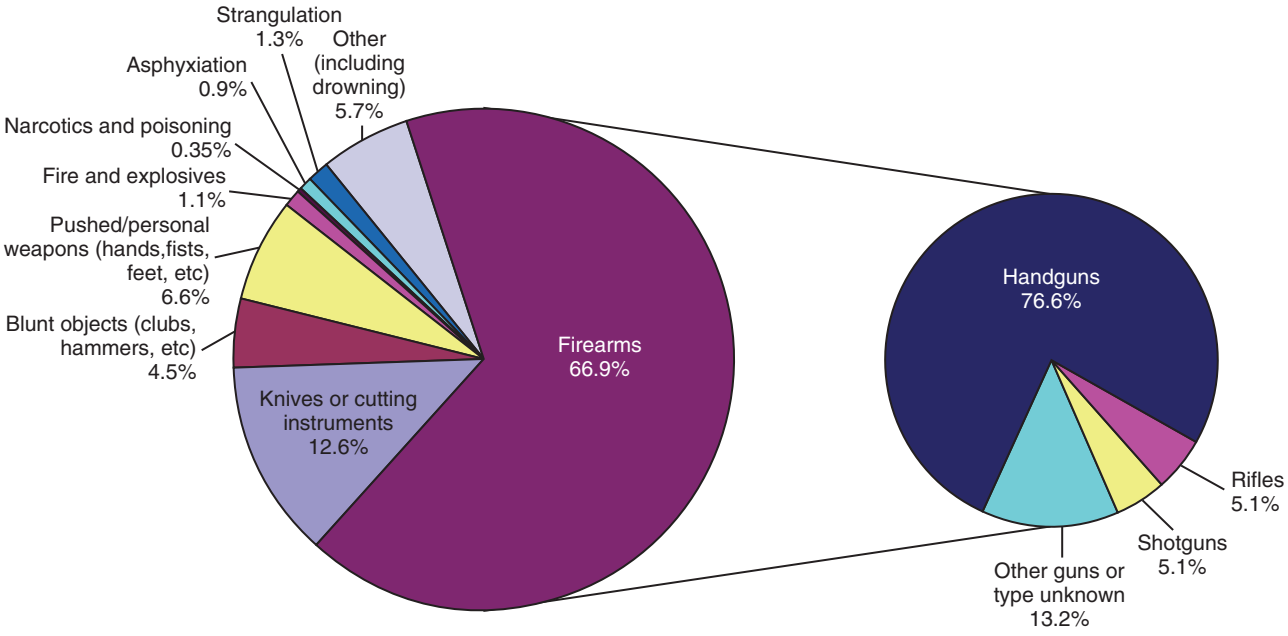
Unintentional firearm injuries and deaths represent a very small proportion of the total number of firearm injuries and have steadily declined since the 1930s.[11,12] Ismach et al evaluated unintended shootings in the Atlanta area from May 1996 through June 2000 in an effort to determine the proportion of accidental injuries that might be prevented by safer storage, handling, or firearm design.[13] Of the 216 unintentional firearm injuries recorded during this period, most victims were 15 to 34 years of age; 25% of the shootings involved victims younger than 18 years. Handguns were involved in 87% of the incidents. Of the 122 cases for which details of causation could be established, 74% of the incidents were associated with mishandling of the firearm, 14% were associated with child access to the firearm, and 32% were associated with perceived deficiencies in firearm design. It was concluded the incidence of unintended shootings could be decreased by improving the safety of firearm storage and limiting access, improving available firearm safety mechanisms, and educating owners about the safe handling of firearms, policies confirmed by additional studies conducted by Kochanek et al and the CDC.[8,14]

Pediatric Firearm Injuries

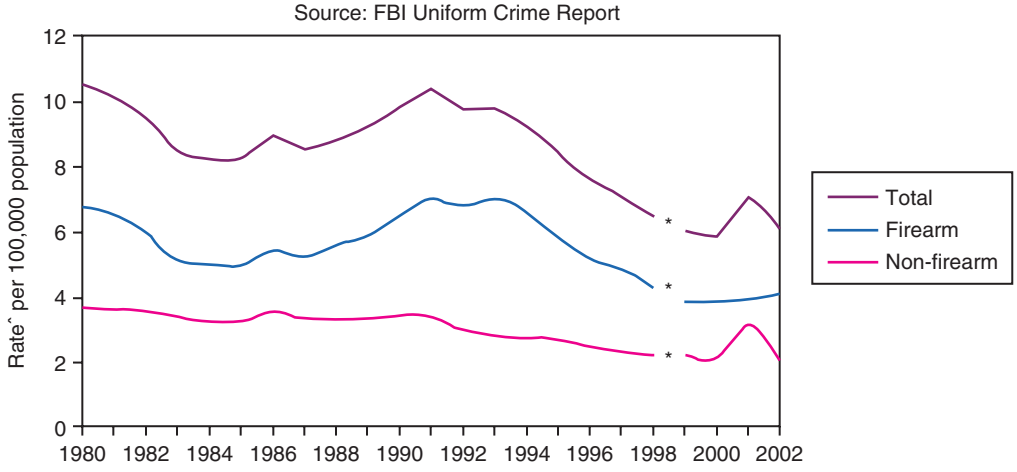
As noted by Kochanek et al, the pediatric population is at a disproportionately higher risk for firearm-related

A

United States Firearm Death Profile, 2003		
	Number	Rate*
Total firearm deaths	30,136	10.36
Suicides	16,907 (56.1%)	5.81
Homicides	11,920 (39.6%)	4.10
Unintentional	730 (2.4%)	0.25
Legal intervention	347 (1.2%)	0.12
Undetermined	232 (0.8%)	0.08
*Death rate per 100,000 population		



B



^Age-adjusted rates per 100,000 U.S. standard population year 2000 standard.
*Beginning in 1999, mortality data have been coded using ICD-10 codes. Graphs that include data from 1999 have a break in the trend line because NCHS has recommended not combining these data.

C

FIGURE 27-2 **A,** Firearm death rates in the United States by intent, 2003. **B,** Homicide weapons in the United States by type, 2003. **C,** Firearm and non-firearm homicide trends in the United States,1980-2002. Note the peak in the early 1990s followed by a steady trend downward. **D,** Top 10 leading causes of violence-related injury deaths in the United States, 2008. (**A, C,** Data from National Center for Injury Prevention and Control: WISQARS leading causes of death reports, 1999-2007 [http://webappa.cdc.gov/sasweb/ncipc/leadcaus10.html]; **B,** Data from Federal Bureau of Investigation: Crime in the United States, 2003 [http://www.fbi.gov/about-us/cjis/ ucr/crime-in-the-u.s/2003/toc03.pdf]; **D,** Data from National Center for Health Statistics: Top 10 leading causes of violence-related injury deaths in the United States, 2008, [http://nchspressroom.wordpress.com/2009/10/21/10-leading-causes-of-violence-related-injury-deaths-suicide-is-leading-killer].)

D

	Age Groups										
Rank	<1	1-4	5-9	10-14	15-24	25-34	35-44	45-54	55-64	65+	Total
1	Unintentional suffocation 1,058	Unintentional drowning 443	Unintentional MV traffic 385	Unintentional MV traffic 532	Unintentional MV traffic 8,647	Unintentional MV traffic 6,358	Unintentional poisoning 7,545	Unintentional poisoning 9,496	Unintentional MV traffic 4,137	Unintentional fall 19,742	Unintentional MV traffic 37,985
2	Homicide unspecified 156	Unintentional MV traffic 346	Unintentional drowning 138	Homicide firearm 143	Homicide firearm 4,394	Unintentional poisoning 5,946	Unintentional MV traffic 5,446	Unintentional MV traffic 5,866	Unintentional poisoning 3,547	Unintentional MV traffic 6,167	Unintentional poisoning 31,116
3	Homicide other spec., classifiable 98	Homicide unspecified 192	Unintentional fire/burn 111	Suicide suffocation 141	Unintentional poisoning 3,188	Homicide firearm 3,612	Suicide firearm 2,796	Suicide firearm 3,789	Suicide firearm 3,079	Unintentional unspecified 4,769	Unintentional fall 24,013
4	Unintentional MV traffic 98	Unintentional fire/burn 169	Homicide firearm 44	Unintentional drowning 123	Suicide firearm 2,009	Suicide firearm 2,357	Homicide firearm 1,966	Suicide poisoning 2,004	Unintentional fall 1,809	Suicide firearm 4,143	Suicide firearm 18,223
5	Undetermined suffocation 46	Unintentional suffocation 145	Unintentional suffocation 41	Unintentional fire/burn 64	Suicide suffocation 1,653	Suicide suffocation 1,752	Suicide suffocation 1,855	Suicide suffocation 1,772	Suicide poisoning 1,164	Unintentional suffocation 3,200	Homicide firearm 12,179
6	Unintentional drowning 41	Unintentional pedestrian, other 111	Unintentional other land transport 28	Unintentional other land transport 64	Unintentional drowning 569	Suicide poisoning 764	Suicide poisoning 1,486	Unintentional fall 1,300	Suicide suffocation 818	Adverse effects 1,677	Suicide suffocation 8,578
7	Homicide suffocation 32	Homicide other spec., classifiable 77	Unintentional pedestrian, other 24	Suicide firearm 50	Homicide cut/pierce 504	Undetermined poisoning 606	Undetermined poisoning 836	Homicide firearm 1,146	Unintentional suffocation 562	Unintentional poisoning 1,296	Suicide poisoning 6,442
8	Undetermined unspecified 28	Homicide firearm 56	Unintentional fall 22	Unintentional suffocation 50	Suicide poisoning 334	Homicide cut/pierce 476	Unintentional fall 540	Undetermined poisoning 1,066	Homicide firearm 489	Unintentional fire/burn 1,118	Unintentional suffocation 6,125
9	Adverse effects 24	Unintentional struck by or against 44	Homicide unspecified 15	Unintentional poisoning 37	Unintentional other land transport 302	Unintentional drowning 429	Unintentional drowning 406	Unintentional drowning 510	Unintentional fire/burn 476	Suicide poisoning 675	Unintentional unspecified 5,911
10	Unintentional fire/burn 22	Unintentional fall 38	Unintentional struck by or against 13	Unintentional firearm 29	Undetermined poisoning 299	Unintentional fall 297	Homicide cut/pierce 393	Unintentional suffocation 490	Undetermined poisoning 455	Suicide suffocation 580	Unintentional drowning 3,548

FIGURE 27-2, cont'd

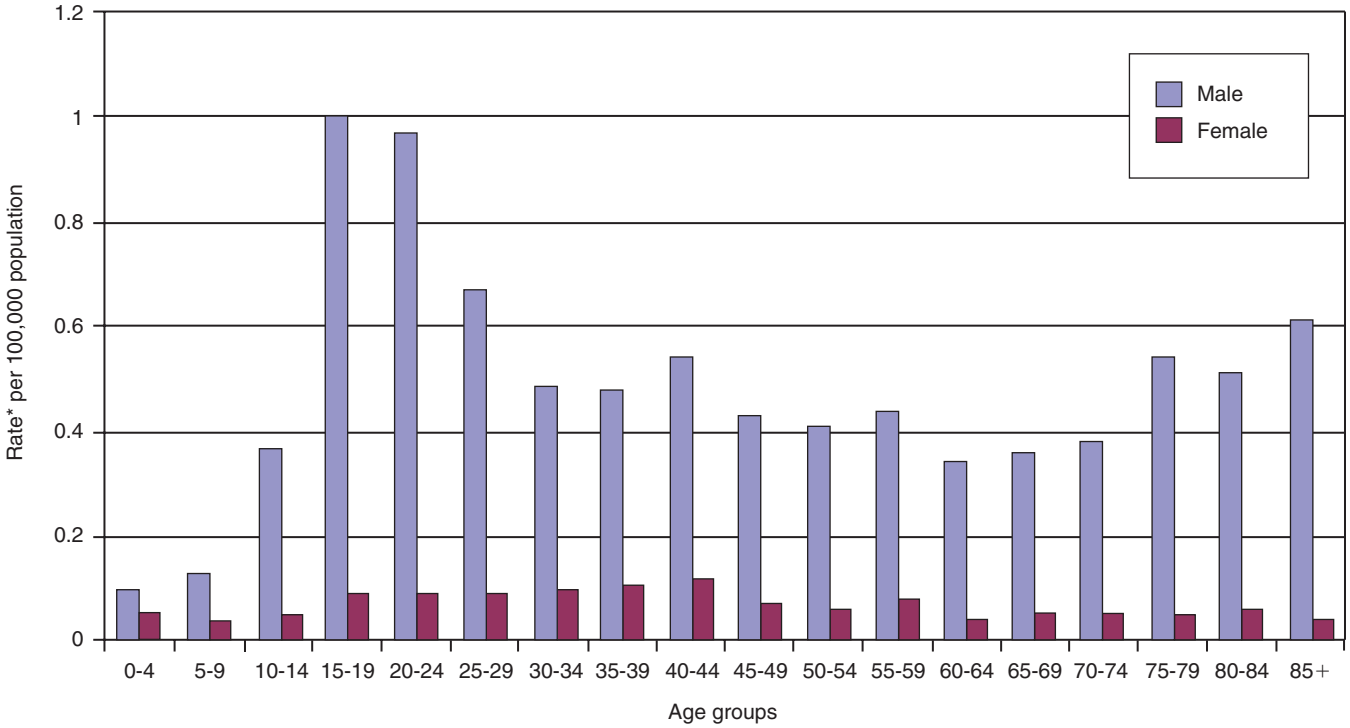


FIGURE 27-3 Unintentional firearm mortality by gender and age in the United States, 1999-2002. (Data from National Center for Injury Prevention and Control: WISQARS leading causes of death reports, 1999-2007 [http://webappa.cdc.gov/sasweb/ncipc/leadcaus10.html].)

injuries.[14] Paris et al have evaluated risk factors associated with nonfatal firearm injuries among inner city adolescents in the United States and found that children living with less than two parents, with frequent school absences, with a previous arrest history, and being black were factors placing them at increased risk for being the victim of firearm injury.[15] Nance et al have evaluated demographics and contrasting urban versus rural firearm injury trends for Pennsylvania and found that the rate of serious firearm injury in children was 10 times higher in urban areas than in rural areas.[16] Urban firearm injuries tended to be assaults, whereas rural injuries tended to be nonintentional. Of these injuries, 90.7% were in male adolescents, with an average age of 16.5 years. Unintended injuries predominated in children 5 to 9 years of age (61.4%), with 56.7% of these injuries occurring in rural areas. Handguns were the most commonly involved firearms, regardless of geographic region, age, or injury circumstances. Nance et al have appropriately that concluded that prevention strategies must be region- and situation-specific, taking these demographic variations into account.[16] Heninger and Hanzlick have reported similar results in 2008, with firearms being used in 88% of homicides and 61% of suicides in adolescents and teenagers in the Atlanta metropolitan area[17] (Fig. 27-3).

Alcohol and Drug Abuse

An intuitive and well-demonstrated risk factor for firearm-related injury is the use or abuse of alcohol or other recreational or prescription drugs. Madan et al have reported on the blood and urine analyses of 126 traumatically injured patients, regardless of the cause of injury, younger than 21 years, with 42% of them testing positive for alcohol or drugs.[18] When the grouping was narrowed to gunshot wounds in patients younger than 18 years, 72% of victims demonstrated evidence of substance use, with the 14- to 15-year old age group showing the highest association. Studies conducted by McLaughlin, Shuck, and Johnson et al have demonstrated an association between drug or alcohol use and the risk of firearm injury.[19-21]

Other Risk Factors

Although this is not a complete listing of all factors associated with an increased risk of firearm injury, known influences include the following[19-27]:

1. Previous or current involvement in the justice system
2. History of incarceration
3. Depression
4. Suicidal ideation or active psychiatric disease (Fig. 27-4)
5. Presence of firearms in the home
6. Ethnic minority status (particularly black)
7. Poverty or financial crisis
8. Presence of alcohol or recreation drugs

CHARACTERISTICS OF MAXILLOFACIAL BALLISTIC AND MISSILE INJURIES

The management of ballistic injuries to the craniomaxillofacial zone is predicated on a basic understanding of the two mechanisms whereby projectiles cause tissue

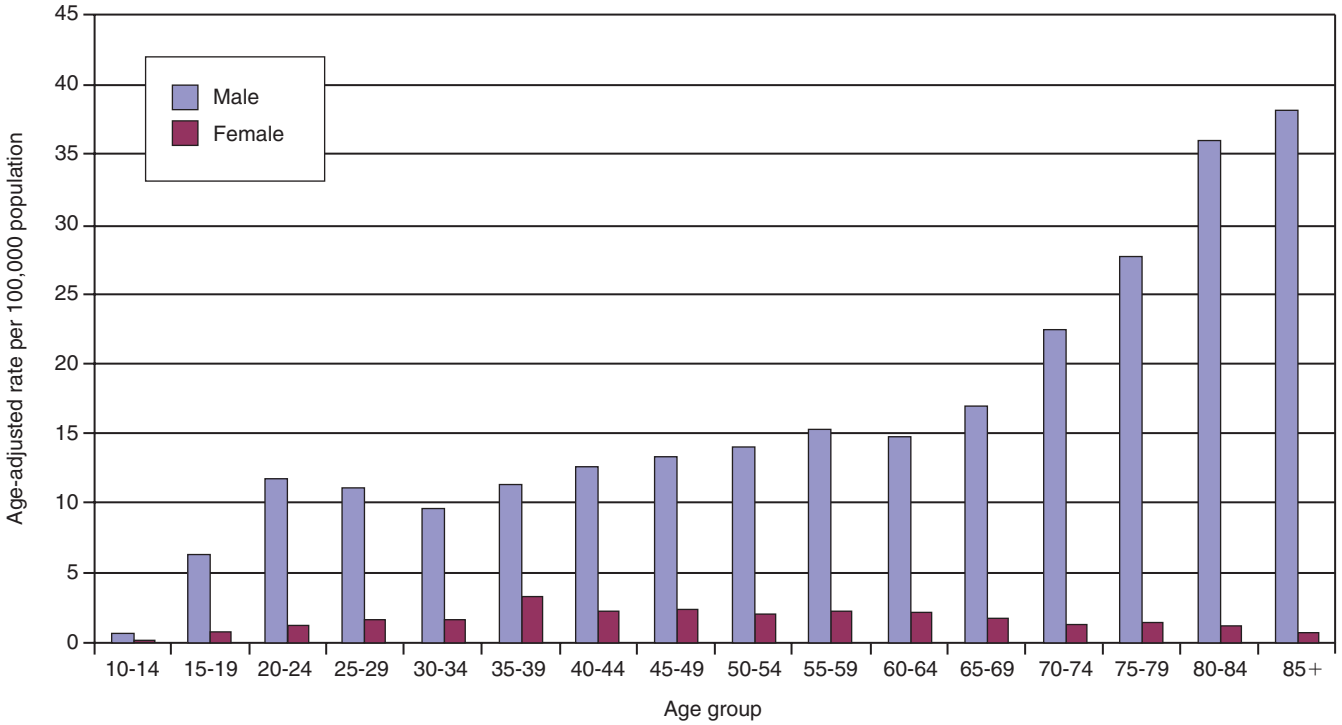


FIGURE 27-4 Firearm suicide by gender and age in the United States, 2002. (Data from National Center for Injury Prevention and Control: WISQARS leading causes of death reports, 1999-2007 [http://webappa.cdc.gov/sasweb/ncipc/leadcaus10.html).)

damage, crushing and stretching. As the recent conflicts in Iraq and Afghanistan have highlighted, missile injuries to the maxillofacial region may not only be caused by conventional weaponry, with a bullet fired from a cylindrical chamber, but devastating projectile collisions with the facial skeleton may be caused by massive bomb blasts and shock wave impacts from improvised explosive devices (IEDs).[28] Although a comprehensive analysis and discussion of the physics of energy transfer and ballistic theory is beyond the scope of this chapter, a brief review of the basic concepts of ballistics and kinetic energy is warranted.

CATEGORIZATION OF GUNSHOT INJURIES

Gunshot injuries have been categorized in the literature as penetrating, perforating, or avulsive.[10] Penetrating wounds are caused by the projectile striking the victim but not exiting the body. Perforating injuries have entrance and exit wounds, classically described as being without appreciable tissue loss. Avulsive injuries have entrance and exit wounds, generally presenting with an acute loss of tissue associated with passage of the projectile out of the victim. Rifles, handguns, and submachine guns have rifled barrels—essentially, spiral grooves cut into the length of the interior of the bore of the barrel.[29] The grooves are separated by segments of metal, called lands, which project into the middle of the barrel. The diameter of the barrel measured between the lands represents the caliber of the projectile.

Caliber specifications based on nomenclature used in the United States can be difficult to comprehend. The .30-06 and Winchester .308 cartridges are both loaded with bullets that have a diameter of .308 inches.[29] The "06" in this term describes the year, 1906, when the cartridge was introduced to the market. The term *grains* originally was applied to black powder charges and refers to the weight of the powder in the cartridge, not the number of granules contained in the cartridge case. A .30-30 cartridge has a .308-inch diameter bullet propelled by 30 grains of smokeless powder. As newer forms of gunpowder were developed, this powder charge was no longer used, but the terminology persists to this day. Additional confusion regarding caliber exists because North Atlantic Treaty Organization (NATO) and United States military projectiles are described using the metric system (7.62- or 9-mm rounds), while United States civilian firearm munitions are generally referred to in measurements relating to inches (.357 or .38). Although the question regarding caliber is commonly asked by medical personnel in the management of ballistic injury, the reality is that caliber has minimal practical impact on the care of the patient.[30-32]

The terms *high velocity* and *low velocity* as they relate to projectiles can be somewhat misleading. There is no consensus between United States and European research in the literature, with varying definitions correlating to where the study was performed. The U.S. literature designates high velocity as being between 2000 and 3000 feet/second (610 to 914 m/second), whereas studies from the United Kingdom designate the line between low- and high-velocity projectiles as being 1100 feet/second (335 m/second), which is the speed of sound in air.[33] The earliest recognized entry of high-velocity projectiles having an association with increased wounding potential occurred during the Vietnam War. In 1967, Rich et al have reported that bullets fired from the M16

rifle inflict tremendous tissue destruction and injuries on enemy combatants.[34] The muzzle velocity of the projectile shot from the M16 was 3100 feet/second. When coupled with erroneous information published by Rybeck in 1974, and in the 1975 edition of the *Emergency War Surgery* manual regarding the size of the temporary cavity caused by the missile, this information led to the common misperception that high-velocity projectiles cause more significant injuries.[35]

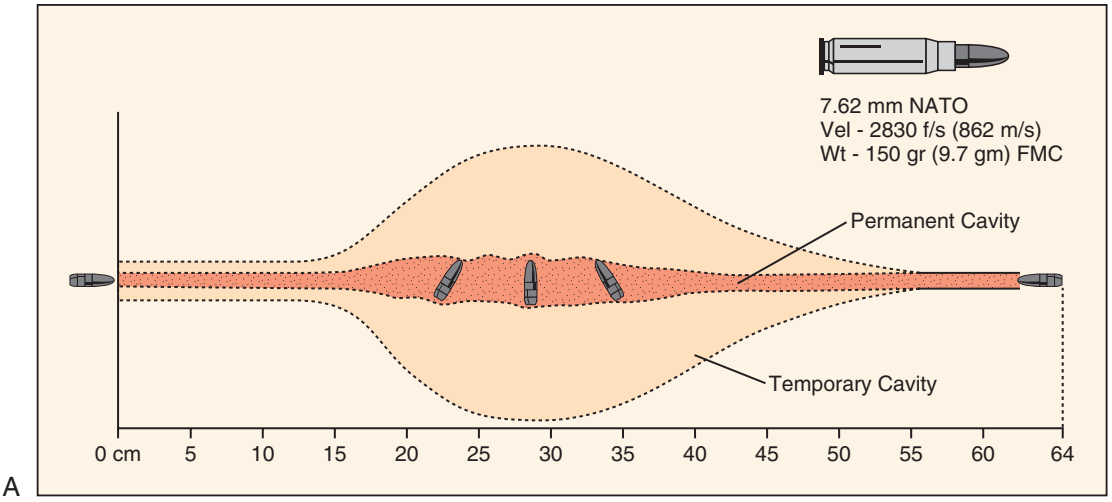
Part of the confusion regarding the wounding potential of high-velocity projectiles is becsause of misinterpretation of ballistic gelatin model studies. Ballistic gelatin is 10% to 20% gelatin refrigerated to 4° to 10° C (39° to 50° F) and is used as the tissue model for ballistic studies.[36,37] The wound profile diagrams included in this chapter represent the findings of these studies. The validity of the ballistic gelatin model has been confirmed by comparison with human autopsies, although there is confusion in correlating these studies to living patients, because the human body is more resistant to deformation than gelatin The effects of skin resistance, clothing, and opposition to separation of the fascial planes cannot be replicated in gelatin.[38,39]

Harvey et al evaluated the two types of pressure waves produced by penetrating objects in 1947, the sonic pressure wave and temporary cavity.[40] The first wave is the sonic pressure wave, sometimes referred to as the shock wave. This relates the sound of the projectile striking the target. This wave transmits at the speed of sound (i.e., ≈4750 feet/second [1450 m/second]) and is traveling considerably faster than the projectile entering the target. No temporary cavity is formed with the sonic pressure wave; in that regard it is analogous to the lithotripsy devices used for renal calculi destruction, with corresponding minimal risks for tissue injury.[31] Although American and Swedish researchers have tried to disprove Harvey's conclusions, no definitive evidence suggests that his findings are in error, and additional studies by French and American researchers have supported the original findings.[32,41-45]

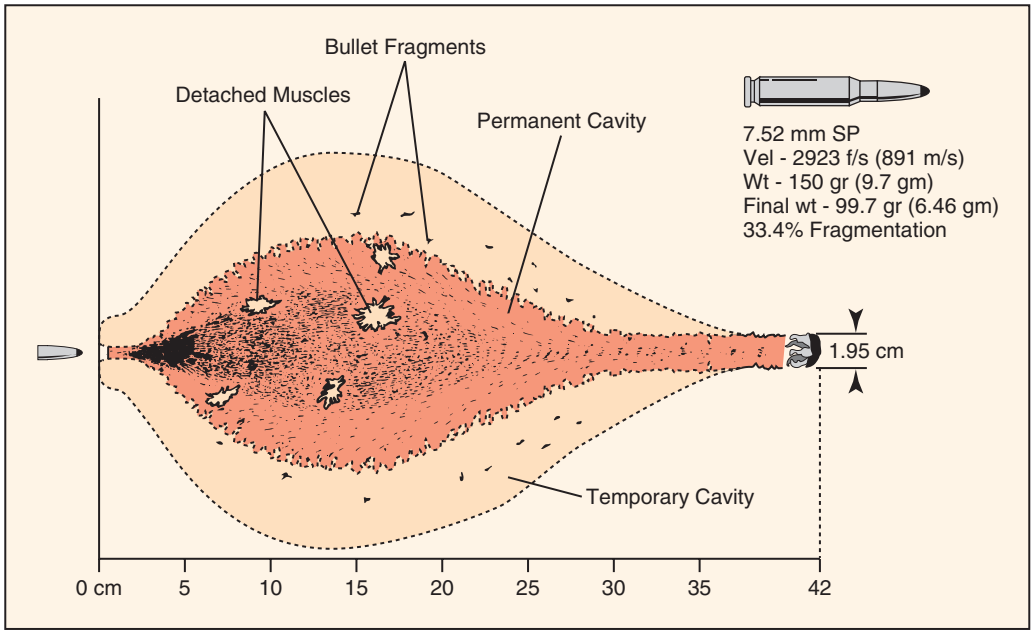
The secondary pressure wave, referred to as the temporary cavity, is formed when the penetrating projectile strikes tissue and the wave radiates away laterally away from the permanent cavity of the projectile path. After being struck by the projectile, the ballistic gelatin or tissue displays an obvious temporary cavity, which potentially injures tissues such as muscle, vessels, and organs. The clinical significance of this cavity is variable, with no real consensus in the literature. The temporary cavity caused by the M16 in animal laboratory models is much smaller than the approximately 18-cm temporary cavity seen in ballistic gelatin.[30] Dog models have indicated that acute tissue injury secondary to temporary cavity formation sustained with high-velocity projectile strikes were no more than 5 cm and were able to resolve within 72 hours.[46,47] The U.S. military has conducted extensive research into the wounding patterns of projectiles (see Fig. 27-5). The unique anatomic differences of the craniomaxillofacial skeleton, a relatively thin soft tissue layer overlying a dense foundation of bone, mitigate some of the expected responses of the temporary tissue stretch, because the overall thickness of the soft tissue envelope is generally less than the required total distance needing to be traveled prior to exhibiting secondary cavitation. Although sequential soft tissue necrosis and small vessel damage can occur, it is much more likely to be in response to the exaggerated permanent cavity of the projectile, which is greatly enhanced after striking the underlying facial skeleton.[31-33,48] The key point for understanding the management of ballistic injuries is the permanent cavity, which involves all the tissues that are pushed aside or destroyed during the flight of the projectile, and is the site of the extent of the initial, or immediate, damage. The size and shape of the permanent cavity are determined by the density and anatomic characteristics of the tissue lying in the projectile's path, velocity of the projectile, shape and characteristics of the projectile and, likely most importantly, the degree of deformation of the missile as it travels through the tissues.[36-39]

The type of firearm used has implications in regard to the wounding potential of the projectile. Generalized discussions of craniomaxillofacial wounding patterns center on handguns, rifles and shotguns. The wounding patterns of these weapons are unique and do not fall under the classic description of firearm-based injuries.[28] The actual projectiles expelled by firearms are limited in type only by time and the imagination for description, such as hollow point, round nose, full metal jacket, alloy-jacketed, and Teflon-tipped (Fig. 27-6*A*). The components of a bullet provide a basic understanding of the principles of firearm injury (Fig. 27-6*B*). The projectile

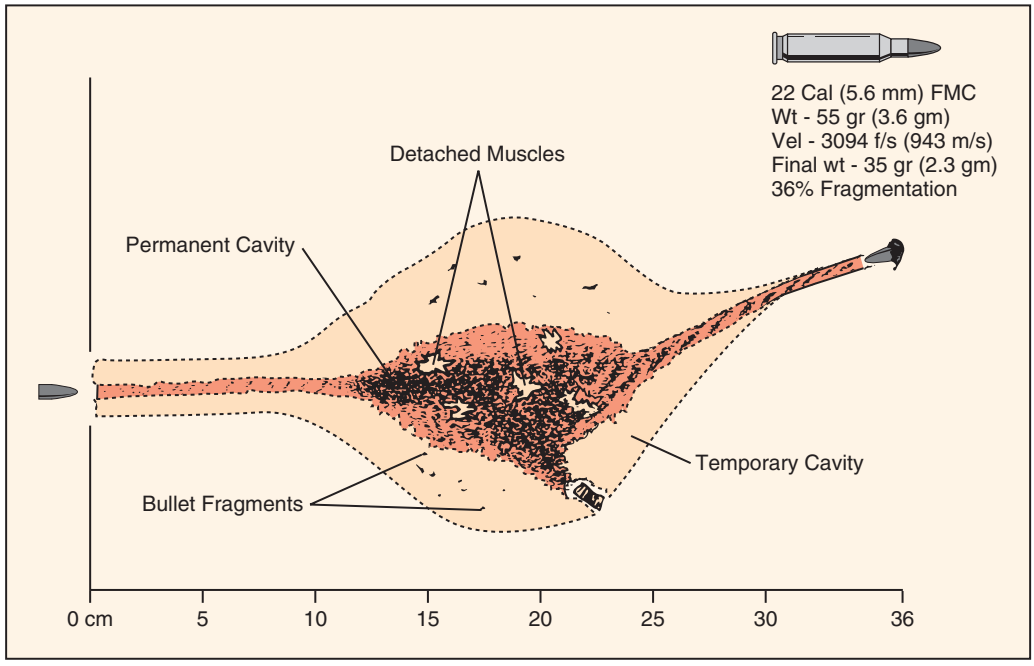
FIGURE 27-5 **A,** Ballistic representation of NATO 7.62-mm round fired from an M16 rifle. Observe the relatively consistent permanent cavity and laterally radiating temporary cavity, which begins to develop at approximately 20 cm into the tissue as the projectile begins to tumble. This chart represents the projectile not striking any hard structures causing deformation or alteration in trajectory. The anatomic characteristics of the head and neck do not have over 20 cm of soft tissue present prior to encountering the bony skeleton. This would have clinical significance in regard to the temporary cavity if the projectile's trajectory only encounters soft tissue and misses the underlying facial bones. **B,** Ballistic representation of a 7.52-mm soft point (SP) round striking muscle and bone. Note that as the projectile strikes the underlying structures, there is a tremendous increase in the permanent and temporary cavities as the projectile deforms and fragments due to the soft tip construction. This deformation in the structural characteristic of the projectile, and associated increase in the permanent and temporary cavities, greatly enhances the wounding potential of this round. **C,** Ballistic representation of a 22-caliber (5.6-mm) full metal case (FMC) round striking bone and muscle. Note that as the relatively small-caliber projectile strikes the underlying structures, there is a tremendous increase in the permanent cavity and associated temporary cavity as the projectile deforms and continues on a new trajectory. This illustrates the wounding potential of a smaller caliber weapon if the projectile actually strikes the target and engages in energy transfer to the tissues. (Adapted from Szul AC, Davis LB, Walter Reed Army Medical Center Borden Institute: *Emergency war surgery*, rev 3, Washington, DC, 2004, U.S. Government Printing Office.)


7.62 mm NATO
Vel - 2830 f/s (862 m/s)
Wt - 150 gr (9.7 gm) FMC
Permanent Cavity
Temporary Cavity
0 cm 5 10 15 20 25 30 35 40 45 50 55 60 64

A


Bullet Fragments
Detached Muscles
Permanent Cavity
7.52 mm SP
Vel - 2923 f/s (891 m/s)
Wt - 150 gr (9.7 gm)
Final wt - 99.7 gr (6.46 gm)
33.4% Fragmentation
1.95 cm
Temporary Cavity
0 cm 5 10 15 20 25 30 35 42

B


22 Cal (5.6 mm) FMC
Wt - 55 gr (3.6 gm)
Vel - 3094 f/s (943 m/s)
Final wt - 35 gr (2.3 gm)
36% Fragmentation
Detached Muscles
Permanent Cavity
Temporary Cavity
Bullet Fragments
0 cm 5 10 15 20 25 30 36

C



FIGURE 27-6 **A,** Photograph of the tremendous variety of caliber, projectile composition or construction, and variable volumes of propellant and casings available for the modern firearm. **B,** Cross-sectional analysis of a bullet and shotgun shell.

is the portion of the bullet that is expelled and strikes the victim.[49] The composition of the projectile (e.g., soft lead, hollow point, full copper covering) has a direct correlation with the wounding potential of the weapon. As a projectile deforms after striking the victim, either as a result of metallurgic composition during manufacture or as a direct consequence of striking the underlying bone, the energy transfer to the patient and potential injury to associated tissues is increased.

The case is the container in which the projectile, propellant (gunpowder), and primer are packaged as a single unit for placement into the firing mechanism of the weapon. Wads are generally plastic frameworks with a paper or felt insert that holds the various pellets (projectiles) together in relation to the propellant to allow for accurate and safe release of all the projectiles simultaneously from the barrel. The propellant, or gunpowder, is the accelerant that actually allows for expulsion of the projectile from the weapon. The more propellant in a cartridge, as is seen in Magnum and rifle rounds, the greater velocity the projectile exhibits. The primer is the only portion of the bullet with an explosive charge. As the primer is struck by the firing pin of the weapon, the explosive charge is activated, igniting the propellant and sending the projectile on its flight. Although traditional concepts of ballistics teach that impact kinetic energy (KE) is equal to half the mass (m) of the projectile times velocity (v) squared ($KE = \frac{1}{2}\,mv^2$), the increased energy transmitted from a high-velocity projectile does not necessarily translate to increased wounding capacity. Cunningham et al have suggested that modifications need to be used to correct the kinetic energy estimate of wounding potential for the type of tissue being struck by the projectile.[10,33,39,50] They indicated that softer tissues, such as brain and muscle, should be associated with a lower exponent of injury (0.5) than harder tissue, such as bone, which would have a higher exponent (2.5) and therefore a higher likelihood of permanent injury. The corrected formula for estimating wounding capacity by kinetic energy should be $KE = \frac{1}{2}mv^{0.5}$ to $KE = \frac{1}{2}mv^{2.5}$. Handguns are handheld firearms, with a barrel length generally 10.5 inches or less, which usually fire projectiles of a lower velocity and caliber.[49] The characteristic low-velocity wound has a small rounded, or slightly ragged, entrance wound, causing fragmentation of teeth and bony comminution, often exhibiting no exit wound[51,52] (Fig. 27-7). If an exit wound does occur, it is generally slit-shaped or stellate.

Handgun injuries generally have a tendency to push away, or stretch, soft tissues, including vessels or nerves as opposed to avulsive loss. Rifles are long guns with barrel lengths of more than 26 inches.[10,49] At a distance, rifle wounds create a low-energy transfer similar to that seen with handguns. At close range, the wounding characteristics are different due to the increased potential injury associated with velocity and high-energy transfer[49] (Fig. 27-8). The presence of an exit wound is usually found, which may be stellate and larger than the entry wound. The presence of avulsive soft or hard tissue wounds and significant bone fragmentation can be characteristic findings of rifle wounds.

A shotgun is a long gun that may fire a single pellet, or numerous pellets, at a relatively low velocity. The gauge of the shotgun is classified as the number of lead balls or pellets placed together, equaling the interior diameter of the barrel, which would weigh 1 pound. For contact for close range injuries, the effect of the gas discharged under pressure into the wound also needs to be considered. This scenario is extremely important in shotgun and IED blast wounds due to the higher degree of contamination and presence of propelled gas and shock waves.[28] Powder gases are expelled from the muzzle of

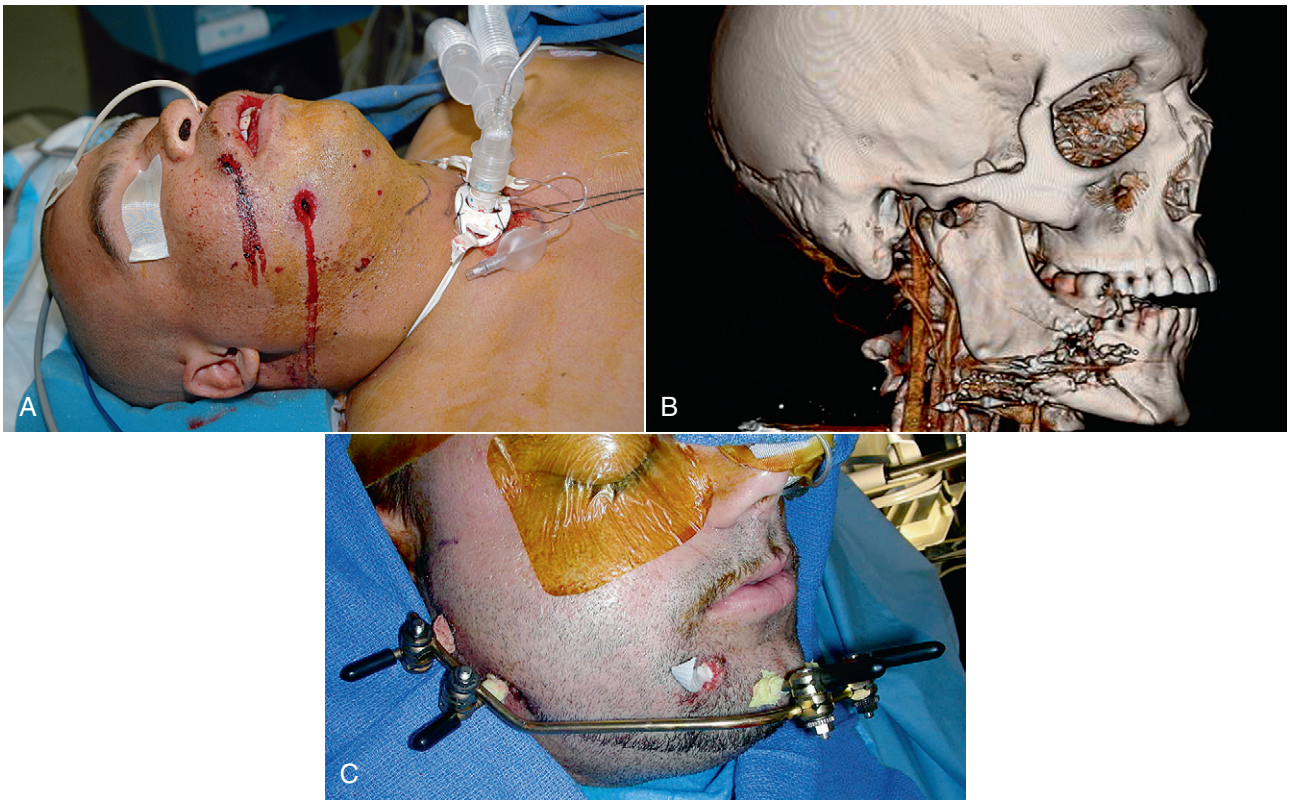


FIGURE 27-7 **A,** Characteristic clinical appearance of a low-energy, low-velocity gunshot wound to the anterior mandible. No exit wound was detected and the patient underwent an emergency tracheostomy secondary to airway concerns. **B,** Three-dimensional reconstruction of CT scan indicating the degree of comminution associated with this gunshot wound. The superior aspect of the projectile can be appreciated at the extreme bottom left of the CT scan. Three-dimensional reconstructions provide superior visualization and localization of anatomic variants in the management of ballistic injuries to the craniomaxillofacial unit. **C,** Application of a modern external fixator for the management of a low-energy, low-velocity gunshot wound to the mandible. Note the conservative treatment of the gunshot wound, with minimal decontamination and débridement and placement of a wet to dry gauze dressing.

the weapon after combustion of the gunpowder and follow the projectile out of the barrel. When the muzzle of the weapon is in contact with the target, this can be an additional source of tissue displacement, injury, and thermal burning.[38] Shotgun pellet injuries essentially depend completely on how far the weapon is from the target at the time of discharge. Sherman and Parrish have devised a classification system to describe shotgun wounds in relation to the distance from the target. Type I injury occurs from a distance longer than 7 yards, type II injury is sustained when the discharge is within 3 to 7 yards, and type III injury is within 3 yards.[53] Type III injuries usually sustain dramatic soft and hard tissue injuries and avulsion of tissue, whereas type I injuries may be minimal (see Fig. 27-9*A*). Because victims often have difficulty in determining how far away the shotgun was at the time of discharge, Glezer et al have revised this classification system and directed their attention to the size of the pellet scatter. Type I injuries occur when pellet scatter is within an area of 25 cm^2, type II injuries are within 10 to 25 cm^2, and type III injuries have pellet scatter less than 10 cm^2.[54] (Fig. 27-10). Although the Glezer classification originally was developed for abdominal injuries, the information is transferable to other areas of the body and determinations of tissue injury can be correlated directly to the size of the pellet scatter. Intuitively, the closer the shotgun is to the patient, the more dramatic is the hard and soft tissue damage. For rifles and handguns, the clinical difference in whether the weapon was 10, 100, or 1000 feet away from the patient otherwise has no bearing on treatment. The range of a .22-caliber handgun is approximately 1 mile, whereas the range of a rifle can be as long as 3 to 4 miles Although one could argue that at the outer limit of a projectile range it is less likely to penetrate a target, rarely does a shooting occur outside the effective range of a weapon.[49]

As noted earlier, current conflicts in the Middle East and Afghanistan have interjected a newer mechanism for the delivery of maxillofacial missile projectiles that cause gruesome and avulsive craniomaxillofacial injuries, the IED[28] (Fig. 27-11). Although not a new entity—the concept of IEDs has been deployed by guerilla forces since World War II—the description and media interest in the IED warrants a brief discussion of its characteristic properties. An IED is a bomb fabricated in an improvised manner designed to destroy or incapacitate military personnel or civilians. The bomb itself may be a conventional military grade weapon, or an assortment of explosive components such as gasoline, or agricultural fertilizer, as seen in the Oklahoma City bombing of 1995. An IED has five components—a switch (activator), initiator (fuse), container (body), charge (explosive), and

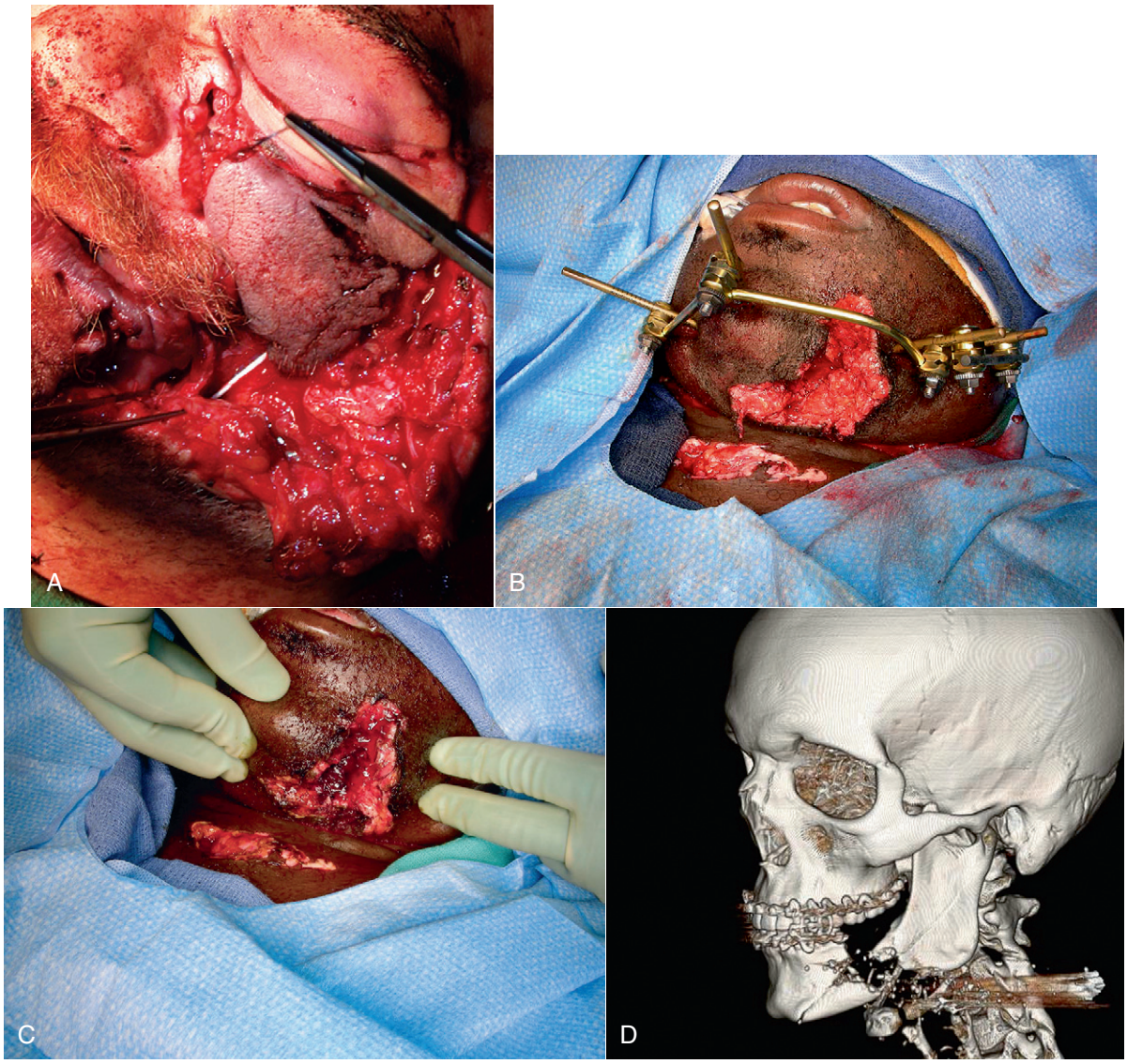


FIGURE 27-8 **A,** Cannulation of the severed parotid duct with Silastic tubing. All ballistic injuries to the craniomaxillofacial complex should undergo evaluation under some form of magnification for potential remnants of the salivary ducts and cranial nerves because primary repair is best accomplished at the time of initial injury. **B,** High-energy gunshot wound to the anterior mandible. Note the presence of soft tissue disruption and a second gunshot wound across the anterior neck, glancing off the surface epithelium and providing only superficial injury to the platysma. **C,** Initial stabilization of the patient was accomplished with an external fixator secondary to the presence of other life-threatening gunshot wounds that were sustained at the same time. The patient was subsequently treated with open reduction and internal fixation with a reconstruction plate and corticocancellous bone graft from the anterior ilium. **D,** Three-dimensional reconstruction of CT scan indicating the degree of comminution and avulsive bone loss associated with this gunshot wound. The projectile can easily be identified at the bottom right of the CT scan.

power source (battery). Antipersonnel IEDs typically contain shrapnel generating components such as nails, ball bearings, metal fragments, wood, and/or glass. Direct shrapnel injury is only a single element to be considered, because detonation of any powerful explosive generates a blast wave of high pressure that spreads out from the point of explosion and travels hundreds of yards in all directions. The relative proximity of the victim to the site of the explosion, the greater the exposure to the shock wave energy. The initial shock wave of very high pressure is followed closely by a so-called secondary wind, which is a huge volume of displaced air flooding back into the area, again under high pressure. It is these sudden and extreme differences in pressures, and associated dispersal of secondary projectiles, that can lead to significant neurologic, skeletal, or soft tissue injury. While a comprehensive review of IEDs, and their increasing use in armed conflict—from the Belarussian Rail War in World War II to the current use by insurgents in Iraq and Afghanistan—is beyond our intent here, but its inclusion as a wounding source for modern craniomaxillofacial injuries is warranted.

INJURY PATTERNS AND ASSOCIATED INJURIES

Hollier et al have retrospectively evaluated 84 patients with facial gunshot injuries.[26] Of these, 67% suffered

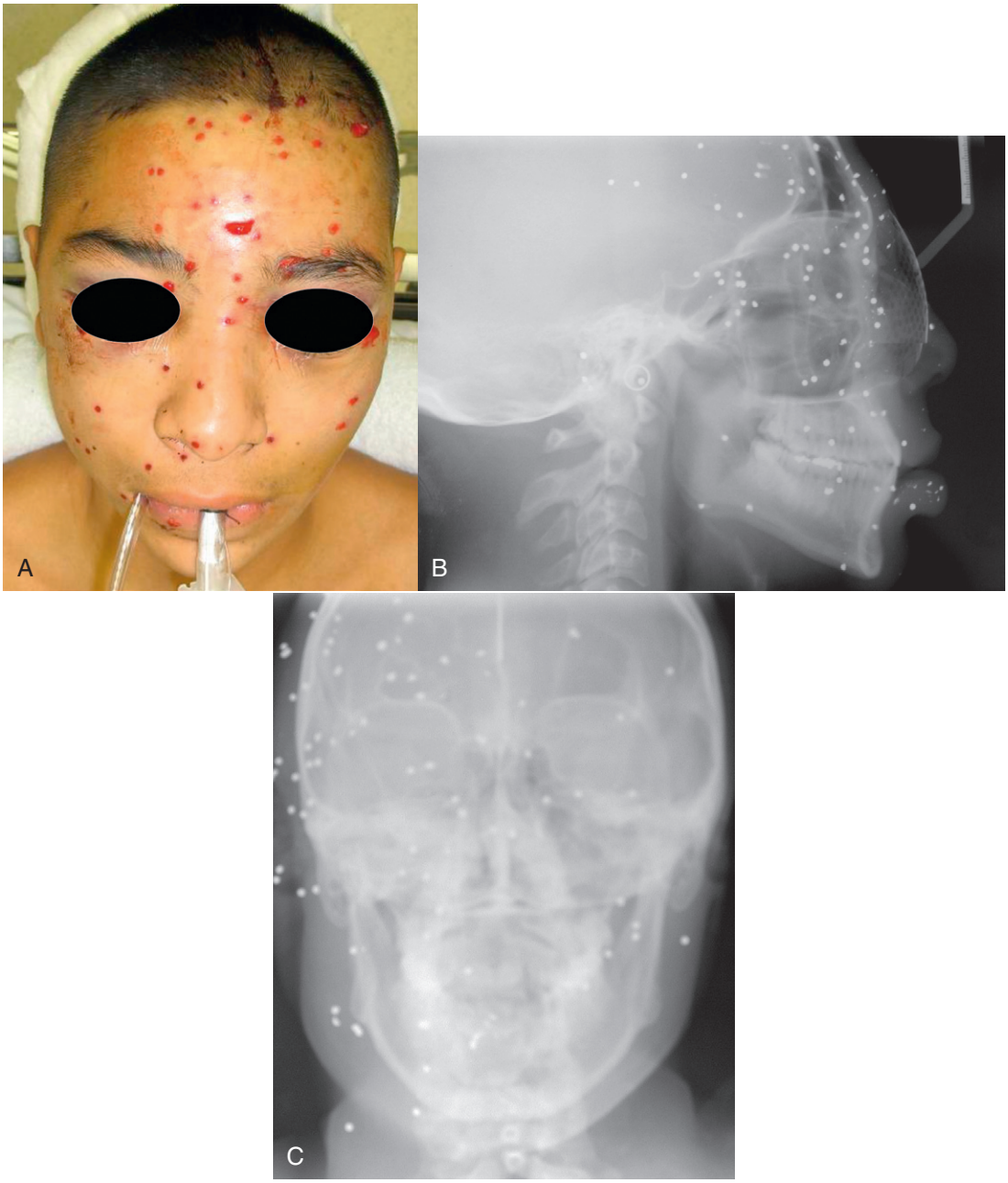


FIGURE 27-9 **A,** Characteristic facial appearance of a patient sustaining a shotgun wound from a distance. (Sherman and Parrish, class I; Glezer, class I). Note the presence of multiple punctate entry wounds, but no significant disruption of the facial features. **B, C,** Classic radiographic appearance of a patient sustaining a shotgun wound from a distance (Sherman and Parrish, class I; Glezer, class I). Note the presence of multiple shotgun pellets on the radiographs.

facial fractures, and 75% of them received surgical treatment of these injuries; 21% required emergent tracheostomy for airway control (all had injury in the lower third of the face); and 14% had great vessel injury diagnosed by angiography, of whom 50% required surgical treatment (1 in 12 was managed by embolization in the radiology suite). Other associated injuries included eye (31%), brain (18%), and tongue (13%). Facial fracture distribution included zygoma (35%), mandible (30%), orbit (26%), skull (21%), and nasoethmoid (11.9%).[26] An evaluation of 54 gunshot wound patients by Kihtir et al[55] revealed central nervous system injuries in 22%, orbital involvement in 40%, and midfacial involvement in 38% of patients in their data set. The most common fractures were the maxilla (41%) and mandible (28%). Vascular injury was present in 5 of 54 (9%) as determined by angiography.

Lew et al have reported that the incidence of penetrating soft tissue injuries and fractures for U.S. military personnel in Iraq and Afghanistan was 58% and 27%, respectively, with 76% of the fractures being open.[56] The location of the facial fractures in descending order of incidence was mandible (36%), maxilla-zygoma (19%), nasal (14%), and orbit (11%). The remaining 20% were

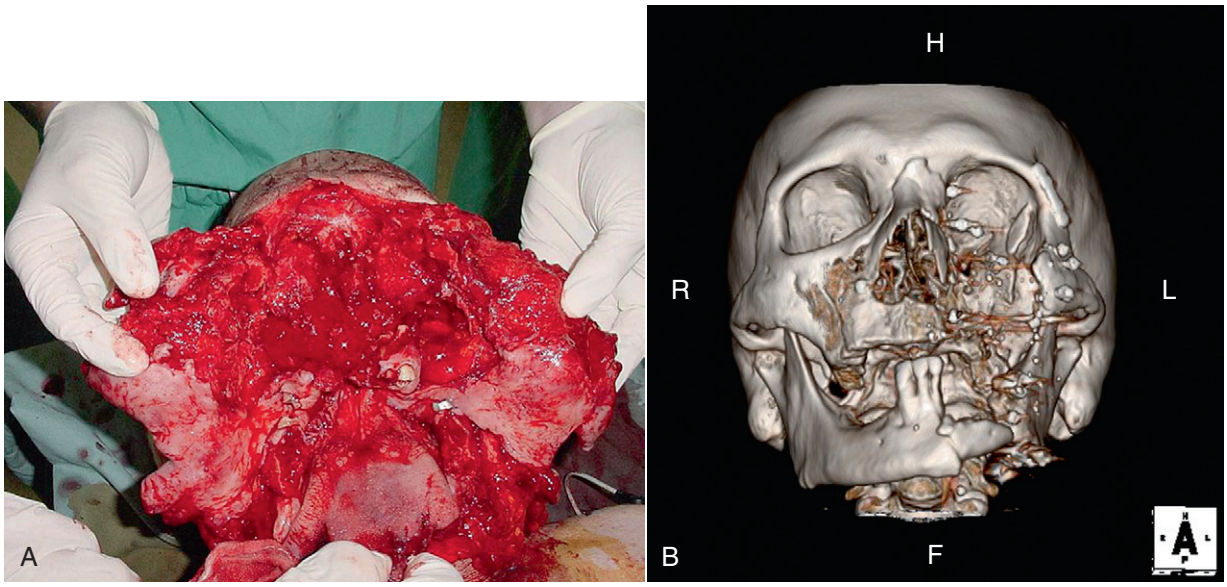


FIGURE 27-10 **A,** Self-inflicted shotgun wound in a suicide attempt. Note significant hard and soft tissue disruption and avulsion (Sherman and Parrish, Class III; Glezer, Class III). **B,** Three-dimensional reconstruction of CT scan showing the degree of hard tissue loss experienced in a suicide attempt with a high-energy weapon.



FIGURE 27-11 Characteristic facial injuries sustained by an improvised explosive device (IED). Note concomitant burns, tissue loss, shrapnel injuries, ruptured right globe, and avulsed soft tissues. Only a small portion of the left side of the patient's lateral nasal complex was unaffected by this injury.

not otherwise specified, with the primary mechanism of injury involving missile injuries from IEDs (84%).[56] As noted, Tan et al completed an experimental study in dogs in which simulated gunshot injuries were induced and vascular tissue subsequently studied by angiography, high-speed x-ray photography, and light and electron microscopy following injury; microthrombus formation, vascular endothelial loss, and necrosis as far as 3 cm from the wound margin were found.[48] Despite the extent of soft tissue injuries sustained in Iraq and Afghanistan, and the ongoing civilian gunshot wound experience in the United States, Loos recently noted the lack of standardized, controlled clinical studies highlighting the complexity of evaluating the progression of soft tissue injury for traumatically injured tissues in the zone of injury and the impact on proposed microvascular and/or pedicled tissue transfer.[57]

ACUTE CARE CONSIDERATIONS

As evidenced by the absolute priority highlighted in the *Emergency War Surgery* manual and the American College of Surgeons Advanced Trauma Life Support (ATLS) course, the primary focus in the immediate post-trauma period is securing the airway.[31,58] The rapid assessment for signs of airway obstruction in the trauma patient should focus on evaluation for potential foreign bodies and facial, mandibular, or laryngotracheal fractures, which may result in airway obstruction. Ballistic injuries to the face, head, and neck have direct bearing on ATLS protocols involving initial assessment and primary management of the airway, because bony instability and substantial bleeding from the abundant vasculature of the facial and great vessels of the neck often accompany these injuries. The presence of catastrophic wounds to the craniomaxillofacial complex may also serve as a distraction to emergency medical service personnel, focusing their attention away from potentially life-threatening injuries and wasting valuable time that could be better used for treatment of potentially lethal injuries, stabilization, and preparation for transport.

AIRWAY MANAGEMENT

As mentioned, Hollier et al found that 21% of facial gunshot injury patients required emergent tracheostomy for airway control, all of which involved injuries to the



FIGURE 27-12 Anterior mandibular avulsion as a consequence of a gunshot wound to the face. The patient is completely alert and oriented at the time of presentation and is sitting upright in a slightly forward positioning to maintain the patency of the airway. Due to the loss of soft tissue attachments to the anterior mandible, when the patient is positioned supine, the airway is obstructed. Recognition of this fact is an important component of the initial assessment and during the securement of the airway.

lower third of the face.[26] Kihtir et al's review of 54 civilian gunshot wounds found that 33% (18 of 54) required urgent airway control, with the following distribution: orotracheal intubation (24% [13 of 54] and 72% [13 of 18] of patients requiring airway management), cricothyroidotomy (4% of patients [2 of 54] and 11% of airway control patients), tracheostomy (4% of patients [2 of 54] and 11% of airway control patients), and nasotracheal intubation (2% of patients [1 of 54] and 6% of airway control patients).[55] Airway compromise is usually secondary to edema in close approximation to the airway, but may also be due to loss of structural integrity to the anterior region of the mandible (Fig. 27-12). On an urgent basis, manually supporting collapsed segments of the mandible and base of tongue attachments may quickly resolve posterior airway collapse, as can patient repositioning. Forward traction on the tongue and/or anterior collapsed mandible can be accomplished by the use of a towel clip or large-gauge suture material passed through the tongue or tissue in the symphysis region. Mask ventilation is unlikely to be successful for patients with injuries to the lower third of the face and may introduce air emphysema into tissue. The use of nasopharyngeal and oropharyngeal airways may provide some temporary benefit, but endotracheal intubation is still the emergency airway of choice when partial or complete obstruction is present and the patient displays a diminished level of consciousness or obvious evidence of respiratory distress or impending respiratory failure. The challenges encountered intubating a patient with a ballistic injury to the lower third of the face should not be underestimated because edema, bleeding, and macerated soft tissues may completely obscure the airway. The use of surgical cricothyroidotomy should be considered, although this should be accomplished by the most experienced surgeon available, because by definition, an emergent cricothyroidotomy should be considered as one of final acts to secure an airway, not the first in the sequence of airway establishment.

After successful tracheal intubation, the determination for a surgical tracheostomy to minimize interference for the reconstruction of the dental occlusion will need to be made. If prolonged intubation is expected based on the nature or severity of the craniomaxillofacial injuries, or concomitant intracranial injury, consideration should be given to early elective tracheostomy.[26,28,55] If multiple staged surgical procedures are anticipated, the lower third of the face has been injured, or there are extensive soft tissue injuries of the tongue, floor of mouth, submandibular triangles, and neck, serious consideration for elective tracheostomy should be given. Recent literature reports by Mahon et al have advocated the use of the submental or submandibular intubation technique if it has been determined that the patient will not require long-term ventilator support, and the indication for tracheostomy is primarily to minimize occlusal interference.[59]

HEMORRHAGE MANAGEMENT

Hemorrhage control, especially in close approximation to the airway, is a critical step to improve airway patency and to maintain normal hemodynamic status. Hemostasis can be accomplished by a variety of measures, including the use of pressure to stop acute hemorrhage and suctioning to clear accumulated blood and enhance the surgeon's ability to identify and ligate, clamp, or cauterize bleeding vessels. In case of difficulty in achieving primary hemostasis, application of pressure in conjunction with hemostatic dressings such as QuickClot or Combat-Gauze, kaolin-impregnated gauze dressings used extensively by combat medics in Iraq and Afghanistan (Z-Medica, Wallingford, Conn), can establish a significant reduction or elimination of high-volume vascular bleeding without the risks of distal emboli previously encountered with other hemostatic agents.[60-62] Blind clamping is discouraged in the head and neck region due to the proximity of motor and sensory nerves or other specialized components, such as salivary ducts, suspensory ligaments, and previously uninjured vascular structures, which may suffer iatrogenic injury. Active hemorrhage from the head and neck region may require image-guided embolization or surgical access for proximal vessel ligation to control bleeding not responsive to other more immediately available and less invasive measures. If major vessel injury is suspected based on entrance and/or exit wounds, or on expanding mass effect in tissues of the face or neck, then radiographic evaluation of the vascular components of the neck via angiography is indicated. Hemodynamic status must be assessed and managed, as necessary, with appropriate fluid resuscitation and blood component replacement. An important clinical point to remember is that once the patient's hypovolemia has been corrected, and mean arterial pressure has been stabilized, areas of head and neck hemorrhage previously controlled may begin to bleed actively

again.[32] The importance of ongoing evaluation cannot be understated; reports of second or multiple gunshot injuries can be as high as 36%, and the patient could easily be undergoing an exploratory laparotomy or thoracic procedure with the head and neck region obscured from view, allowing the blood loss to continue unabated and unrecognized.[26,28] The presence of a devastating craniomaxillofacial ballistic injury may often mesmerize less experienced trauma care staff or emergency medical personnel, drawing attention from other potential life-threatening sites of injury.

MANAGEMENT OF GUNSHOT WOUNDS TO THE FACE

Tremendous variation exists in literature reports and in U.S. trauma centers regarding the most appropriate management protocol for the treatment of ballistic injuries to the craniomaxillofacial complex. Single-stage versus multiple-stage procedures, or complex, extensive, early reconstruction versus initial débridement and/or closed reduction with subsequent later revision surgeries, are several of the management philosophies supported. Numerous authors have advocated a multidisciplinary approach to the treatment of the more complex of these injuries.[26,28,63-66] Although not specifically mentioned by some, however, it is unlikely that a single surgical specialty can appropriately manage the myriad of surgical subspecialty level injuries sustained in complex ballistic injuries to the craniomaxillofacial region, except perhaps for the simplest of these cases. Plastic, ophthalmology, otorhinolaryngology, interventional radiology, neurosurgery, trauma, general and/or maxillofacial prosthetic dental, and subspecialty microsurgery care is (are) often indicated for these individuals, as well as other evaluation and management based on an individual patient's injuries. These would include occupational and physical therapy, nutritional medicine, and behavioral science. Adequate nutritional support is often delayed in the management of ballistic injuries to the face. Once interruption of normal oral feeding has been identified for any length of time secondary to lower face, floor of mouth, or neck involvement, consideration should also be given to early gastrostomy tube placement, generally in conjunction with a planned anesthetic for débridement, early reconstructive treatment, or surgical tracheostomy.

For all facial gunshot injuries, and any complex craniomaxillofacial trauma case regardless of cause, consideration must be given to a comprehensive ophthalmologic evaluation. Hollier et al have found that 31% of patients experience some form of ocular injury and 54% of them have ongoing residual visual problems.[26] Hollier's recommended protocol includes careful evaluation of ophthalmologic status before any surgical intervention to ensure that injury-associated visual disturbance is properly documented. Cho et al have reported that 32.7% of patients with ballistic intracranial injuries sustain concomitant ocular injuries.[67] Both of these studies encouraged ongoing ophthalmologic evaluation to monitor for traumatic optic neuropathy, specifically subtle decreases in visual acuity and loss of red color perception, which may indicate progressive swelling of the optic nerve attributable to the initial injury. Based on these findings, surgical repair of facial fractures is often delayed in an effort to minimize progression of this optic nerve edema.

The concept of débridement is well established in the literature and is critical in the management of these ballistic injuries.[28,55,65,66] Serial washouts and débridement of tissue have become mainstays for craniomaxillofacial trauma surgeons in the treatment of these injuries. Unfortunately, for many surgeons, the term *débridement* is synonymous with the absolute removal of tissue in the operating room. Although devitalized necrotic tissue does require excision, a more accurate definition of the goal of serial washouts should be decontamination. Preservation of all viable tissues is a critical component in the management of gunshot wounds to the maxillofacial region. Once tissues are lost, the surgeon is faced with two choices—compromise anteroposterior projection to allow for primary closure of native tissue, or transfer additional tissue to the region via pedicled or microvascular grafts. Avoidance of the need for tissue transfer should be the goal and judicious use of the practice of decontamination will assist in achieving the desired results.

SOFT AND HARD TISSUE INVOLVEMENT

Before the initiation of surgical reconstruction of ballistic craniomaxillofacial injuries, an absolute understanding of the extent of the injuries and any functional and physiologic deficits is necessary. A critical mistake in the management of these disorders is the prevalent desire by the surgical team to provide definitive reconstructive care to the patient in an expedited manner. Rushing the patient to the operating room without a goal-oriented sequential surgical plan and without having the necessary preoperative treatment planning or required medical-surgical specialty consultations, will undoubtedly lead to a less than desirable surgical result. Evaluation of the soft tissues in the maxillofacial region is accomplished by direct observation and evaluation of the clinical response to serial decontamination and débridement. The accuracy of this appraisal is compromised by edema, which often distorts the remaining tissue, artificially increasing volume assessment and masking the true tissue deficit present. Cellular soft tissue damage persists and progresses for several weeks, ultimately affecting the quantity and quality of soft tissue remaining after completion of healing.[28,66] Before bony reconstruction occurs, appropriate imaging is necessary to define the magnitude and nature of the fractured segments clearly. Computed tomography (CT) with three-dimensional reformatting is essential for obtaining this information. Axial and coronal tomography imaging also provide this data, but the three-dimensional reconstructions improve conceptualization of the size and location of the fractured segments and their relationship to one another, which improves the ability to localize and reposition the segments intraoperatively. The accuracy and clarity of three-dimensional reconstruction have significantly increased during the past decade and should now be part of the standard workup for the treatment of complex ballistic

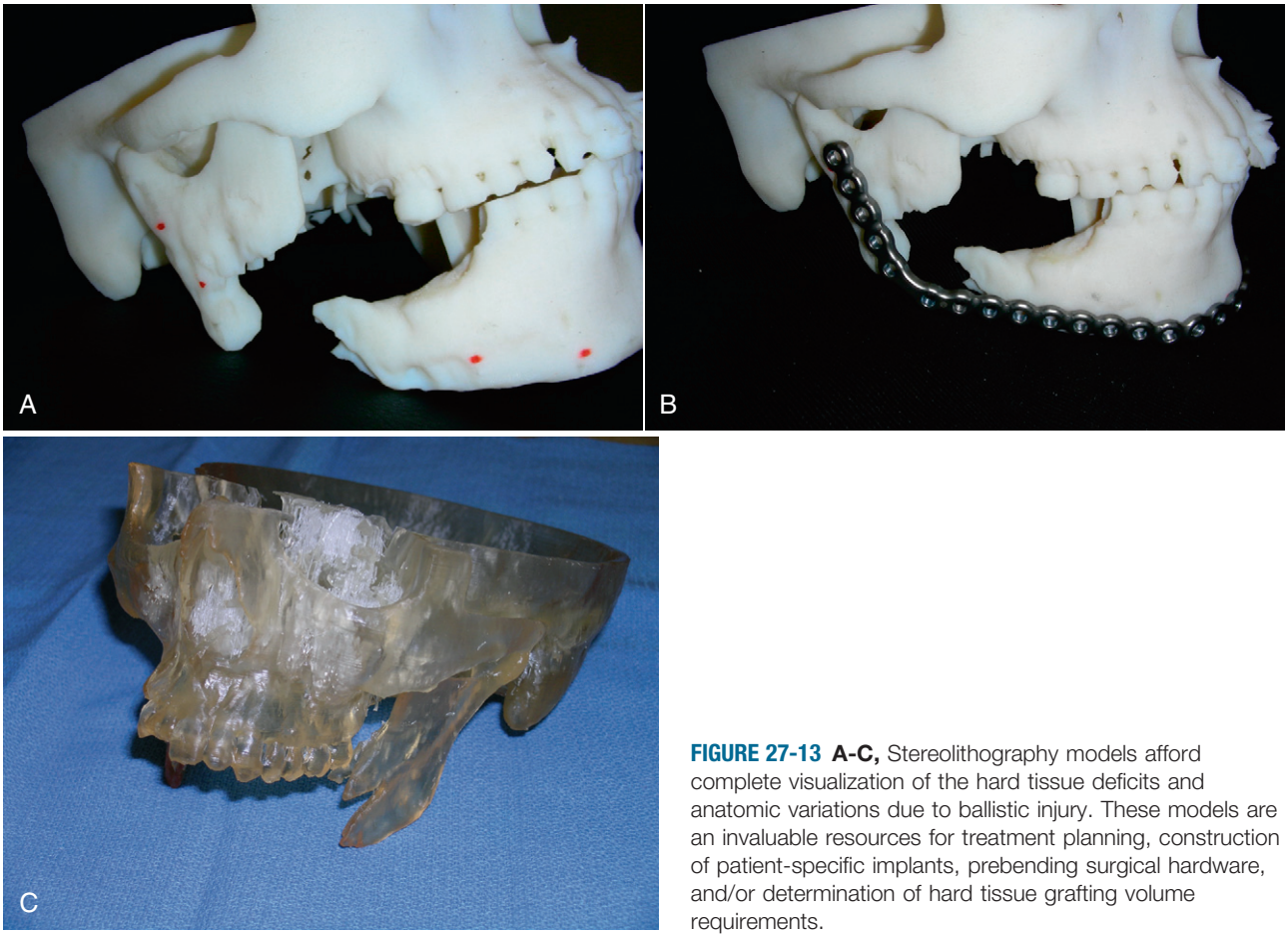


FIGURE 27-13 **A-C,** Stereolithography models afford complete visualization of the hard tissue deficits and anatomic variations due to ballistic injury. These models are an invaluable resources for treatment planning, construction of patient-specific implants, prebending surgical hardware, and/or determination of hard tissue grafting volume requirements.

craniomaxillofacial trauma. Stereolithographic models, extremely accurate acrylic resin representations of the CT scans, can be used for presurgical planning and prefabrication of surgical reconstruction plates, increasing accuracy of the reconstruction while decreasing time in the operative theater. The ultimate treatment goal for casualties with ballistic facial injuries is the restoration of function and cosmesis. Many patients injured by ballistic projectiles have significant avulsive defects of craniomaxillofacial structures. A vitally important aspect of treating these avulsive defects is using appropriate imaging to develop a staged reconstruction plan, with the final end point in mind before any reconstruction begins.

Items for consideration when reviewing the soft tissues, radiographic studies, and stereolithography models are as follows[28,66] (Fig. 27-13):

- Which structures are missing and which structures remain
- Effect of each of these on the reconstruction goals
- Which structures require replacement
- How those structures will be replaced (nonvascularized versus vascularized tissue)
- Identifying stabilization points for replacement structures
- Soft tissue considerations
- Choice of grafting material
- Effect of grafting plan on future implant reconstruction or dental rehabilitation

BONE AND SOFT TISSUE RECONSTRUCTION

The various bony and soft tissue reconstruction options for tissue replacement are covered elsewhere in detail in this text and are beyond our scope here. Concepts consistent with the principles of bone and soft tissue reconstruction will be addressed, with representative citations included for further review.

Controversy regarding the timing and extent of reconstruction exists largely because outcomes for catastrophic facial ballistic injuries are uniformly disappointing, regardless of the treatment chosen. At a minimum, at the time of initial surgical treatment, the remaining bone segments should be reduced and stabilized with some form of fixation, as guided by preoperative imaging and intraoperative evaluation. External fixation with closed reduction obviates the need for elevation of the periosteum, which is a necessary component for the application of rigid internal fixation. The elevation of periosteum, especially in the presence of comminution and multiple small bone fragments, compromises the vascular supply, which could lead to resorption, volume loss, or necrosis.[48,56]

Maxillomandibular fixation is necessary when the tooth-bearing portion of the maxilla or mandible is involved and should be applied early to ensure that the occlusion is properly restored. Care should be taken in the application of arch bars in the presence of a comminuted mandible fracture, as often encountered with

ballistic injuries. Overtightening the wires may lead to lateral displacement of the mandibular angles bilaterally, excessively widening the patient's facial profile and losing the angularity normally associated with facial projection.[28] The use of dental impressions to fabricate surgical splints can similarly be complicated by the presence of complex maxillary and mandibular fractures and loss of normal dental arch anatomy. A widened dental arch form, often produced in the laboratory by plaster models lacking definitive bony support and skeletal references, may result in inadequate anterior projection of the maxilla and zygomatic arches bilaterally, resulting in a patient profile potentially flattened and wide.[66] Avulsion or loss of hard and soft tissue volume should always be identified before the initiation of reconstructive surgery, because a strategy must be in place for the early or delayed replacement of this tissue. Possibilities include immediate microvascular or pedicled tissue transfer to the area covering bone, obturation of a defect, or provision of a vascular bed for a projected bone graft to the site. The ideal time for identifying soft tissue, and possibly hard tissue, volume loss is after stabilization of the bony skeleton. Residual continuity, or volume deficits, should be detectable at this stage, affording proper planning for potential tissue transfer procedures preoperatively. Thus, you can obtain proper consent, prep, drape, and position the patient properly, and have present all the instrumentation and personnel that will be necessary to complete the tissue transfer.

A number of authors have advocated immediate or early reconstruction, with vascularized free or pedicled tissue transfers; they believe that these procedures minimize scar formation of soft tissue into bony defects, or the development of wound contracture, which is difficult to repair subsequently.[26,64,66-77] Although clinical and research experience supports the viability of immediate tissue transfer if indicated by the presence of acute tissue loss, the appropriate use of this technique involves accurate determination of tissue transfer needs and the viability of the recipient bed. Robertson and Manson have reported that high-energy ballistic wounds to the craniomaxillofacial region may exhibit progressive necrosis, similar to injuries seen in electrical burns, compromising the small vessel anastomosis required for microvascular transfer and the margins of the recipient bed for a pedicled flap.[65] As noted, Tan et al's study involved the completion of microvascular anastomoses for small vessels at the ballistic wound site bed.[48] The anastomoses were completed at various times following injury and the short-term success of these anastomoses was evaluated. Repairs completed 3 days or later following injury maintained their patency best, confirming Manson's belief that sequential decontamination and débridement procedures can effectively result in the recipient tissues achieving the desired state of healing to support the transfer of tissues to the site.[28] Others have advocated less surgically aggressive approaches to ballistic injuries, opting for decontamination and débridement, closed or open reduction of fractures, and secondary reconstruction of residual defects using autogenous bone grafting, with or without delayed tissue transfer.[27,55,63] This form of treatment is best applied to low-energy injuries or injuries with minimal to no tissue loss because, when used for midfacial or complex avulsive injuries, cosmetic and functional results may be suboptimal. Delayed reconstruction of these catastrophically injured patients is complicated by scarring and wound contracture, which will undoubtedly occur in the aftermath of a gunshot injury. Although implied in the literature outlining complex tissue transfer reconstructions, thorough decontamination and débridement of foreign material from the wound site is indispensable, coupled with proper anatomic reduction and stabilization of the fractured segments with salvage and/or preservation of the remaining native soft and hard tissues. Even the most ambitious reconstructions require these necessary initial steps for optimization of the recipient site on which reconstruction is planned.

Others have advocated the use of distraction osteogenesis or other tissue traction for replacement of hard and soft tissue lost to facial gunshot injuries.[78,79] Shvyrkov et al have reported on 33 males with 3- to 8-cm bone and soft tissue defects of the mandible initially managed by débridement and collapsing the residual mandibular segments into the defect, using a transport disc distraction technique to generate bone and soft tissue across the osseous gap. Of these patients, 28 had distraction completed immediately following injury and 5 had their distraction completed after the initial gunshot injury had healed. According to the authors, all 33 patients had acceptable functional and aesthetic outcomes within 3 to 4.5 months of reconstruction.[78] The advantage to this procedure is obvious; bone and soft tissue volume are restored without harvesting tissue from a remote site, preventing the morbidity associated with harvest and preventing the mismatch of tissue characteristics inherent with distant tissue transfer. Although the rationale and results of this reported technique are exciting, it should be noted that a comprehensive review of the literature was unable to discover use of this technique for ballistic injuries by other authors. Herford and Boyne, however, have successfully published on the regeneration of four mandibular body defects secondary to pathologic ablations and isolated trauma defects with distraction osteogenesis, reporting effective hard and soft tissue generation across traumatically compromised and/or irradiated tissue beds.[80-82] Nonimmediate reconstruction with vascularized pedicled or microvascular tissue transfer has also been described.[83-93]

Because gunshot injuries frequently have tissue loss secondary to avulsion and/or necrosis, the replacement of tissue is necessary. In areas in which the soft tissue is adequate, free bone grafting may be all that is necessary to restore facial form and function. Inadequate soft tissue volume can sometimes be expanded, using standard soft tissue expansion techniques.[94,95] After adequate soft tissue expansion, progression to the bone-grafting procedure to reconstruct missing osseous elements or the placement of custom-fabricated surgical implants can occur, depending on the location of the defect.[96] Pedicled and free microvascular composite grafts are other accepted and generally highly successful options for replacing moderate or large volumes of hard and soft tissue. A summary evaluation of the current literature

indicates that the trend is toward early definitive reconstruction of missing hard and soft tissue using vascularized tissue transfer techniques as composite grafts or in association with free bone grafts. A key consideration of this method of treatment is to have reestablished the bony projection and angular shape of the facial skeleton as soon as possible, but definitively within 10 to 14 days, prior to the development of intractable facial scarring and contracture or the formation of soft or hard tissue infection.[28,65]

POSTOPERATIVE COMPLICATIONS

As described independently by Manson, Powers, and others, the most common complication associated with ballistic injury management in the craniomaxillofacial region is delay in definitive soft or hard tissue reconstruction, with the subsequent development of a flattened wide face as the tremendous forces of facial scarring and wound contracture alter the facial composite.[28,65,69,77] Once facial soft tissue scarring and wound contracture have matured, and the underlying bony architecture has remodeled, the opportunity to re-create any semblance of normal facial form, contour, and projection is lost. As eloquently stated often by Manson, there is no second opportunity to perform a satisfactory primary reconstruction.[65] Hollier et al have reported the following postoperative complication rates: cranial nerve palsy (19%), blindness (17%), hemiparesis (12%), visual disturbance (12%), wound dehiscence (4.7%), generalized sepsis (2.4%), and epiphora (2.4%).[26] Other isolated complications reported were cerebral vascular accident, speech difficulty, cerebrospinal fluid leak, facial nerve palsy, seroma, acute renal failure, disseminated intravascular coagulation, and ptosis of the upper eyelid. Kassan et al have reported a postoperative infection rate, or sepsis, of 19%, which is higher than that reported by other authors, primarily associated with comminuted low-energy handgun wounds.[27] Kassan also noted similar rates of neurologic and ocular complications as described by Hollier and Cho, in addition to restricted mandibular range of motion.[26,67] Others have described similar rates of the same complications listed, especially infection and ophthalmologic compromise.[97,98] The higher incidence of ocular injuries is also seen in the presence of IEDs in modern military action and terrorist attacks; Gataa has reported a 29% ocular injury rate for civilian craniomaxillofacial wounds, primarily caused by IED blasts.[99] Kihtir et al reported that 9.3% of ballistic injury patients (5 of 54) experienced coincident palatal injuries acutely and were treated by only limited early surgical management at a large metropolitan hospital in New York.[55] Oronasal fistulas developed in two of these patients at the completion of healing, for a complication rate of 13%. Of the 54 patients, 8 received closed reduction of their mandible fractures with one resulting in nonunion. One mandibular fracture patient was treated with open reduction and internal fixation, subsequently developing osteomyelitis, although the reported algorithm from this study encouraged delayed treatment and management of all maxillary, orbital, and zygomatic fractures without reduction, which may be a component of the elevated complication rates.

Sherman and Gotleib have reported a case of carotid-cavernous sinus fistula developing subsequent to a severe midfacial gunshot injury.[100] Although a rare complication of craniomaxillofacial trauma, this trauma-induced pathologic entity, which matures between the internal carotid artery and cavernous sinus, may result in retinopathy, optic atrophy, blindness, or fatal epistaxis. The characteristic findings associated with carotid-cavernous sinus fistula include chemosis, pulsatile exophthalmos, and supraorbital bruit. If suspected by physical examination or clinical response, CT angiography is the accepted practice for confirmation of the presence of this condition, which is treated in coordination with vascular surgery or interventional radiology with ligation of the internal carotid, embolization or, in some cases, observation.[100] Tattooing attributable to material incorporated into the wound at the time of injury can be minimized or eliminated with the use of a Q-switched Nd:YAG laser,[28] but this should not be used to remove tattooing attributable to dermal inclusions of gunpowder, which is shot into the skin at close range. Fusade et al have found that laser use on these individuals induces bleeding transdermal pits, which subsequently scar in a poxlike fashion, with spreading of the pigment into the skin surrounding the initial areas of the tattoo.[101] The theory is that laser energy transfers to the gunpowder, causing microexplosions and subsequently leading to the observed tissue damage.

Early dermabrasion has been suggested as a mechanism for the prevention of gunpowder tattooing. Pallua has suggested that dermabrasion for the removal of embedded gunpowder ideally should be completed within the first 6 hours following gunshot injury and never later than 72 hours postinjury[102] (Fig. 27-14). For later gunpowder tattoos or for deeper penetration of powder, the recommended treatment is use of a minipunch excision technique for the removal of these remnants, as described by Kaufmann and Powers et al.[28,103] Although time-consuming, it is technically simple to perform and yields superior aesthetic results, without excessive removal of uninvolved tissue. Many of the techniques used for immediate, early, and secondary reconstruction can also be used for secondary reconstruction or for the management of complications.

SUMMARY

Craniomaxillofacial ballistic injuries pose incredible challenges to the facial trauma surgeon, offering the opportunity for a tremendously rewarding—or emotionally draining—personal and operative experience. Technologic advances in surgical hardware manufacture, radiologic imaging capabilities, and improved operative techniques have afforded the facial trauma surgeon the tools necessary to accomplish the primary goal of surgical treatment: returning these injured patients to a reasonable level of aesthetics, form and function, and allowing them to integrate back into society. Although the catastrophic nature of these injuries places obvious limits on what can be realistically achieved, continuously striving



FIGURE 27-14 **A,** Blast injury patient with embedded gunpowder, shrapnel, and other debris. **B,** The same patient immediately postoperatively after conservative decontamination and débridement, foreign body removal, and superficial dermabrasion.

for surgical excellence should remain the focus of all surgical interventions. This is best accomplished by a comprehensive presurgical evaluation, identification of injured and noninjured tissues, understanding of the functional and anatomic limitations imposed by the wounds, and clarity of purpose and rationale for the proposed reconstructive procedures. The application of the principles outlined in this chapter, coupled with the primary tenet of the Hippocratic Oath, *Primum non nocere* ("First, do no harm"), will serve the facial trauma surgeon well.

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CHAPTER

28 Head and Neck Burn Injury

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OUTLINE

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Primary Burn Wound Management
Secondary Burn Wound Management

The acute and chronic management of facial burns is one of the most challenging injuries affecting the oral and maxillofacial region. Such injuries require the combined expertise in trauma management, complex soft and hard tissue reconstructive techniques such as composite vascularized free tissue transfer, skin grafts, facial re-animation, scar revisions, dental implants, and regenerative medicine. Traditional treatment of facial burns relied on the inherent healing capabilities of the patient with minimal reconstructive efforts. Modern management of facial burns integrates complex local and distant soft and hard tissue grafting. The re-creation of facial animation and underlying musculature to restore facial expression continues to elude treating surgeons. Injury to the muscles of facial expression and associated facial nerve are extremely difficult to reconstruct. Frequently, the patients remain seemingly expressionless, with a masklike facial stigmata, compounded by mismatched graft colors and anatomic discrepancies. Injuries to the eye and periorbita are debilitating with possible loss of vision with associated reconstructive challenges, such as orbital enucleation and prosthetic eye rehabilitation. Despite the many technologic and scientific advances since the treatment of facial burns by Simon Hullihen in the nineteenth century, there remains extensive room for scientific and technical improvements in this field. The recent advances in facial transplantation are exciting; however, their application to facial burns remains unclear, yet promising. The future will bring advanced grafting and practical transplantation technology. More importantly, enhanced tissue engineering, growth factors, and application of gene therapy modalities to reconstruct and rehabilitate the burn victim will be developed. Given the existing challenges in burn management, and despite our advances in reconstructive modalities, however, the best treatment of facial burns remains the emphasis on prevention.

EPIDEMIOLOGY

A burn is defined as a traumatic injury to the skin or other organic tissue primarily caused by thermal or other acute exposure. Burn injury to tissues is caused by the contact with heat, flame, chemicals, electricity, or radiation. Timely and adequate management of burn injuries is paramount. Such injuries are painful and can result in mutilating and scarring, amputation or necrosis of affected tissue or, in extreme cases, death. Peripheral nervous system, vasculature, skeletal muscles, and bones could be affected at a distant site to the burn.[1] Burns are one of the most distressing injuries and contribute to major global morbidity and mortality.[2,3] Each year, approximately 450,000 people in the United States seek medical care for burns.[4,5] The mortality rate for the hospitalized patients reaches 3.9%. An estimated 3,000 deaths result from residential fires and 500 from other sources, including motor vehicle accidents (MVAs) and aircraft crashes and contact with electricity, chemicals, or hot liquids and substances. About 75% of these deaths occur at the scene or during initial transport. Fire and burn deaths are combined because deaths from burns in fires cannot always be distinguished from deaths from smoke poisoning. Most burn injuries, approximately 66%, occur in the home setting. The 2011 National Burn Repository has reported that burns due to fire and flame predominate in the 5-year and older age group. Scalds were most frequent in children younger than 1 to 5 years. In Middle East countries, burning from oil explosion are

among the most common types of the burn in oil well workers.[6] Self-burning suicide also is prevalent in these countries. Patients with a total burn size of 40% burn surface or more accounted for only 4.2 % of cases. Fatality clearly increases with burn size. The burn size associated with a 50% case fatality appears to be approximately 70% total burn surface (TBSA). Complications such as pneumonia, cellulitis, and urinary tract infections are the most prevalent complications noted in burn center patients. Many burn injuries are minor and do not require hospital admission.[7,8] The mean length of hospital stay was approximately 9 days in 2010.

MECHANISMS OF BURN INJURY

Burns may be caused by several different mechanisms with associated complications:

THERMAL INJURY

The depth of the burn injury is related to contact temperature, duration of contact of the external heat source, and thickness of the skin. Thermal injury is generally classified as scald or flame injury.[5] As the temperature rises, increasing molecular collisions occur, resulting in altered molecular conformation and the disruption of intermolecular bonds. This process leads to cell membrane dysfunction as ion channels are disrupted, resulting in sodium and water intake. As the temperature rises further, protein denaturation occurs, oxygen radicals are liberated, and eventually cells die with the formation of the burn scar.[9]

Scalds

These types of burns result when skin comes into contact with hot liquids. About 70% of burns in children are caused by scalds.[10]

Flame Injury

Flame burns comprise 50% of adult burns. They are often associated with inhalational injury and other concomitant trauma. Flame burns tend to be deep dermal or full-thickness burns. Flame injuries and scalds are the most common causes of burns in children and adults worldwide.[5]

Cold Exposure (Frostbite)

Damage occurs to the skin and underlying tissues when ice crystals puncture the cells or when they create a hypertonic tissue environment. Blood flow can be interrupted, causing hemoconcentration and intravascular thrombosis, with tissue hypoxia.[11]

CHEMICAL BURNS

These burns result from contact of the skin with chemicals or by ingestion of chemicals. Most chemicals that cause chemical burns are strong acids or bases. Contact with acid produces tissue coagulation, whereas alkaline burns generate colliquation necrosis.[11]

ELECTRICAL BURNS

Electrical energy is transformed into thermal injury as the current passes through poorly conducting body tissues (Fig. 28-1). The amount of heat generated, and hence the level of tissue damage, is equal to 0.24 multiplied by (voltage)^2 times resistance. Domestic electricity is low voltage and tends to cause small deep contact burns at the exit and entry sites. Electricity greater than 1000 V may cause muscle damage, rhabdomyolysis, and renal failure.[10]



FIGURE 28-1 Classic example of an electrical oral commissure burn, which is a full-thickness injury with a well-demarcated eschar. (From Ward Booth P, Eppley B, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, St. Louis, 2012, Saunders.)

RADIATION BURNS

Radiofrequency energy or ionizing radiation can cause damage to skin and tissues. The most common type of radiation burn is sunburn.[10]

PATHOPHYSIOLOGY OF BURNS

SKIN ANATOMY

The skin is the largest organ of the body. It functions as a neurosensory organ and protects against the invasion of foreign bodies and organisms. It has specific immune and metabolic functions and is important in regulating body temperature and fluid, protein, and electrolyte homeostasis. Loss of the functional skin barrier after thermal injury remits increases the susceptibility to infection, which is the major cause of morbidity and mortality postburn.[12] Skin thickness varies with age and body location, but averages only 1 to 2 mm (0.04 to 0.08 inch) thick. The skin is composed of several layers, with the two

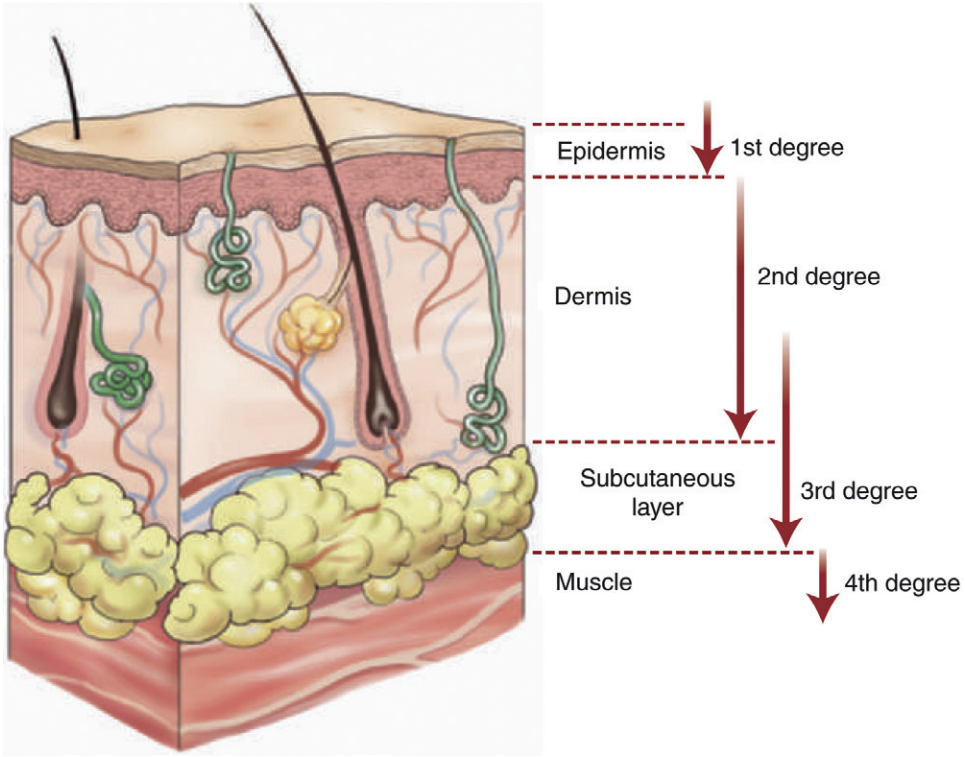


FIGURE 28-2 Anatomic layer of the skin and burn depth classification according to the anatomic layer of the skin. (From Shiland B: Mastering health care terminology, ed 2, St. Louis, 2006, Mosby.)

primary layers being the epidermis and underlying dermis (Fig. 28-2). The thickness of skin affects susceptibility to burning. The skin on the palms of the hands and on the soles of the feet, for example, is thick and more resistant to burning than that of the forearms or eyelids. Skin consists of three different layers of varying thickness throughout the body. The epidermis is the outermost layer of the skin with self-renewal properties and protects the skin from the exogenous environment. The average thickness of the epidermis is 0.1 mm.[13] The dermis has an average thickness of 2 mm and is a fibrous network of tissue that gives resiliency to the skin. Major proteins found in the dermis are collagen and elastin produced by fibroblasts. The vasculature in the dermis helps with thermoregulation and provides nutrients for the epidermis. The superior part of the dermis is called the papillary dermis, which abuts the epidermis. It consists of loose connective tissue with a lower amount of collagen and elastic fibers. The inferior part of the dermis, the reticular dermis, with lower cell density, contains higher amounts of collagen and elastin fibers. An areolar subcutaneous tissue lies below the dermis and is the third anatomic layer of the skin.

BODY RESPONSES TO BURNS

Local Responses

Fairly high temperatures can be sustained by the skin because of its substantial water content. As long as the water content is not totally eliminated by the heat in thermal injury, the temperature of the skin will not exceed 82° C (179.6° F).[14] Areas of increased vascularity allow heat to be transferred from the burn site because of high blood flow. This heat dissipation may decrease the depth of burn in such areas.[15] Major burns have three distinct zones of injury (Jackson's thermal wound theory), which usually appear in a bull's-eye pattern.[16]

Zone of Coagulation. The zone of coagulation comprises the dead tissues that form the burn eschar, located at the center of the wound nearest to the heat source.

Zone of Stasis. The tissue just adjacent to the necrotic area is called the zone of stasis, which is still viable. However, decreasing perfusion can lead to necrosis. Increasing tissue perfusion in the initial wound management stage has paramount importance in the inhibition of irreversible injuries. Prolonged edema, infection, unnecessary surgical intervention, and hypotension can convert this zone to the zone of coagulation.

Zone of Hyperemia. In this outermost zone, tissue perfusion is increased. The tissue here will invariably recover unless there is severe sepsis or prolonged hypoperfusion.[17]

Systemic Responses

Where the area of the burn wound exceeds 20% TBSA, there is a risk that the protective inflammatory response becomes overwhelmed, with proinflammatory mediators and subsequent significant systemic manifestations. The early phase of burn edema, lasting from minutes to 1 hour, is attributed to mediators such as histamine, products of platelet activation, eicosanoids, and proteolytic products of the coagulation, fibrinolytic, and kinin cascades. Vasoactive amines may also act by increasing microvascular blood flow or vascular pressures, accentuating the burn edema.[18]

Cardiovascular Changes. Capillary permeability is increased, leading to loss of intravascular proteins and fluids into the interstitial compartment. Histamine is probably responsible for the early phase of increased vascular permeability after burn injury because it is released in large quantities from mast cells in burned skin immediately after injury.[19] Peripheral and splanchnic vasoconstriction occur. The cardiomyocyte shock state is a result of impaired calcium homeostasis and subsequent intracellular signaling dysregulation.[20]

Respiratory Changes. Inflammatory mediators cause bronchoconstriction; in severe burns, adult respiratory distress syndrome can occur.

Metabolic Changes. The basal metabolic rate increases up to three times its original rate.[17] Glucose metabolism is elevated in almost all critically ill patients, including those with burn injuries. Gluconeogenesis and glycogenolysis are increased in burn patients. In addition, plasma insulin levels typically are elevated in burn patients. The basal rate of glucose production is elevated despite this hyperinsulinemic state, which can be defined as hepatic insulin resistance.[21] Exogenous insulin administration to achieve euglycemia has been shown to decrease donor site healing time and decrease length of stay while ameliorating skeletal muscle catabolism.[22] Lipolysis occurs at a rate in excess of the requirements for fatty acids as an energy source due to alterations in substrate cycling. Proteolysis is increased in burn patients. Following utilization, protein is excreted primarily in the urine as urea. This results in an increased efflux of amino acids from the skeletal muscle pool, including gluconeogenic amino acids. Protein intake greater than 1 g/kg/day has been recommended for all thermally injured patients and, for burn patients with normal renal function, the recommended protein intake is 2 g/kg/day.

Immunologic Changes. All injuries, including burns, are immunosuppressive. Nonspecific downregulation of the immune response occurs, affecting cell-mediated and humoral pathways.

SEVERITY INDEX

Assessing the severity of a burn injury requires accurate clinical staging, which permits appropriate therapy to be initiated in the proper medical facility. It is important to determine the source and circumstances of the injury, probable depth of cutaneous destruction, and extent of the body surface involved.[14]

CLASSIFICATIONS OF BURN DEPTH

A precise classification of burn injury may be difficult. The depth of a burn can be assessed clinically by its appearance. Punch biopsy of burned tissue with subsequent histologic assessment has been regarded by some as the criterion standard of depth diagnosis, serving as the basis for comparison of other diagnostic modalities.[23] Burn lesions are originally classified based on the clinical signs and symptoms, from first- to fourth-degree burns. The American Burn Association in 2009 released a newer classification based on anatomic depth involvement of the skin:



FIGURE 28-3 Superficial scalp burn from house fire, which will heal on its own. (From Ward Booth P, Eppley B, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, St. Louis, 2012, Saunders.)

- Superficial or epidermal (first-degree burn; Fig. 28-3)
- Partial-thickness (second-degree burn; Fig. 28-4)
- Full-thickness (third-degree burn; Fig. 28-5).

Burns extending beneath the subcutaneous tissues and involving fascia, muscle, and/or bone are considered fourth-degree burns (see Fig. 28-2 and Table 28-1).[5,11,24]

Superficial Burn (First-Degree)

These burns affect the outer layer of skin, causing pain, redness and swelling (see Fig. 28-3). Superficial burns equal to first-degree burns involve minimal tissue damage and involve the epidermis (skin surface). Sunburn is a good example of this type of burn.

Partial-Thickness Burns (Second-Degree)

Partial-thickness burns involve the epidermis and portions of the dermis and can be clinically categorized as superficial partial-thickness or deep partial-thickness burns (see Fig. 28-4). Superficial partial-thickness burns characteristically form blisters between the epidermis and dermis. Because blistering may not occur for some hours after injury, burns that initially appear to be only epidermal in depth (first degree) may be determined to be partial-thickness burns 12 to 24 hours later. Most superficial partial-thickness burns heal spontaneously in less than 3 weeks, and do so typically without functional impairment or hypertrophic scarring. Deep partial-thickness burns extend into the lower layers of the dermis. They possess characteristics that are distinctly different from superficial or mid-dermal partial-thickness burns. If infection is prevented and spontaneous healing is allowed to progress, these burns will heal in 3 to 9 weeks. However, they invariably cause considerable scar formation. These burns are best treated by excision and grafting. For the patient, a partial-thickness burn that fails to heal within 3 weeks is functionally and cosmetically equivalent to a full-thickness injury.

Full-Thickness Burns (Third-Degree)

Full-thickness burns extend down into the hypodermis or subcutaneous tissue (see Fig. 28-5). These burns in



FIGURE 28-4 Partial-thickness facial burns. **A,** Flash burn from gas grill. **B,** Flash burn from throwing gasoline on burning wood. **C,** Deeper partial-thickness burn from flames from house fire. (From Ward Booth P, Eppley B, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, St. Louis, 2012, Saunders.)



FIGURE 28-5 Full-thickness facial burn from house fire involving the forehead, cheek, and ear. (From Ward Booth P, Eppley B, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, St. Louis, 2012, Saunders.)

TABLE 28-1 Burn Depth Classification

Depth	Cause	Appearance	Sensation	Healing Time (days)
Superficial	Ultraviolet exposure Very short flash	Dry, red Blanches with pressure	Painful	3-6
Superficial partial thickness	Scald (spill or splash) Short flash	Blisters Moist, red, weeping Blanches with pressure	Painful to temperature and air	7-20
Deep partial thickness	Scald (spill) Flame Oil Grease	Blisters (easily unroofed) Wet or waxy dry Variable color (patchy to cheesy white to red) Does not blanch with pressure	Perceptive of pressure only	>21
Full thickness	Scald (immersion) Flame Steam Oil Grease Chemical Electrical	Waxy white to leathery gray to charred and black Dry and inelastic No blanching with pressure	Deep pressure only	Never (if >2% TBSA)

Mertens DM, Jenkins ME, Warden GD: Med Clin North Am 1997: 32:343, Peate WF, Am Fam Physician, 1992:45:1321; Clayton MC, Solem LD: Postgrad Med 1995:97:151.

TABLE 28-2 American Burn Association: Burn Injury Severity Grading System

	SEVERITY		
Burn Type	Minor	Moderate	Major
Criteria	<10% TBSA burn in adults <5% TBSA burn in younger or older patients <2% full-thickness burn	10%-20% TBSA burn in adults 5%-10% TBSA burn in younger or older patients 2%-5% full-thickness burn High-voltage injury Suspected inhalation injury Circumferential burn Medical problem predisposing to infection (e.g., diabetes mellitus, sickle cell disease)	>20% TBSA burn in adults >10% TBSA burn in younger or older patients >5% full-thickness burn High-voltage burn Known inhalation injury Any significant burn to face, eyes, ears, genitalia, or joints Significant associated injuries (fracture or other major trauma)
Outcome	Outpatient	Admit to hospital	Refer to burn center

Reproduced with permission from Hartford CE, Kealy CP: Care of outpatient burns. In Herndon DN (ed): Total Burn Care, ed 3, Philadelphia, 2007, Elsevier.

themselves are generally not painful; however, there may be surrounding areas of partial-thickness burns that are painful. Skin appearance can vary from waxy white to leathery gray to charred and black. The skin is dry and inelastic and does not blanch with pressure. Hairs can easily be pulled from hair follicles. Vesicles and blisters do not develop.

Fourth-Degree Burns

Fourth-degree burns are deep and potentially life-threatening injuries that extend through the skin into underlying tissues such as fascia, muscle, and/or bone.[11] However, the clinical methods outlined may not provide sufficient accuracy of evaluation of burn depth to support crucial treatment decisions, such as the extent of excision and grafting required. Laser Doppler imaging (LDI) has been shown to provide a more objective measurement on which to base the decision to operate.[25]

BURN ASSESSMENT

To determine the need for referral to a specialized burn unit, an assessment tool is needed to aid in the decision making process.[5,11] Under this system, burns can be classified as major, moderate, and minor. This is assessed based on a number of factors, including TBSA burnt, involvement of specific anatomic zones, age, and associated injuries (Table 28-2).

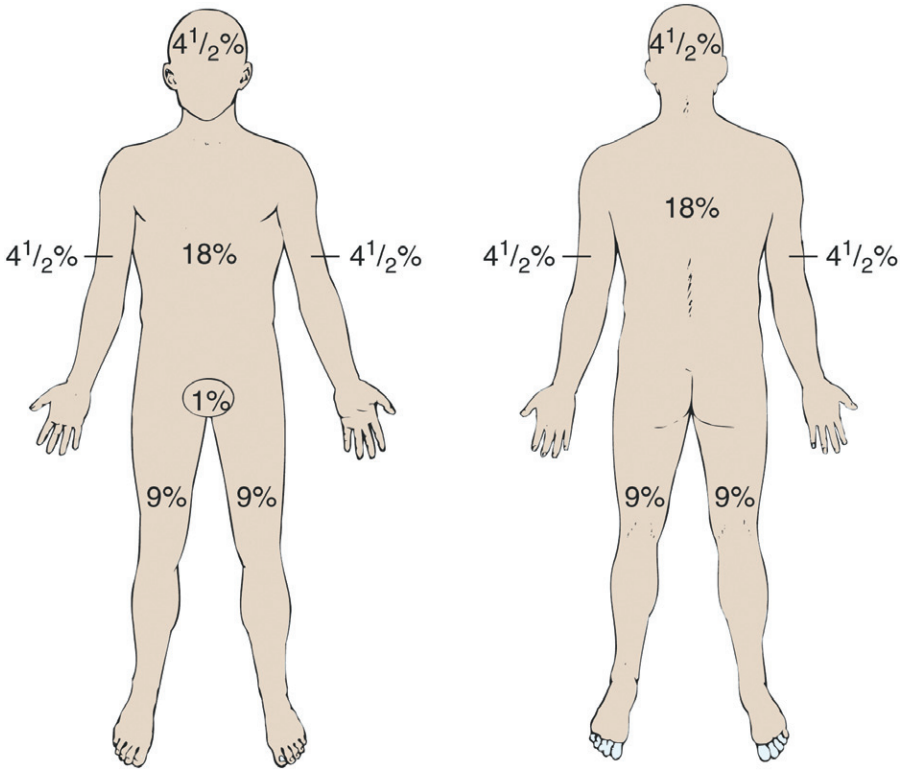


FIGURE 28-6 Burn assessment—the rule of nines. (From Veenema TG: Handbook for disaster nursing and emergency preparedness, ed 2, St. Louis, 2009, Mosby.)

Burn Size

The Rule of Nines. To approximate the percentage of burned surface area, the body has been divided into eleven sections: head, right arm, left arm, chest, abdomen, upper back, lower back, right thigh, left thigh, right leg (below the knee), left leg (below the knee). Each of these sections takes about 9% of the body's skin to cover it. Added all together, these sections account for 99%. The genitals make up the last 1% (Fig. 28-6). This rule is relatively accurate in adults, but inaccurate in children. In children, the Lund-Browder chart is the recommended method because it takes into account the relative percentage of body surface area affected by growth (Table 28-3).[11] In small burn injuries, the extent of injury can be quickly estimated because the palm of a patient's hand represents approximately 1% of the TBSA.

TABLE 28-3 Lund-Browder Chart

	AGE (YR)				
Area	**0-1**	**1-4**	**5-9**	**10-15**	**Adult**
Head	19	17	13	10	7
Neck	2	2	2	2	2
Ant. trunk	13	17	13	13	13
Post. trunk	13	13	13	13	13
R. buttock	2½	2½	2½	2½	2½
L. buttock	2½	2½	2½	2½	2½
Genitalia	1	1	1	1	1
R.upper arm	4	4	4	4	4
L. upper arm	4	4	4	4	4
R. lower arm	3	3	3	3	3
L. lower arm	3	3	3	3	3
R. hand	2½	2½	2½	2½	2½
L. hand	2½	2½	2½	2½	2½
R. thigh	5½	6½	8½	8½	9½
L. thigh	5½	6½	8½	8½	9½
R. leg	5	5	5½	6	7
L. leg	5	5	5½	6	7
R. foot	3½	3½	3½	3½	3½
L. foot	3½	3½	3½	3½	3½

Adapted from MacAfee KA II, Zeitler DL, Mayo Kathleen: Burns of the head and neck. In Fonseca RJ, Walker RV (eds): Oral and maxillofacial trauma, Philadelphia, 2007, Saunders, pp 949–966.

MANAGEMENT

INITIAL ASSESSMENT

The initial evaluation includes assessing for evidence of respiratory distress and smoke inhalation injury, evaluating cardiovascular status, looking for other injuries, and determining the depth and extent of burns. Initial assessment for the burn occurs concomitantly with the burn resuscitation.

CRITERIA FOR HOSPITAL ADMISSION

The initial problem in the management of a patient with thermal injury is to determine whether it is advantageous to admit the patient to the hospital or whether he or she

can be safely treated as an outpatient. Table 28-1 shows the indications for referral to a burn center when meeting the criteria for a major burn. The following burn patients are usually considered for admission:

1. Partial-thickness and full-thickness burns totaling >10% TBSA in patients younger than 10 or older than 50 years
2. Partial-thickness and full-thickness burns totaling >20% TBSA in other age groups
3. Partial-thickness and full-thickness burns involving the face, hands, feet, genitalia, perineum, or major joints
4. Full-thickness burns >5% TBSA in any age group
5. Electrical burns, including lightning injury
6. Chemical burns
7. Inhalation injury
8. Burn injury in patients with preexisting medical disorders that could complicate management, prolong the recovery period, or affect mortality
9. Any burn with concomitant trauma (e.g., fractures) in which the burn injury poses the greatest risk of morbidity or mortality. If the trauma poses the greater immediate risk, the patient may be treated initially in a trauma center until stable before being transferred to a burn center. The physician's decision should be made with the regional medical control plan and triage protocols in mind.
10. Burn injury in children admitted to a hospital without qualified personnel or equipment for pediatric care
11. Burn injury in patients requiring special social, emotional, and/or long-term rehabilitative support, including cases involving suspected child abuse[21]

PREHOSPITAL CARE

Eliminating the Heat Source

Eliminating the heat source is the single most important action to be taken at the scene of the injury. The involved clothing should be removed, as well as rings, watches, and other jewelry. Cooling and/or neutralization with water or water gel dressing may also be appropriate to stop the initial burning process; however, once the heat source has been removed, cooling is no longer of benefit and may result in significant hypothermia and peripheral vasoconstriction that can extend thermal damage. Cooling cannot halt the process of burning through the physical skin barrier, but there is some evidence that decreasing the amount of the prostoglandins and lessening the inflammatory process around the damaged zone is helpful.[26,27] Chemical burns should be copiously irrigated with water; however, dry chemicals should be gently brushed off the skin before irrigation is begun.

Airway Management and Oxygen Administration

Patients with upper airway burns should be intubated early, before airway anatomy becomes distorted by edema. Soot in the mouth, facial burns, and body burns may be more useful predictors of inhalation injury than symptoms of stridor, hoarseness, drooling, and dysphagia.[28] Any patient rescued from a burning building or exposed to a smoky fire should be placed on 100% oxygen via a non–rebreathing mask if there is any suspicion of smoke inhalation in awake patients.[29]



FIGURE 28-7 Patient with high suspicion of inhalation injury with perinasal and perioral burns from a house fire. Bronchoscopy is mandatory. (From Ward Booth P, Eppley B, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, St. Louis, 2012, Saunders.)

Fluid Replacement

Emergency medical personnel should place an IV line and begin fluid administration with lactated Ringer's (LR) solution at a rate of approximately 1 liter/hr in the case of a severe burn; otherwise, a maintenance rate is appropriate assuming there is no concomitant nonthermal trauma.[21]

HOSPITAL CARE

Inhalation Injury

Inhalation injury is the most common cause of death in the burn patient[30] (Fig. 28-7). Burn lesions larger than 70% of TBSA increase the risk of inhalation injury.[31] Upper airway edema can rapidly occur and distort the normal anatomy of the airway tracts. In the absence of hypoxia, there is no definitive tool to assess whether inhalation injury has occurred.[32] Given the progressive nature of burn injury in the first 48 hours, it is preferable to secure an airway early. Any patient with deep facial or intraoral burns, facial or upper airway edema, or suspected inhalation injury should be considered for early oral or nasotracheal intubation before edema causes airway compromise. There is some evidence for using aerosolized heparin and *N*-acetylcysteine to remove bronchopulmonary casts and reduce edema.[33]

Carbon Monoxide Toxicity. Carbon monoxide (CO) is a tasteless, odorless, and colorless gas present in the smoke of the combustion of organic materials, such as wood, coal, and gasoline. Because CO has a 200 to 300 times greater affinity to stay bound to hemoglobin than oxygen, the oxygen-carrying capacity of hemoglobin is reduced. The clinical findings of CO toxicity are highly variable and largely nonspecific. Symptoms and signs may include headache, nausea, malaise, altered cognition, dyspnea, angina, seizures, cardiac dysrhythmias, heart failure, and/or coma. The presence of bright, cherry red lips is an insensitive indicator of CO poisoning.[34] Pulse oximetry is an unreliable tool in measurement of the oxygen saturation in CO toxicity. Monitoring of the end-tidal CO_2 using capnography and assessing arterial blood gas levels can help determine the efficacy of the primary

resuscitation care.[35,36] An electrocardiogram (ECG) is also obtained to assess for cardiac dysfunction.

Fluid Resuscitation. A burn is a dynamic wound. Cytokines enter the circulation when the burn reaches 20% of TBSA and results in a systemic inflammatory response.[37] Prostaglandins and leukotriene cause leaking of the fluid and protein to the interstitial tissue, so cardiac output decreases and burn shock occurs. Peripheral vasoconstriction happens due to the sympathetic response leading to conversion of the zone of stasis to the zone of necrosis. Children younger than 2 years with more than 5% and any patient with more than 15% body surface area (BSA) burns will require IV fluid therapy.[5] In patients with major burns, an IV line should be placed through nonburned skin. Overadministration of fluids and electrolytes can lead to pulmonary edema, peripheral edema, and compartment syndrome.[35] The Parkland (also known as Baxter) formula is the most widely used guide to administer fluid in burn patients.[38,39] According to this formula, the fluid requirement during the initial 24 hours of treatment is 4 mL/kg of body weight for each percent of TBSA burned, given IV.[40] Superficial burns are excluded from this calculation. Half of the calculated fluid needed is given in the first 8 hours and the remaining half is given over the subsequent 16 hours.[41]

Another formula for resuscitation of burn patients is the modified Brooke formula, which recommends administering 2 mL/kg of body weight for each percent of TBSA.[35,42,43] The modified Brook formula lessens edema formation and decreases the incidence of pulmonary complications in those with preexisting cardiopulmonary disease. To avoid overhydration, resuscitate patients with a urinary output in the range of 1 to 2 mL/kg/hr for children less than 30 kg and 0.5 to 1 mL/kg/hr for those weighing 30 kg or more.[44,45] Peripheral pulses should be checked regularly, especially in limbs with a circumferential burn, to determine whether there is distal perfusion. Pulse rates are not very useful as a guide to resuscitation because tachycardia with a rate of 100 to 120 beats/min is common in even adequately resuscitated patients. Capillary refill may also be useful in assessing adequate distal limb perfusion.[46]

Tetanus Consideration

Tetanus immunization should be administered to children with burns deeper than superficial-thickness burns who have not received booster immunizations in more than 5 years.[35]

Pain Control

Although thermal injuries are usually extremely painful, burn patients frequently do not receive analgesia in the emergency department.[47] In small burn injuries, empirical analgesic therapy is with nonsteroidal anti-inflammatory drugs (NSAIDs) and, in larger burn injuries, a combination of an opioid and NSAID can be useful.[48]

Gastrointestinal Interventions

Shock from thermal burn injuries results in mesenteric vasoconstriction predisposing to gastric distension, ulceration (so-called Curling's ulcer), and aspiration. Therefore, a nasogastric tube should be placed in patients with burns more than 20% TBSA.[35,49,50]

PRIMARY BURN WOUND MANAGEMENT

Traditional management of the burn wound involves careful débridement of loose necrotic tissue, gentle cleansing of the wound with a bland soap, and application of dressings.[51] Burn wounds should initially be cleaned with mild soap and water. Disinfectants are typically avoided because they may inhibit normal wound healing. Clothing and debris that are embedded in the wounds should be removed. Débridement of devitalized tissue (including ruptured blisters) decreases the risk of infections.[35] Needle aspiration of blisters should be avoided, because this increases the risk of infection.[49,52-54] A variety of proteolytic enzymes, such as collagenase, has also been used for débridement of burn wounds.[55,56]

Wound Dressing

Superficial burns, especially minor burns in the face, do not require dressings and treatment consists of gentle cleansing with a mild soap followed by the application of a topical agent.[49] For patients who are being rapidly transferred to a burn unit, burns should be covered with dry sterile dressings.[35]

Topical Antibiotics. The goal of therapy is not to sterilize the wound but to control bacterial density and decrease the likelihood of burn wound infection. Early administration of systemic antibiotics to prevent burn wound infection is of little or no benefit and therefore is not recommended. This practice is ineffective in reducing morbidity and mortality and is likely to promote the rapid emergence of resistant microorganisms.[57] There is no consensus on which topical antimicrobial agent or dressing is best suited for burn wound coverage to prevent or control infection.[58] They are generally divided into potent agents used to prevent burn wound invasion (e.g., silver sulfadiazine, mafenide acetate, silver nitrate) and milder agents (e.g., bacitracin, Neosporin, Polysporin, mupirocin) used to treat small or superficial wounds. The more potent agents may delay epithelialization and should be reserved for use in managing more extensive and deeper burns. The milder agents, when used in combination with nonadherent gauze, provide a comfortable protective environment that promotes epithelialization of the wound.

Silver-Containing Dressings. Silver-containing dressings slowly release silver into the wound. Activated silver has broad spectrum antimicrobial activity and may also have an anti-inflammatory benefit.[59] Silver nitrate solution (0.5%) is an effective agent but has decreased in popularity over the past 2 decades. It is painless on application, has a wide spectrum of antimicrobial activity, and has no known bacterial resistance. Its use is limited due to its staining, requirement for greater nursing care, and the leeching of electrolytes from the wound. Paraffin gauze over a silver-based dressing was shown to be effective in superficial burns.[60]

Silver Sulfadiazine. Silver sulfadiazine cream (SSD) is the most commonly used topical agent for dressing.[58] It is bacteriostatic but poorly diffusible and limited in its penetration of the burn wound. It is painless on

application and has a soothing effect. The antimicrobial spectrum of SSD includes *Staphyloccus aureus, Escherichia coli, Enterobacter,* and *Candida albicans.*[46] Transient leukopenia has been reported in up to 5% of patients; this usually resolves spontaneously, even with continued use of the drug.[61] SSD should not be used in women who are pregnant or breastfeeding, or in infants younger than 2 months.[56,62]

Chlorhexidine. Chlorhexidine gluconate, a long-lasting antimicrobial skin cleanser, is often used with a gauze dressing for burn wound coverage in superficial partial-thickness burns. Chlorhexidine dressings do not interfere with wound reepithelialization, in contrast to silver sulfadiazine.[56]

Mafenide Acetate. Mafenide acetate (MA) cream is bacteriostatic, freely soluble, and readily diffuses through burn eschar to the viable tissue interface. This agent also has the broadest spectrum against *Pseudomonas* spp. and gram-negative organisms. MA inhibits protein synthesis in *P. aeruginosa.* Up to 90% of the dose enters the wound within 5 hours and it reaches peak concentration within 1 to 2 hours. Its concentration decreases to subinhibitory levels within 10 hours; therefore, it must be applied at least twice daily.[46] The principal limitations of this agent are the pain produced when applied to partial-thickness wounds and the inhibition of carbonic anhydrase that predisposes to the development of metabolic acidosis. Use of this agent is generally limited to wounds with or at high risk for invasive infection.[56,63]

Biosynthetic Dressings. Tissue engineering has progressed in the last decade and can now be applied for the replacement of injured tissue. Bioengineered skin dressing, also called semibiologic skin substitute, is used to increase the healing potential of the recipient bed and decrease the number of dressing changes.[56,64] Biobrane and Integra have been recognized by the U.S. Food and Drug Administration (FDA) as wound dressing materials. Both substitute as a matrix-like structure for harvesting fibroblasts and forming collagen. Following the activity of the fibroblast cells, the endothelial cells can also promote vasculogenesis, which can help with management of the partial-thickness burn.

Escharotomy

Mechanical obstruction of the airway, as well as distal tissue compartment syndrome, can occur due to the eschar formation in the neck and chest area. Releasing incisions during primary wound management can help provide distal tissue pressure that does not exceed 30 mm Hg.[35,65]

SECONDARY BURN WOUND MANAGEMENT

Full-thickness or partial-thickness burns that fail to heal within 3 weeks should be excised and treated with grafting. An early appropriate decision for the burn excision and grafting can lead to shorter hospitalization and fewer complications.[66] If early excision and grafting is the treatment of choice, it may be a one- or two-stage technique. With the one-stage technique, the operation consists of excision of the burn to viable tissue and the placement of a graft. In the two-stage technique, the first operation is for the excision of the burn to viable tissue and coverage with a biologic dressing. The wound is reevaluated and grafted 24 to 72 hours later.[67] Critical areas must be grafted first to achieve optimal functional and cosmetic results. Blood loss is a major consideration in excisional therapy. In adults, approximately 200 mL of blood is lost per percentage of TBSA excised and grafted.[68] In children, blood loss is approximately 3% to 4% of the circulating blood volume per percentage of TBSA excised and grafted.[69] Following burn wound excision, skin grafts are applied to the viable tissue bed. Split-thickness skin grafts are harvested with a dermatome at a depth of 0.008 to 0.016 inch (0.2 to 0.4 mm). For small burns, full-thickness skin grafts (with primary closure of the donor site) should be used because they result in minimal donor site morbidity and excellent long-term functional and cosmetic results. For larger burn defects, meshed or nonmeshed split-thickness skin grafts can be used. Allogenic cadaveric skin from a human tissue bank also would be an option for a total-body burn patient for whom additional donor site for grafting is not available. TransCyte is a cultured epidermal autograft produced by culturing dermal fibroblasts onto a synthetic scaffold, which consists of a mostly nylon mesh. Skin fragility and susceptibility to infection are the disadvantages.[56]

Facial Burns

The head and neck area is the anatomic site most frequently involved in burn injuries. Facial burn injuries can produce devastating cosmetic and social alterations, which can affect self-image and societal perception. Marked edema can develop with partial-thickness burns in the face due to the looseness of the tissue and rich blood supply. Inhalational injuries should be suspected in any facial burn. The basic principles of grafting procedures performed on the head and neck must include the importance of aesthetic facial units. When grafting is undertaken, replacing an entire aesthetic unit is indicated, rather than applying grafts in patches.[70] Skin grafts to the face are thicker than those used elsewhere in the body to provide less contracture; they are not meshed to enhance aesthetics.[71] Grafting of the face is done by aesthetic region and every effort should be made to reconstruct the dermatologically defined facial units, including the forehead, eyebrows, upper eyelid, lower eyelid, cheek, upper lip, lower lip, and chin. Immobilization of the graft by pressure dressings, nasogastric feedings, and avoidance of speaking are essential.[46] Donor sites of good quality for the face are the scalp, neck, supraclavicular region, and inner thigh or arm.[67] Flame or contact burns in the head and neck may occur concomitantly with facial trauma in MVAs (Fig. 28-8).

Eye Burn Injury

Chemical burns of the eye constitute ocular emergencies. Acid burns of the eye are much better tolerated than alkali burns, which may result in injury ranging from mild corneal erosions to severe and generalized eye burns that manifest as blurring of the pupil and blanching of the conjunctiva and sclera.[14,71,72] The initial treatment is copious irrigation with normal saline. Staining of the eyes with fluorescein is performed to detect corneal injury. If corneal abrasion is present, a topical antibiotic



FIGURE 28-8 Facial burn associated with maxillofacial trauma.

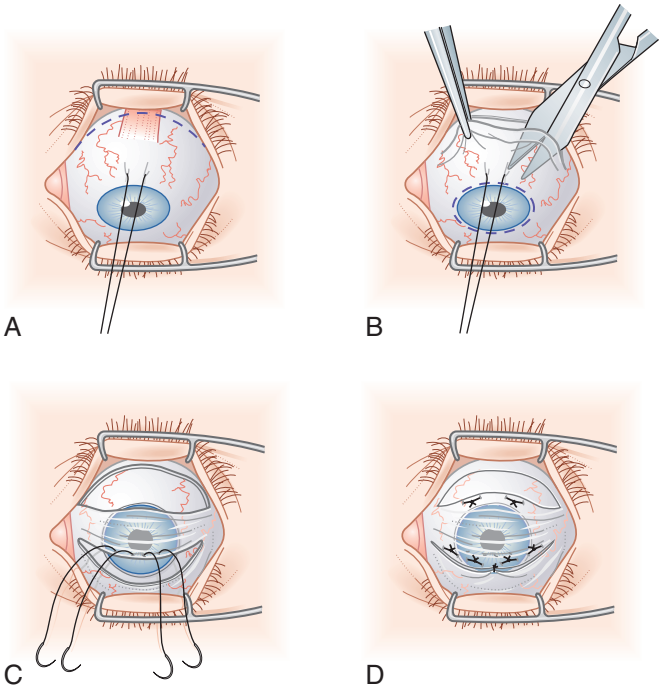


FIGURE 28-9 In severe full-thickness eyelid burns, the globe can be protected by the raising and closing of upper and lower conjunctival flaps, which are then covered by a skin graft. **A,** Outline of conjunctival flaps. **B,** Sharp elevation of conjunctival flaps. **C,** Suturing upper and lower conjunctival flaps together for corneal coverage. **D,** Vascularized conjunctival bed for skin graft placement. (From Ward Booth P, Eppley B, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, St. Louis, 2012, Saunders.)

is prescribed. The main complications of chemical burns of the eye are symblepharon and corneal ulceration.

Eyelid Burn

The eyelid as a protector of the globe may be involved in most burn injuries of the face (Fig. 28-9). The eyelid has the thinnest skin on the face, which makes it susceptible to early contracture and exposure of the cornea. An ophthalmologist should be consulted because corneal abrasion and exposure keratitis are the most common associated sequelae. An early grafting with tarsorrhaphies should be considered in management of the eye burn. In severe full-thickness eyelid burns, the globe can be protected by the raising and closure of the upper and lower conjunctival flaps, which are then covered by a skin graft. Split-thickness grafts for the upper lid and full-thickness grafts for the lower lid are generally indicated.[73] With superior lid débridement, one should be aware of the levator musculature and possible lid ptosis. The most common complication of eyelid burns is contracture and the development of ectropion.

Brow Burn

A number of reconstructive techniques are available for full-thickness brow burns, including punch grafts, single-hair transplants, temporal vessel-based scalp flaps, and composite hair- bearing scalp grafts. The composite scalp graft should be no wider than 3 mm in a vascularly compromised bed. Multiple strips can be grafted and the intervening skin excised after healing. The procedure can be performed under local anesthesia, with good success.[74,75]

Ear Burn Injury

Lack of subcutaneous tissue, thinning of the skin, and lateral prominence of the ear can aggravate the severity of the burn in this area (Fig. 28-10). Direct injury to the ear cartilage by burn and/or secondary infection can lead to chondritis. The organisms most commonly responsible are *S. aureus* and *P. aeruginosa*.[76] Ear injuries may need local débridement or resection, but a regimen with minimum débridement and use of a topical antibiotic may be useful. Avoiding pressure on the ear is another important aspect of primary management. If chondritis occurs, it can be managed by removal of the affected cartilage. Attempts to salvage viable cartilage may be made by burying cartilage in soft tissue pockets and later performing secondary graft reconstruction. Some patients may require local regional temporalis fascia flaps for coverage. Denuded cartilage can be covered by skin grafts or local cutaneous advancement flaps from the retroauricular region. Flaps are elevated in the supraperichondrial plane. Small segments of nonviable cartilage can be removed without altering aesthetics. Larger defects may need to be reconstructed with a composite graft.[46,77] For total ear reconstruction, the use of osseointegrated implants and prosthesis is probably the best treatment.[78]

Nasal Burn Injuries

The nose has a prominent feature in the face and burn injuries are common. The thick skin on the lower portion of the nose can provide some protection to this area, but the skin across the lateral aspect and bridge of the nose



FIGURE 28-10 Partial-thickness ear burns. **A,** Superficial partial-thickness ear burn 3 days after injury. **B,** 3 months postinjury with spontaneous healing. **C,** Deeper part-thickness ear burn on admission. **D,** Complete healing by 1 month after injury. **E,** Combination partial and full-thickness ear burn 3 days after admission. (From Ward Booth P, Eppley B, Schmelzeisen R: Maxillofacial trauma and esthetic facial reconstruction, ed 2, St. Louis, 2012, Saunders.)

is thin.[72] Generally, the nose is allowed to heal by spontaneous epithelialization. A deep partial-thickness burn defect of the nose requires a full-thickness skin graft. Maintaining skeletal cartilage support is an essential factor for the optimum aesthetic result. Auricular cartilage provides a contoured graft material and costochondral rib grafts also may be sculpted.[46] Nasal injuries can be devastating and hard to reconstruct. Flattening of the alar region is often encountered secondary to contracture. Intraoral flaps, because of their proximity, can be used in reconstruction of the lower third of nose structures.[79] Providing a vascular bed in scar recipient sites can increase the viability of the composite graft (Fig. 28-11).

Scalp Burn Injuries

Scalp burns are often partial-thickness burns due to the thickness of the skin in this anatomic area and deep placement of the hair follicles. Split-thickness skin grafts can be used in partial-thickness injuries. Rotational flaps or tissue expansion can be part of the treatment plan in



FIGURE 28-11 **A,** Depressed lower left alar rim due to a previous burn. **B,** Pedicle rotational intraoral flap for reconstruction of the avascular bed. **C,** Suturing of the helical composite graft over the mucosal flap. **D,** 12-month follow-up of the patient. (Courtesy Dr. F. Pourdanesh, Shahid Beheshti University of Medical Sciences, Tehran, Iran.)

a highly aesthetic area. Perforation of the cortical bone for the appropriate vascularization can help in better survival of the flap. In severe burns, removal of the outer table of the cortical bone is an alternative option. Technetium bone scans can be used to show a lack of perfusion in areas of nonviable bone and increased uptake in areas of bone sequestration and regeneration.[71,80]

Mouth Burn Injuries

Burns that involve one or both lips can lead to severe microstomia. Hypertrophic scars in this area lead to difficulty in eating or intubation of the burning patient. Oral splinting devices should be fabricated and inserted as soon after the burn as possible. In a child, the appliance conforms to the teeth and stabilizes the commissure by an attached horizontal bar. Adults are generally more cooperative and will wear a mouth splint, such as the one depicted in Figure 28-12.[81] The most frequently encountered burn injury in children is an electrical burn injury. Electrical burns of the mouth predominate in 1- to 2-year-old children and generally result from putting the socket terminal of an extension cord into the mouth or sucking on the wall socket.[82] The tongue, lower and upper lips, and commissures all may be affected.[14,83] The oral commissure burn deserves special mention because it is not uncommon and is easily treated. Typically, the burn area is sharply demarcated and the eschar is separated slowly. Conservative treatment with an orthodontic appliance to avoid microstomia is the first step. The second step would be correction of the scar, especially in the vermilion border. Upper and lower lip grafts can be placed to reconstruct the vermilion. Mucosal advancement flaps can also be used. Later, secondary reconstruction may be necessary, including scar-releasing procedures.

See Figures 28-13 to 28-18 for illustrations of repair of various facial burn injuries.

SUMMARY

Complex burn reconstruction requires all the skills of the facial reconstructive surgeon. In the acute phase, patients frequently have concomitant medical and metabolic abnormalities requiring a team approach for comprehensive treatment. Burn patients are ideally treated in centers dedicated to burn care. Oral and maxillofacial surgeons are an essential part of the burn team for facial and oral reconstruction.

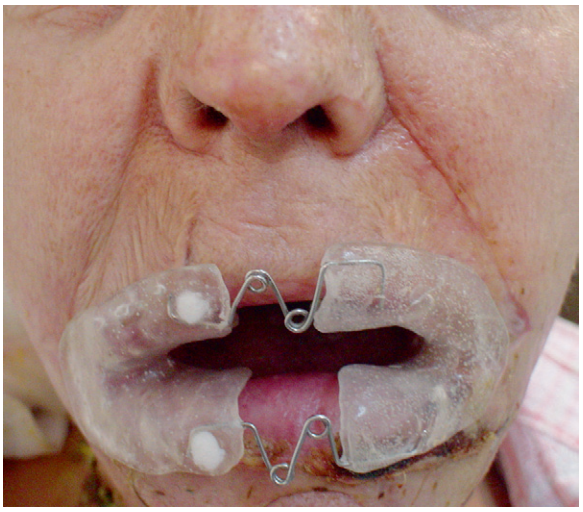


FIGURE 28-12 Custom-made device for the inhibition of the microstomia following burn.

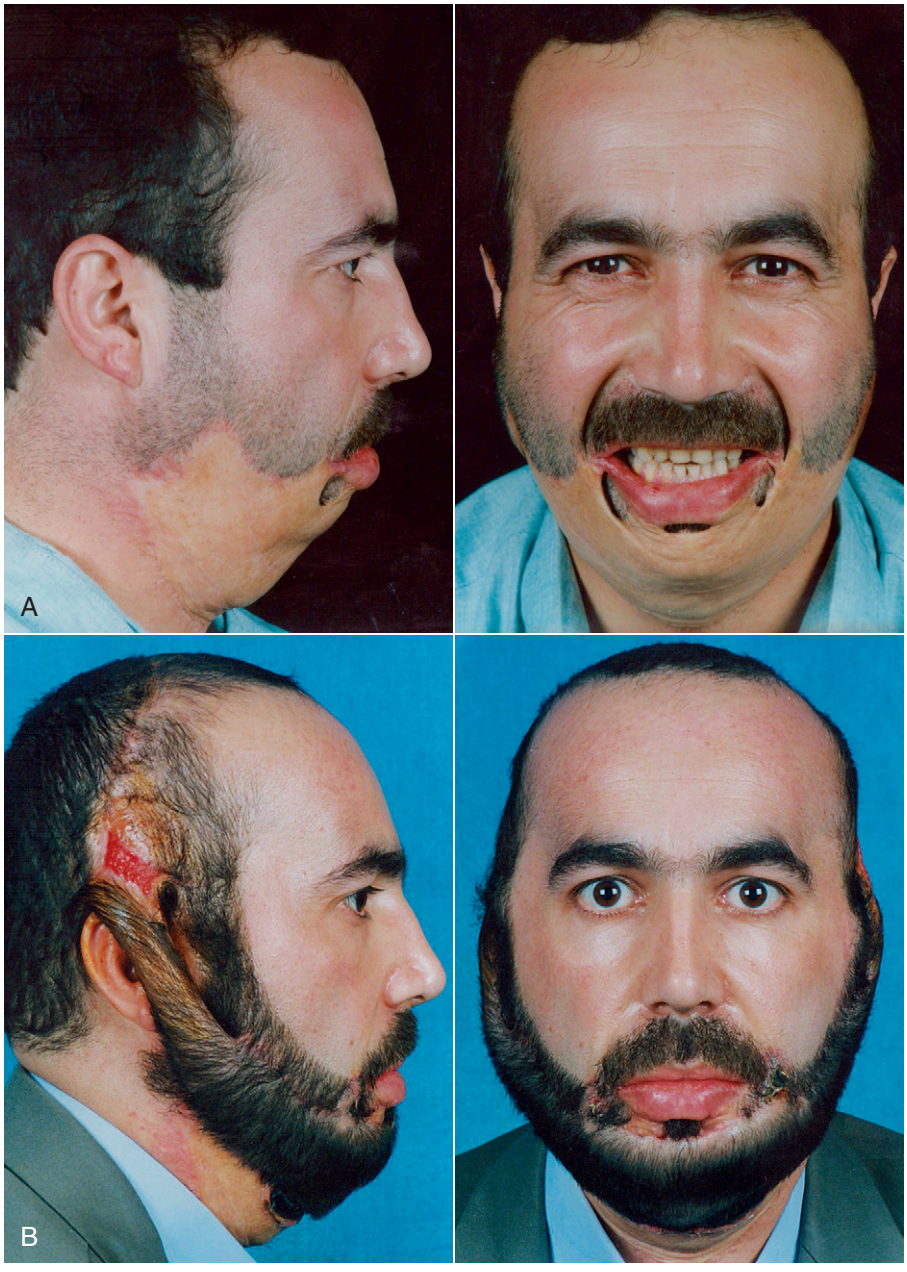


FIGURE 28-13 **A,** Patient not happy with lower face skin graft and would like to have a beard. **B,** Bilateral rotational scalp skin graft.

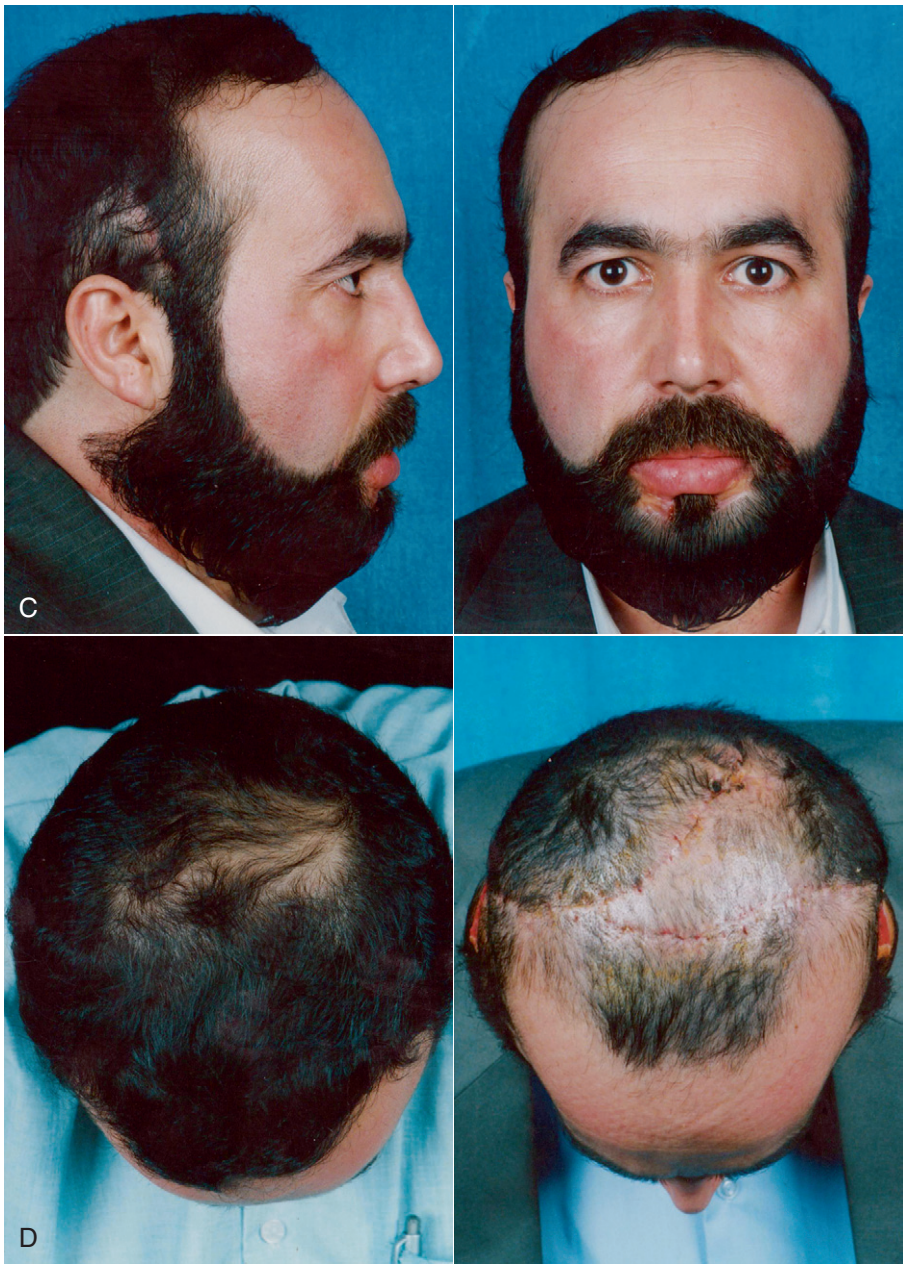


FIGURE 28-13, cont'd **C,** Final postoperative result after a few months. **D,** Preoperative and postoperative views of vertex. (Courtesy Dr. Hossein Haghshenas.)



FIGURE 28-14 Almost total facial burn, including neck, midface, lower face, and right ear. (Courtesy Dr. Hossein Haghshenas.)



FIGURE 28-15 Multiple facial reconstruction with skin graft and forehead flap for nasal reconstruction. (Courtesy Dr. Hossein Haghshenas.)



FIGURE 28-16 **A,** Application of tissue expander at left neck for reconstruction of lower face burn. **B,** Postoperative photograph. (Courtesy Dr. Hossein Haghshenas.)

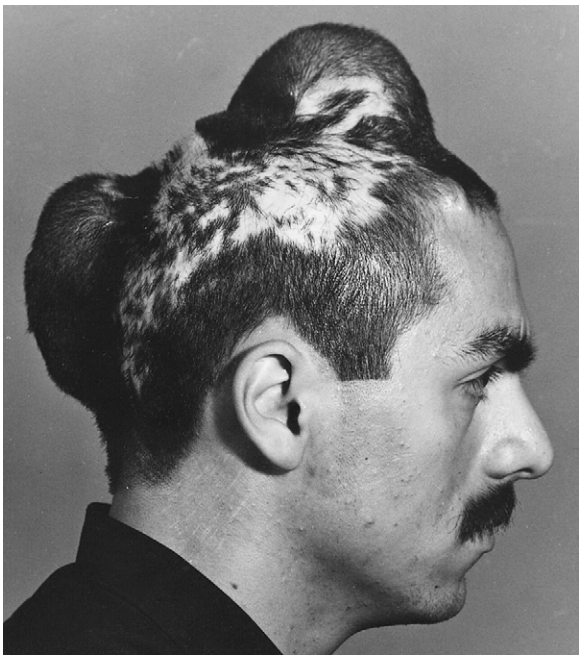


FIGURE 28-17 Double tissue expander application for reconstruction of a scalp burn. (Courtesy Dr. Hossein Haghshenas.)



FIGURE 28-18 Total facial reconstruction, including nose, facial soft tissue, lower lip and right brow graft. (Courtesy Dr. Hossein Haghshenas.)

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CHAPTER

29

Management of Pediatric Facial Fractures

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OUTLINE

Anatomic Considerations
Epidemiology of Facial Fractures
Diagnosis
Clinical Examination
Radiologic Examination
Types of Fractures
Dental Trauma
Alveolar Fractures
Midfacial Fractures
Frontal Sinus Fractures and Nasal-Orbital-Ethmoid Fractures
Fractures of the Zygomatic Complex
Mandibular Fractures

ANATOMIC CONSIDERATIONS

The primary differences in craniomaxillofacial trauma in the pediatric or growing patient compared with the adult patient are based on developmental anatomy. At birth, the cranium-to- face ratio is 8:1. This decreases to 4:1 by age 5 and 2.5:1 as an adult.[1] As such, in infants, a large cranium protects the face, although infants are more susceptible to cranial trauma. As the child grows older, the cranium-to-face-ratio decreases, making the child more susceptible to facial fractures, in particular midface fractures.[2] The orbits reach skeletal maturity early in life (5 to 7 years of age); therefore, the lower third of the face is relatively protected during childhood. During the mixed dentition years, the mandibular growth catches up with the rest of the facial skeleton in an anterior and downward pattern. Skeletal maturity of the facial skeleton occurs at approximately 14 to 16 years of age in females and 16 to 18 years of age in males.[3] Palatal, midaxillary, and premaxillary suture growth are completed by age 12 years.[4]

Unique to the jaw of growing patients is the consideration of dental development and the potential complications and morbidity that arise from surgical manipulation in the region of developing teeth. By the age of 2 years, chin prominence develops and the primary dentition begins to erupt. Transverse maxillary growth is complete with palatal, premaxillary, and midline maxillary suture growth complete and obliterated by ages 8 to 12 years.[5] The deciduous dentition starts with the replacement of the permanent dentition by 6 years, representing a period of mixed dentition. As the mandible continues to grow, it lengthens and widens to accommodate developing teeth. Growth of the mandible continues by deposition posterior and resorption anterior to the ramus.[6]

The pediatric maxillofacial complex is also malleable, because there is a greater cancellous-to-cortical ratio. As such, greenstick fractures of the facial skeleton occur more frequently in children compared with adults. The consistency of the growing patient's bone (more cancellous than cortical) is less conducive to the use of screw or wire fixation to fixate fractures internally. For these reasons, closed reduction is a viable option for most facial fractures in the growing patient. The osteogenic and bone remodeling potential of a child exceed that of an adult. Fractures of the maxilla and mandible that are not reduced within several days of injury often cannot be adequately reduced because of the rapid bone healing that occurs.

The contour heights of the crowns of deciduous teeth are below the gingival level, which does not lend itself well to circumdental wiring when arch bars are necessary to stabilize fractures during childhood. In addition, resorption of roots and attrition of deciduous teeth make these teeth less stable in keeping arch bars in place. It is often necessary to supplement circumdental wiring with skeletal wiring (e.g., piriform aperture, circumzygomatic, or circummandibular) to maintain adequate fixation of arch bars or splints when relying on the deciduous or mixed dentitions.[7] When placing these skeletal wires, care must be taken not to pull them through the child's soft bone and the position of the developing canine must be considered in the piriform region. In addition to skeletal fixation, the clinician can use Risdon wires in the pediatric patient with complete primary dentition or in the case of mixed dentition.

EPIDEMIOLOGY OF FACIAL FRACTURES

Approximately 8.5 million children are evaluated annually in emergency rooms in the United States.[8] An estimated 11.3% of pediatric emergency room visits overall are a result of craniofacial injuries.[9] Facial fractures are less common in the growing patient than in adults. In an analysis of 1500 facial fractures by Rowe,[10] 5% of all facial injuries occurred in children younger than 12 years and less than 1% of these fractures occurred in children younger than 6 years. Midface fractures in children accounted for less than 1%, with 4% of these fractures being variations of Le Fort type I, in part attributed to

follicular crypts and developing dentition in the maxilla for children younger than 6 years.[11] The lower incidence of fractures in children compared with adults is also secondary to the underdeveloped facial skeleton in children as well as increased support form unerupted dentition. A 2008 survey from the National Trauma Data Bank (2001 to 2005) identified 277,008 pediatric trauma patient admissions, including 12,739 (4.6%) who sustained facial fractures. Of the 12,739 patients, 32.7% sustained mandibular fractures, 30.2% nasal bone fractures, and 28.6% maxillary-zygomatic fractures. Nasal and maxillary fractures were the most common in patients younger than 1 year; mandibular fractures were the most common among teenagers. Of those with mandible fractures, symphyseal, angle, and body were most common areas of fracture; 25% of all these patients required operative intervention. Finally, 68% of pediatric fracture patients were male; motor vehicle accidents (MVAs) accounted for 55% of pediatric facial fractures, followed by assault (14.5%), and falls (8.6%).[12] Literature on the incidence of pediatric facial trauma has indicated that 1.5% to 8% of all facial fractures occur in children younger than 12 years and 1% or less of such trauma occurs in children younger than 5 years.[13] A 2008 Swiss survey of 291 pediatric maxillofacial trauma patients found that 64% were secondary to falls, 22% were secondary to MVAs, and 9% were sports-related accidents.[14]

Dental trauma in the growing patient, as isolated injuries or associated with facial fractures, has been studied extensively. Andreasen, examining a European population, estimated that one in every other child suffered dental injury by the age of 14 years.[15] In the American population, age-specific, population-based incidence of dental trauma to the incisor teeth between the ages of 6 and 50 years has been estimated to be 24.9%.[16]

A 2011 study of 772 patients from the University of Pittsburgh found that the 69% of pediatric facial trauma patients were male, with an average age of 10.7 years. In children younger than 5 years, 56.4% sustained orbital fractures. Falls were the most common mechanism of injury. In children between the ages of 6 to 11 years, orbital fractures were the most common fracture type, with MVAs as the most common mechanism of injury. The 12- to 18-year-old age group comprised almost half of patients in this study. Orbital fractures were again the most common injury, primarily attributed to interpersonal violence. Of the 772 patients, 55% had associated injuries, particularly cervical spine and neurologic trauma (primarily concussions). The incidence of associated neurologic injury decreased as the age of the patient increased. This study also demonstrated the importance of seat belt and helmet use; 45% of pediatric patients injured in this study from MVAs were unrestrained and 67% of patients involved in bicycle and all-terrain vehicle (ATV) accidents were not wearing helmets.[17] In addition, young children using seat belts too soon, rather than other more appropriate means of car restraints, sustained facial fractures 1.6 times more than those appropriately restrained for their age. Of pediatric facial fractures observed in MVAs, 51.4% were nasal fractures, 15.5% were mandibular fractures, 11.6% were orbital fractures, and 8.7% were fractures in the zygoma and maxillary bones.[18] In addition, Winston et al have found that of 13,853 children between the ages of 2 and 5 years involved in MVAs, those inappropriately restrained in seat belts rather than child safety seats, suffered a fourfold increased chance of having significant head trauma.[19]

DIAGNOSIS

CLINICAL EXAMINATION

As with all trauma patients, primary survey of the pediatric patient is indicated, specifically ensuring that the airway is patent, the patient is breathing, and vital signs are stable. Airway assessment in the child is of particular importance because the smaller airway of the child increases the relative airway resistance and ease of obstruction, and the threshold for intubating a child with injury or obstruction of the airway should be low. Once the child's airway and cardiopulmonary status have been stabilized, secondary assessment is completed to identify all areas of injury. In particular, with facial trauma, it is important to rule out neurologic injury.

Prior to examination, a comprehensive history should be obtained. Specific questions focus on the cause of injury, time frame from injury to evaluation, and any history of loss of consciousness. In addition, if dental trauma is suspected, questions regarding loss of dentition are indicated. If teeth were avulsed from the injury, it is important to determine factors such as the location of the teeth, transport medium of the teeth, and whether the teeth were rinsed or swallowed or aspirated. Finally, obtaining a basic past medical history, if feasible, is warranted. The battered child complex must be considered a possibility when the historian's account does not correlate with the extent of the patient's injury. This suspicion must be addressed if unexplained bruises, burn marks, or repeated traumatic incidents appear in the child's medical history or are discovered on physical examination.

Initial examination is focused extraorally by first observing for edema, ecchymosis, or lacerations. Chin lacerations, in particular, are often associated with condylar or symphyseal fractures. Facial edema, periorbital ecchymosis, subconjunctival hemorrhage, subcutaneous emphysema, and nasal bleeding are all indicators of possible facial fracture. Assessment of the trigeminal nerve function can also provide clues about possible injury due to fragments impinging on the peripheral trigeminal nerve branches. Examination should be followed by palpation of the facial skeleton, noting any steps or crepitus. The presence of postauricular ecchymosis (Battle's sign) or hemotympanum is suggestive of a basal skull fracture. A basic ophthalmologic examination should be completed, if possible, including assessment of pupillary reactivity, visual screening, and extraocular movements. If orbital trauma in suspected, an ophthalmologic consultation is indicated. Identification of intraoral ecchymosis, especially within the mucobuccal folds or sublingual area, should alert the examiner to the probability of facial fractures. Furthermore, palpation for steps intraorally, assessment of occlusion, presence of ecchymosis in the floor of the mouth, and mobility of dental segments are all part of a comprehensive clinical examination. Assessing mandibular range of motion and any deviations may indicate facial fractures—in particular,

fractures involving the mandibular condyles. Another often forgotten part of the intraoral examination is assessing the dentition, most importantly counting all teeth and accounting for any missing dentition. If missing dentition is unaccounted for, a chest radiograph is indicated to rule out aspiration of dental hard tissue. A neurosurgical consultation is mandatory if there is loss of consciousness, altered mental status, postauricular ecchymosis, cerebrospinal fluid (CSF) rhinorrhea, facial nerve changes, or hemotympanum. Children are prone to the development of epidural hematomas; it is critical to observe their behavior and level of consciousness following significant facial trauma.

RADIOLOGIC EXAMINATION

A combination of the clinical examination coupled with a radiographic evaluation allows the clinician to diagnose facial fractures. Radiographs should not be used solely to diagnose facial trauma. In the pediatric population, greenstick fractures are often not visualized by conventional radiographs and developing tooth buds may also obscure fractures on plain films. The simplest radiograph to diagnose mandibular, alveolar, or dental trauma is the panoramic radiograph. This film allows for clear assessment of all aspects of the mandible, assessing dentition and tooth buds, and providing a global view of the maxilla and mandible. Limitations of the panoramic film include its two-dimensional nature, distortion of the anterior maxilla and mandible, and inability to differentiate greenstick fractures from bicortical fractures. When dental trauma or alveolar trauma is suspected, dental radiographs including occlusal films and/or periapical radiographs are helpful.

Panoramic and dental radiographs may not be readily available in the emergency department. If this is the case, another choice is plain radiographs or skull films. A complete facial series of radiographs should include left and right lateral oblique views of the mandible to observe the mandibular body and ramus, a Towne projection to identify condylar injuries, a posteroanterior view to examine the mandible and midface, a Waters view for midfacial and nasal fracture detection, and a submental vertex view for visualization of the zygomatic arches. As technology has evolved, 3-mm axial, coronal, and sagittal computed tomography (CT) for imaging in pediatric facial trauma patients has become routine and is now the standard of care, replacing plain films in many institutions. Sagittal images are particularly useful for evaluating orbital floor trauma. Finally, three-dimensional CT is now readily available and should be performed for all complex facial fractures to assess facial fractures globally from multiple angles and assist with surgical planning. Three-dimensional CT imaging is also preferred to assess postoperative outcomes of reduction and internal fixation, particularly injuries in those areas not well visualized by plain films, such as midface trauma and orbital floor injuries.

TYPES OF FRACTURES

DENTAL TRAUMA

Children younger than 6 years have only primary dentition. Children 6 to 12 years of age (or slightly older) will have a mixed dentition of primary and adult teeth. Teenagers and older patients will have adult dentition. Usually, the anterior central incisors are prone to injury due to their position. Trauma to the dentition in the pediatric patient can be divided into primary dentition and secondary dentition. In general, trauma to primary dentition is treated via extraction, although restoration of primary dentition may be warranted if the dental trauma is mild or if there is a concern about space maintenance. If a primary tooth is avulsed, it is not recommended for replantation, unlike permanent dentition. When permanent dentition is injured, treatment is based on the degree of injury. Traumatized adult (secondary) teeth can be classified via the Ellis classification of dental injury:

- Ellis type 1—fractures of the crown that only affect the enamel
- Ellis type 2—fractures of the crown that affect the enamel and dentin
- Ellis type 3—fractures of the crown that affect the enamel, dentin, and pulp chamber
- Dental subluxation—displacement or mobility of teeth secondary to damage to the periodontal ligament
- Dental avulsion —tooth is extracted or lost from the oral cavity

Ellis types 1 and 2 injuries are typically treated via dental restorations. Ellis type 3 requires root canal therapy followed by dental restoration. Dental subluxation often will require splinting of the subluxed tooth to adjacent teeth for 3 to 4 weeks to stabilize the tooth. The treatment of avulsed teeth is based on the time from injury to treatment. The ideal treatment is to reimplant an avulsed tooth immediately after avulsion. It is important not to wash the tooth to ensure that the periodontal ligament is not washed away. If reimplantation is not immediately feasible, the tooth should be transported ideally in saliva. However, keeping the tooth in the vestibule of the mouth during transport is not advisable in a pediatric patient secondary to risk of aspiration; thus, transporting the tooth in a cup of the patient's saliva is preferred. If less than 2 hours has passed since avulsion, the tooth may be replanted directly into the site. If more than 2 hours has passed, the tooth should be rinsed off (at >2 hours out of the mouth, the periodontal ligament has most likely necrosed), pulp chamber obturated, and then replanted. In all cases, once replanted, the tooth needs to be splinted for stability and placed out of direct occlusion. It is important to note that once a tooth has been repositioned and splinted, the patient's occlusion should be checked to ensure that the injured tooth has been repositioned into its pretrauma position.[20]

ALVEOLAR FRACTURES

Alveolar fractures involve the supporting bone of the dentition. These are considered the most common type of pediatric facial fractures[4] and may be often associated with dental trauma or tooth avulsion. Classically, an alveolar fracture may have a segment of teeth that are mobile as a group, with associated soft tissue injury and malocclusion. Primary treatment is conservative, consisting of immobilizing the arch segment using an arch bar, wire ligation, or a composite supported orthodontic wire extended to stable teeth in the injured arch. Significant alveolar injury may result in alveolar bone loss and loss of

primary or permanent dentition. If there is bone loss and loss of primary dentition, consideration must be given to whether the underlying adult dentition will erupt into adequate bone. If the alveolus and permanent dentition are lost, consideration must be given to replacing the alveolar bone and dentition. Ideally, tooth replacement via dental implants should be considered; however, the placement of dental implants should be delayed until alveolar growth is complete. Patients can be managed with a removable prosthesis of fixed bridgework until they are old enough to be candidates for dental implant placement. Patients with significant alveolar bone loss can have the alveolar bone reconstructed via autogenous bone grafting and standard dental reconstruction.[21]

MIDFACIAL FRACTURES

Nasal Fractures

Nasal fractures are the second most common type of pediatric facial fractures.[4] A complete history and clinical findings such as epistaxis, nasal-periorbital ecchymosis, nasal edema, nasal septal ecchymosis, or associated lacerations may indicate the presence of a nasal fracture. Provided the child permits a physical evaluation, palpation of the nasal bones for bony irregularities is then completed. Additionally, intranasal speculum examination is important to rule out a possible septal hematoma, which (if present) requires emergent evacuation to avoid septal cartilage necrosis or resorption, resulting in a saddle nose deformity.[22] Fractures of the nasal structures that occur before growth is completed should be managed like those in the adult. Epistaxis is common with nasal trauma and can be controlled by local means by pinching the nostrils for 5 to 10 minutes, with the head slightly elevated. Although rare, local means of control may fail and the surgeon must surgically cauterize the region with silver nitrate or minimal electrical cauterization or by placement of a balloon inflation catheter, angiographic embolization, or ligation of vessels. CT will allow the clinician to visualize the direction and degree of displacement of the nasal bones.

Treatment consists usually of closed reduction if the injury is less than 1 week old. Older children with minimally displaced nasal bone fractures may be compliant enough to allow closed reduction to be performed under conscious sedation in the emergency department or office setting. However, in younger children or with significantly displaced nasal bone fractures, general anesthesia is indicated to ensure proper reduction of the fracture. Nasal elevators allow for reduction of the nasal bones from an intranasal approach. Nasal packing and splinting are required after satisfactory reduction of the fracture for stabilization. If the injury is more than 1 week old or there is an existing laceration providing access to the nasal facture, open reduction could be considered. Secondary rhinoplasty procedures can be considered after facial growth has been completed, generally after age 16 years.

Maxillary Fractures

Isolated Le Fort fractures are rare in the pediatric population (>10% of all facial fractures).[4,11] Unique characteristics of the child's maxilla include quantity of cancellous bone, unerupted teeth, and underdeveloped maxillary sinuses. The piriform aperture and zygomaticomaxillary buttress are much thicker structures and the soft tissue contains more adipose tissue in the child than in the adult. As such, considerable force is necessary to disrupt the midfacial skeleton of the growing patient. Pediatric Le Fort fractures often occur in combination with other trauma and these concomitant injuries may often be fatal.

Physical examination often reveals the classic signs of a LeFort fracture—maxillary vestibular ecchymosis, facial edema, malocclusion, and gross mobility of the maxilla. For higher level Le Fort fractures (type 2 or 3) there may be periorbital edema, traumatic telecanthus, and periorbital ecchymosis. CT scans help provide a definitive diagnosis (Fig. 29-1*A*). Closed reduction is the treatment of choice; however, in the primary and mixed dentition phases, this presents a challenge because arch bars are

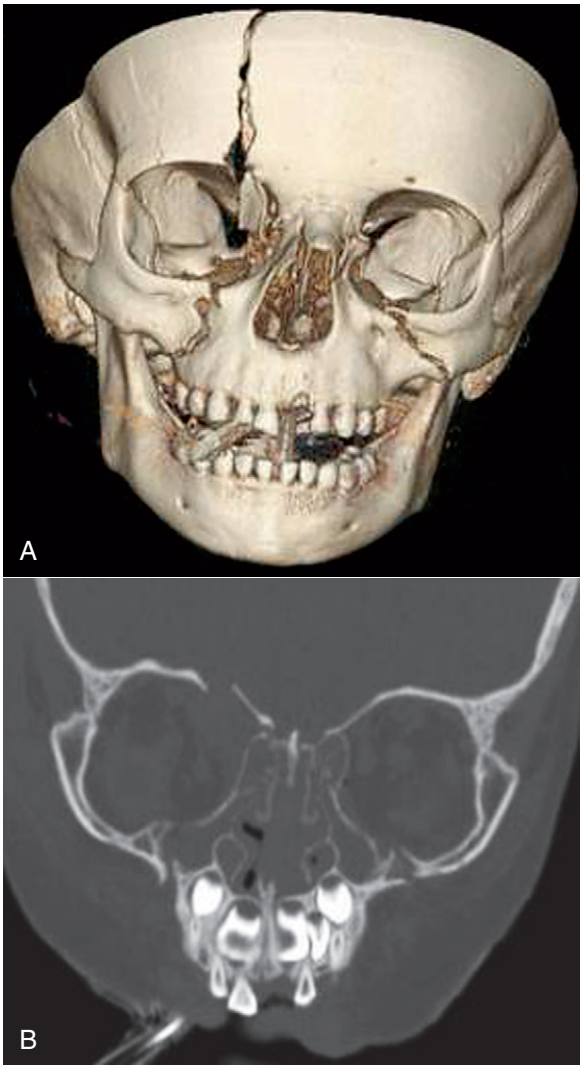


FIGURE 29-1 **A,** Three-dimensional CT scan of 4-year-old boy, status post–MVA, sustaining a bilateral Le Fort 2 fracture and a fracture extending from the right superior orbit to the frontal bone. **B,** Le Fort II fracture in relationship to developing tooth buds. (Courtesy Dr. Edward Kozlovsky.)

often not feasible. Additionally, the presence of unerupted adult dentition or tooth buds will make internal fixation that much more difficult (see Fig. 29-1*B*). An alternative to arch bars is the application of orthodontic brackets to the facial surface of the dentition, which will allow for closed reduction with heavy elastics or thin (26- or 28-gauge) wire; heavier wire will dislodge the brackets from the teeth. Another advantage of this technique is that application of orthodontic brackets is not invasive and can be accomplished in the office setting, provided there is patient compliance. Alternatively, screw fixation of the piriform and zygomatic buttresses bilaterally, as well as placing similar screws in the symphyseal region of the mandible, can be used for intermaxillary fixation and immobilization. Limitations of this technique include the necessity for this to be done under general anesthesia and the risk of damaging underlying tooth buds. However, placing the screws superior to the maxillary fracture and at the inferior border of the mandible minimize this risk. Also, secondary anesthesia for removal of the screws may be required. If these techniques are ineffective, impressions may be taken and models poured and sectioned to facilitate splint construction for closed reduction. Surgical intervention into the tooth-bearing areas of the maxilla for placement of stabilization wires or plates increases the incidence of disruption of developing tooth buds and should be reserved for the most unusual circumstances. Closed reduction is typically performed for 2 to 3 weeks with 26-gauge wire followed by 3 weeks of progressively lighter elastics. Minor occlusal discrepancies may be noted; these can be addressed orthodontically or with occlusal equilibration once healing is completed.

Orbital Fractures

Orbital fractures are not uncommon injuries in the pediatric population, as noted; they can be isolated to the orbit or extend to adjacent facial bones. Before the age of 7 years, most fractures of the orbit in the pediatric patient occur in the orbital roof, with extension to the frontal sinus. This is due to the underdevelopment of the sinuses. After the age of 7 years, injury to the orbital roof, medial and lateral walls, floor, and frontal sinus are more frequent. Because most growth of the orbits is complete after the age of 7, fractures of the orbit in children 7 years or older should be managed like those in the adult, without concern for growth disturbances.[5] Prior to any type of surgical intervention, a complete ophthalmologic examination should precede orbital exploration. CT scanning is again the imaging modality of choice, with coronal, axial, sagittal, and three-dimensional reconstructions. There remains debate about the timing of open reduction; some advocate immediate surgery, whereas others advocate waiting until periorbital edema has resolved. A classification system has been developed based on a 2008 study of 74 pediatric patients with orbital bone fractures[23]:

Type 1: Pure Orbital Fractures

1a—floor fractures
1b—medial wall fractures
1c—roof fractures
1d—lateral wall fractures
1e—combined floor and medial wall fractures

Type 2: Craniofacial Fractures

2a—growing skull fractures

Type 3: Orbital Fractures Associated With Common Fracture Patterns

3a—fractures of floor in inferior orbital rim
3b—zygomatic maxillary complex fractures
3c—naso-orbital-ethmoid (NOE) fractures
3d—other fracture pattern

Findings on physical examination include periorbital edema and ecchymosis, subconjunctival hemorrhage, enophthalmos, diplopia, and infraorbital nerve paresthesia. A forced duction test should be used to evaluate for inferior rectus muscle entrapment. In children, this may require sedation to ensure cooperation. An ophthalmology consultation is indicated to rule out global injury because up to 24% of cases of pediatric orbital trauma have associated injury to the globe.[4,5]

Treatment of pediatric orbital trauma is primarily by conservative management. The most common type of pediatric orbital fracture is the type 1 fracture (pure orbital). Recommendations include conservative management. Surgical intervention is warranted only when there is evidence of entrapment, enophthalmos, or vertical orbital dystopia.[23] Type 2 fractures (orbital fractures occurring in conjunction with the craniofacial skeleton) should also be treated conservatively. Finally, type 3 fractures that occur in conjunction with other facial fractures should be treated surgically as part of the concomitant fracture. Approaches to the orbit are similar to those for the adult and have similar complications, including the subciliary incision and/or transconjunctival incisions. It is recommended that orbital fractures in children with evidence of muscle entrapment be treated sooner to avoid necrosis of the extraocular musculature and associated oculorotary dysfunction.

FRONTAL SINUS FRACTURES AND NASO-ORBITAL-ETHMOID FRACTURES

Frontal sinus fractures in children are rare. Of the paranasal sinuses, the frontal sinuses are the last to develop and do not fully pneumatize until adolescence. Associated intracranial injuries are more common in pediatric patients with frontal sinus fractures compared with adults.[24] A 2005 study of 120 pediatric maxillofacial fractures found 11 with frontal sinus fractures. Of these 11, all suffered concomitant orbital fractures, usually the orbital roof, 7 sustained significant intracranial injury, and 4 had CSF leaks.[25] Management involves identifying and treating any concomitant intracranial injury in the acute setting and preventing long-term complications such as CSF fistula, meningitis, frontal sinusitis, mucocele, and cosmetic deformities. Conservative management is indicated for pediatric frontal sinus fractures with nondisplaced anterior or posterior table fractures, provided there is no CSF leak. Displaced pediatric frontal sinus fractures involving the anterior table require open reduction and internal fixation (ORIF); fixation can use resorbable plates or titanium plates, which may require removal in the future. Posterior table involvement may require cranialization of the frontal sinus. The role of frontal sinus obliteration in the pediatric population is not well described.

CASE 29-1

14-year-old male s/p struck in face with a baseball bat (Fig. 29-2*A*). By report, there was no loss of consciousness. On exam, the patient had significant frontal and periorbital edema. CT scan revealed a left frontal sinus fracture extending to the superior orbital rim as well as a left naso-orbital fracture (see Fig. 29-2*B*). ORIF was performed. Of note, the patient and his mother elected not to have a bicoronal flap performed to access the fracture; rather, they elected to have a hemi–open sky type incision to access the fractures using existing lacerations, which were extended (see Fig. 29-2*C-E*).

NOE fractures are also rare in the pediatric patient. Growth of the middle face is dependent on growth of the anterior cranial fossa (sphenoid), orbit, and nasal septum. When open reduction of the NOE region is undertaken, the periosteum is removed from the nasal and orbital bones, and this alone has the potential to inhibit subsequent growth. Therefore, stripping of the periosteum should be performed with extreme care to prevent growth retardation. Growth in this region is imperative for the formation of the midface and is dictated by the expansion of the cranium to compensate for the brain at the frontoethmoid, frontolacrimal, frontomaxillary, ethmoidal maxillary, and nasomaxillary sutures.[5] Open reduction of the NOE complex should be carried out within 4 days of injury and with minimal disturbance to the nasal septum.

The region can be approached through an existing laceration by extending the inferior lid incision superiorly on the lateral nasal bone, through incisions made over the nasal dorsum or through a coronal incision. Precise reduction of the medial orbital rim, frontal process of the maxilla, and medial canthus is necessary to ensure restoration of aesthetics and lacrimal function. If the nasolacrimal duct is torn, it should be cannulated and splinted for several months. If traumatic telecanthus is present, the medial canthal ligament alone or with the associated fragment of bone from the orbital rim may have detached. The ligament must be reduced and held in place in a superior and posterior position, similar to surgical procedures performed for adults. Children do not tolerate internal or external splinting well; therefore, it is important to reduce and fixate the nasal bridge and medial canthus without the use of splints, if possible.[26]

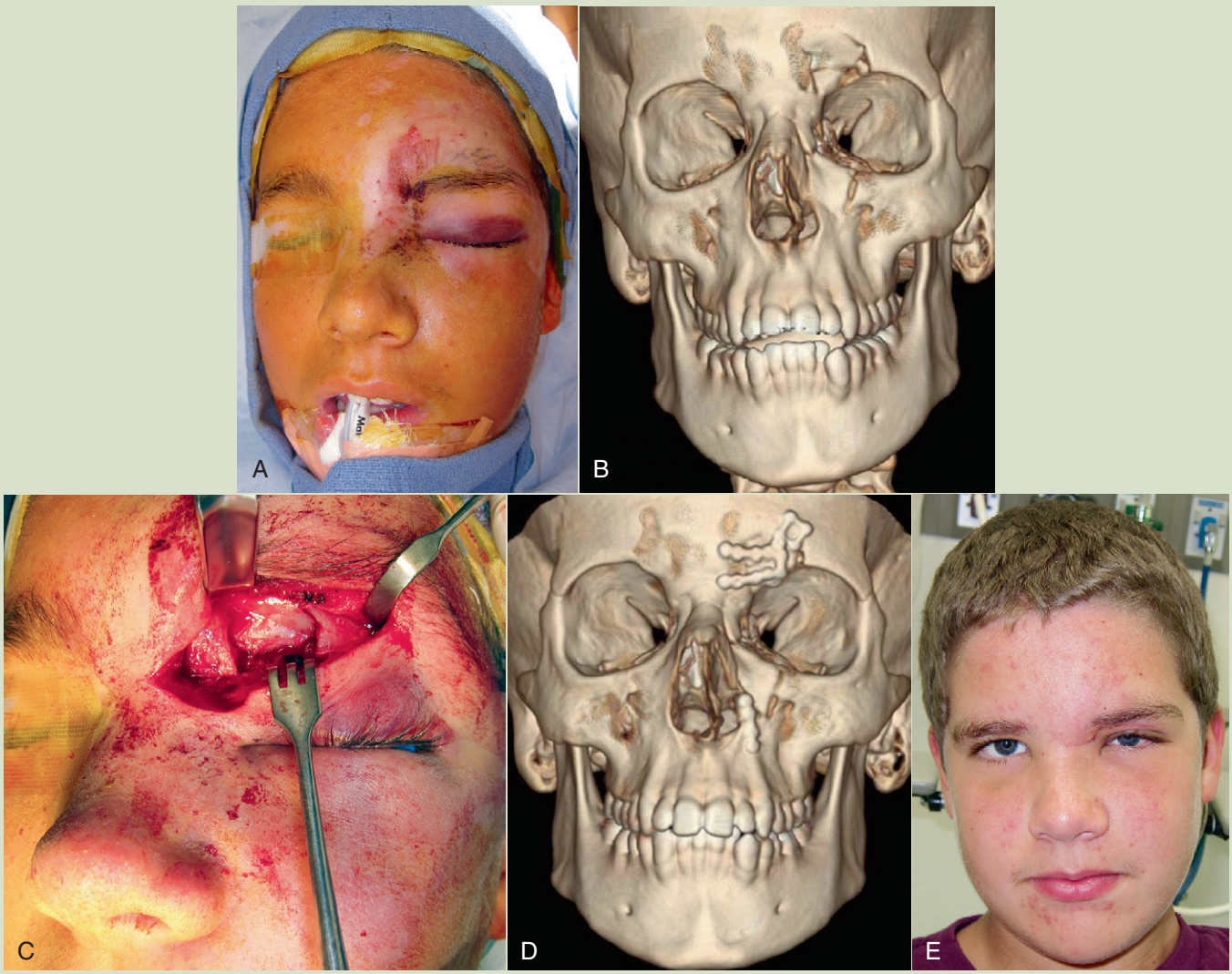


FIGURE 29-2 **A,** 14-year-old boy with a left frontal sinus fracture extending to the left superior orbital rim and left naso-orbital region. **B,** Preoperative three-dimensional Hollender CT scan. **C,** Hemi–open sky approach. **D,** Postoperative three-dimensional CT scan. **E,** 2 weeks postoperatively.

CASE 29-2

An 8-year-old female was impaled with a fence pole during a motor vehicle accident, resulting in avulsion of her left globe and a complex NOE fracture. The patient was taken urgently to the operating room with oculoplastic surgery carried out, in which NOE was plated via the lacerations and globe enucleated. A prosthetic globe spacer was placed at time of surgery. Postoperative picture demonstrates early healing with preliminary prosthetic globe. Patient subsequently required further nasal revision surgery (Fig. 29-3).

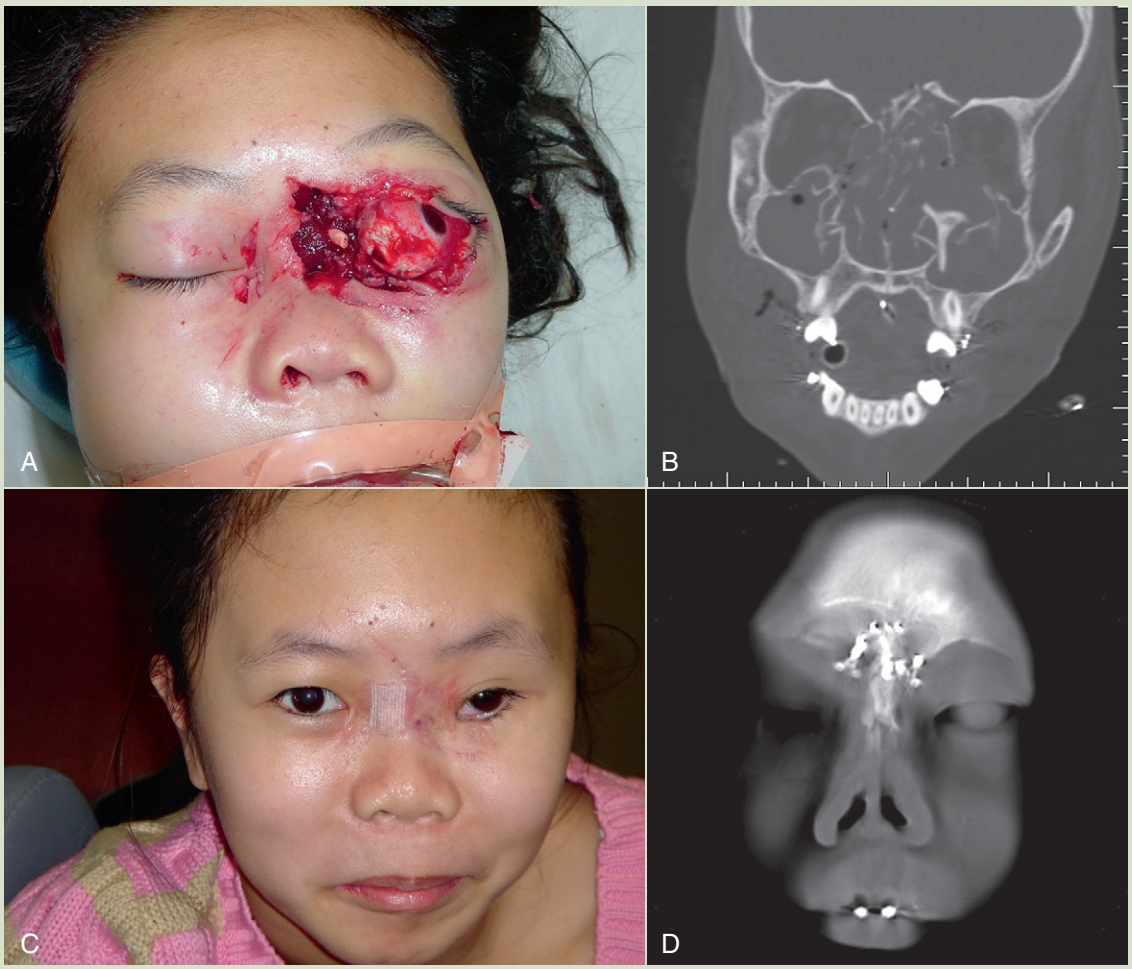


FIGURE 29-3 **A,** 8-year-old girl impaled by a fence pole, resulting in a complex NOE fracture and avulsion of the left globe. **B,** Coronal CT scan view of comminuted NOE fracture. **C,** 6 weeks postsurgery with prosthetic globe in place. **D,** Postoperative CT scan.

FRACTURES OF THE ZYGOMATIC COMPLEX

Zygomatic complex fractures include fractures of the zygoma, zygomatic maxillary complex, zygomatic orbital complex, and zygomatic arch. These are common fractures that represent up to 41% of all pediatric midface fractures.[10] As the patient ages, the zygoma becomes more prominent and fractures to this area increase in incidence. Diagnosis is based on clinical examination coupled with CT evaluation. Physical findings include periorbital edema, possible trismus if the arch is fractured and displaced, infraorbital nerve hypoesthesia, flattening of the malar process, and palpable bony steps. Because there often may be an associated orbital fracture, a thorough ophthalmologic examination is warranted. Reduction of the fractured arch of the zygoma is often the only treatment necessary and, just as in the adult, is performed only if a cosmetic deformity exists or if notable trismus is present. Greenstick or minimally displaced arch fractures in the pediatric patient are treated conservatively by observation, but ORIF may be necessary for significantly displaced or comminuted fractures. Intraoral and extraoral approaches are often indicated to stabilize a true zygomaticomaxillary fracture. Like the adult patient, three-point stabilization is ideal, Our experience has indiacted that often the zygomaticofrontal process, infraorbital rim, and zygomatic buttress are reduced and rigidly fixated. Of note, facial edema often is associated with these fractures, which may require a delay in treatment to allow for the edema to resolve. Additionally, care should be taken when placing the rigid fixation in the zygomatic buttress region to avoid damaging any underlying tooth buds. Isolated zygomatic arch fractures can be treated via an extraoral (Gilles) or intraoral (Keane) approach. In both cases, rigid fixation is not required. The reduced arch must be protected postoperatively from any trauma or pressure during the initial postoperative period to ensure that the reduced arch does not displace. Growth disturbances rarely occur with these types of fractures.[5]

MANDIBULAR FRACTURES

The treatment goals of treating a fractured mandible include restoring occlusion and facial form to preinjury function and appearance. Specific to the pediatric patient is the presence of underlying tooth buds, which may complicate reduction and fixation of mandibular fractures. Similar to pediatric midface trauma, conservative therapy is preferred, with ORIF reserved for select situations.

Fractures of the Mandibular Condyle

Pediatric mandibular fractures represent less than 10% of all mandibular fractures, but up to 50% occur in the condylar region. [27]In younger children (<6 years) these fractures appear in the intracapsular region of the condyle; in older children, they occur in the condylar neck region.[28] The treatment of these fractures is an area of debate in oral and maxillofacial surgery. Treatment options range from observation and soft diet to maxillomandibular fixation (MMF) to ORIF. The two issues unique to pediatric condylar fractures are the risk of temporomandibular joint (TMJ) ankylosis and potential progressive growth disturbances of the face. The intact condylar unit is essential to the integrity of the TMJ apparatus. In addition, the condyle acts as the growth center for the developing lower facial third.[29] Based on the functional matrix theory of Moss—essentially, that form (structure) follows function and condylar cartilage is responsible for the maintenance of a correct condylar head–glenoid fossa relationship during facial and mandibular growth, which in turn supports normal mandibular function and normal mandibular anteroposterior growth. The lack of a condyle interferes with the functional matrix, which leads to an arrest of growth.[30] Disruption of this growth center can result in significant facial asymmetry as an adult, specifically an open bite on the contralateral side resulting from decreased ipsilateral vertical dimension, deviation of the mandible toward the fracture site in opening and closing movements, changes and remodeling of the contralateral joint, ankylosis, and TMJ dysfunction.

Commonly, in children, a fall with a point of impact in the symphyseal region of the anterior mandible can force the growing condyle into the glenoid fossa, resulting in an intracapuslar condylar fracture. As such, the clinician should rule out condylar fractures in children with chin lacerations. On physical examination, there may be some degree of trismus and the mandible deviates to the affected side if dislocation of the proximal segment occurs. Preauricular swelling may be present on the affected side. Malocclusion may also be present; in a unilateral condylar or condylar-ramus fracture, an open bite malocclusion on the unaffected side is often present, a result of shortening of the condylar-ramus complex and premature occlusal contacts on the injured side. Bilateral condylar fractures result in an anterior open bite secondary to bilateral shortening to the condylar-ramus unit.

CASE 29-3

6-year-old male 5 years s/p motor vehicle accident, presents with severe trismus—maximal incisal opening is 5 mm. He also has a significant anterior open bite. Three-dimensional CT scanning demonstrates bilateral TMJ ankylosis. The patient subsequently underwent bilateral gap arthroplasties and bilateral coronoidectomies, followed by aggressive physical therapy (Fig. 29-4).

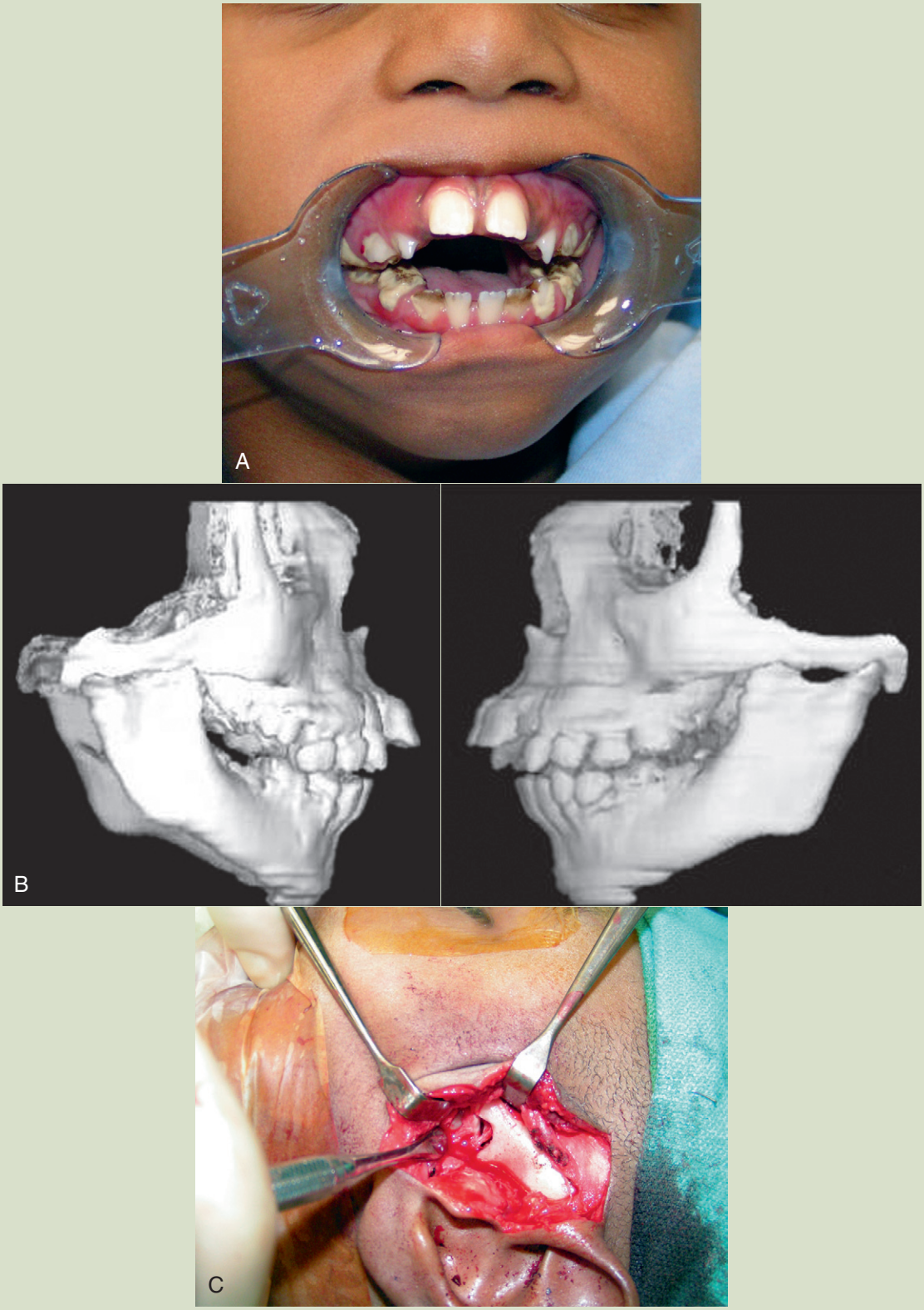


FIGURE 29-4 **A,** 6-year-old boy 4 to 5 years after a MVA, resulting in bilateral condylar fractures. Currently, the patient presents with severe trismus and an anterior open bite. **B,** Preoperative three-dimensional CT scans demonstrating TMJ ankylosis. **C,** Intraoperative view of gap arthroplasty.

In the literature, there is a documented remodeling of the condyle postinjury, especially in the pediatric population. Remodeling, in this case, can be defined as an uprighting of the condylar process and restoration of TMJ joint function. It appears that there are two active mechanisms involved in remodeling—resorption of the fractured, displaced condylar segment plus apposition of bone to create a new condyle.[31] In a series of articles by Lindahl and Hollender, it was noted that young patients (<12 years) have an exceptional ability to remodel postcondylar fracture.[32] In this study, patients from 3 to 11 years demonstrated a complete straightening to a normal relationship between the long axis of the ramus and condylar process. The authors termed this *restitutional remodeling*, unique to children. Teenagers and adults did not share this ability to heal postcondylar fractures. Lindahl went on to state that there exists a genetic guidance for increased cellular activity to rebuild the condylar process in children sustaining condylar fractures. In teenagers and adults, once skeletal maturity has been reached, there is a decreased proliferation of cells in the condylar process—hence, a much-decreased ability to regenerate and remodel.

CASE 29-4

A 27-month-old male was referred from the pediatrician's office for evaluation of the lower jaw 2 days after a fall. Physical exam was significant for a 1-cm contusion of the right symphysis as well as a slight occlusal prematurity on the left. There was a decreased range of motion of the mandible as well as significant tenderness to palpation over the left TMJ joint region. A facial series performed on the day of injury was consistent with a high left condylar fracture, demonstrating head of the condyle displaced anteromedially (Fig. 29-5*A*). Treatment at this point was conservative, limited to a soft nonchewing diet as well as close follow-up and assessment: 1 week follow-up revealed reestablished centric occlusion, improved range of mandibular motion, and decreased tenderness on examination; 1 month follow-up revealed full range of mandibular motion as well as toleration of a regular diet; at 2-year follow-up, the patient was without complaint, demonstrating full range of mandibular motion and a maintained stable class I occlusion. His face was noted to be well formed and symmetrical in all planes. At 2.5 years postinjury, MRI studies were performed, revealing normal-appearing TMJ apparatus and condyles bilaterally (see Fig. 29-5*B*).

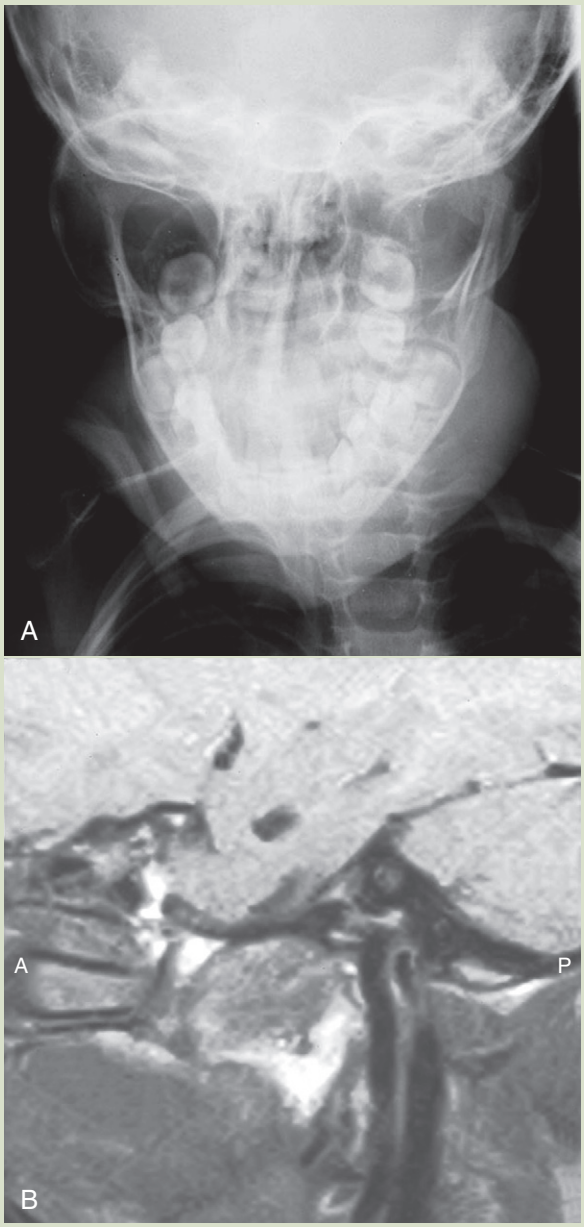


FIGURE 29-5 **A,** 27-month-old child after a fall, hitting the chin. This Towne's view demonstrates a left condylar fracture, with the condylar head displaced medially. **B,** Magnetic resonance imaging (MRI) scan obtained 30 months after injury, demonstrating a new condyle. (Courtesy Dr. Barry Wolinsky.)

Although plain films or panoramic radiographs are good diagnostic tools, CT is necessary to evaluate condylar fractures properly. Studies have shown that CT scans have 90% accuracy in diagnosing pediatric condylar fractures, but panoramic radiographs have only 73% accuracy.[33]

Treatment of Condylar Fractures

In pediatric patients with minimally displaced condylar fractures, conservative management is the treatment of choice. If the occlusion is reproducible, a soft diet for 2 to 3 weeks is indicated. In condylar fractures that are not easily reproducible or mandibular asymmetry accompanies a condylar fracture, MMF may be indicated for 2 weeks followed by physical therapy. Placement of Erich arch bars, used for MMF in adults, is often not feasible in the primary or mixed dentition stages because of primary teeth risk avulsion after application of the arch bar. As such, we have found that using an orthodontic appliance, applied to the facial surface of the primary dentition, is an ideal method to provide for MMF. Longer fixation may result in potential TMJ ankylosis.

Absolute indications for open reduction of pediatric condylar fractures are similar to those for adults and include: displacement of the condyle into the middle cranial fossa, inability to obtain adequate occlusion by closed reduction, lateral extra capsular condylar displacement, and the presence of a foreign body. Relative indications for open reduction of pediatric condylar fractures include: severe seizure disorder, mental retardation, severe upper airway obstruction, and psychologically unable to tolerate MMF.[34]

A critical aspect of managing pediatric patients with condylar fractures is monitoring mandibular and facial growth. One recommended monitoring protocol is clinical examination at 1 and 4 weeks postinjury, followed by clinical and radiographic examination at 2, 6, and 12 months post injury. The child should then be followed annually until cessation of growth and stabilization of the permanent dentition occur.[35] Examination should include obtaining panoramic films, as well as assessing occlusion and TMJ function. If there is any crepitus, decreased range of motion, malocclusion, or facial asymmetry noted, further imaging is indicated as well as functional appliances and orthodontics.

Mandibular Angle, Body, and Symphysis Fractures

Isolated fractures of the mandibular angle, body, and symphysis region in the growing patient are typically treated conservatively via closed reduction, because these fractures are usually easily reduced. In addition, the osteogenic potential in children to heal bone is significantly enhanced compared with the adult. As such, only 2 to 3 weeks of MMF is required. As noted, a simple technique of MMF in the pediatric patient is bonding orthodontic appliances. When MMF is not feasible, an alternate technique is to fabricate a lingual splint from dental models. This requires alginate impressions of the fractured mandible and upper arch; in the uncooperative patient, this may require conscious sedation or general anesthesia. From these models, model surgery is performed to reduce the fracture on the stone cast. From the reduced mandibular cast, a lingual acrylic splint is fabricated with holes interdentally to hold the splint in place. Once the splint is completed, the patient's mandible is manually reduced and the splint is wired into place and held there for 2 to 3 weeks. Generally, MMF is not required in addition to lingual splint application for treating pediatric mandibular symphyseal fractures.

Open reduction of the angle, symphysis, or body of the mandible in the pediatric patient is rarely indicated. In patients with associated condylar fractures, however, internal fixation of the symphysis fracture limits the need for MMF and permits early function of the condyles. Typically, open reduction is limited to patients in whom there is a severely displaced fracture, closed reduction is not feasible, or there is an associated condylar fracture. In these cases, we have used midface (1.5-mm profile) plates to reduce the fracture. It is important to note that the plates should be placed as inferiorly as possible to avoid tooth buds; in addition, these plates will require removal 4 to 6 months after reduction to ensure that there is no restriction in mandibular growth. The intraoral approach for the placement of intraosseous wires or plates is the method of choice because it eliminates visible scars and possible injury to the facial nerve. Consideration could be given to the use of resorbable plates; however, some are large caliber and not suited for younger patients due to size limitations.

ACKNOWLEDGMENT

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CASE 29-5

10-year-old male s/p struck by a car. Sustained a left parasymphyseal fracture and a displaced right angle fracture. Because of his mixed dentition phase and displacement of the right angle fracture, closed reduction was not feasible. As such, the patient underwent ORIF of the fractures, with removal of plates planned 4 months after open reduction (Fig. 29-6).

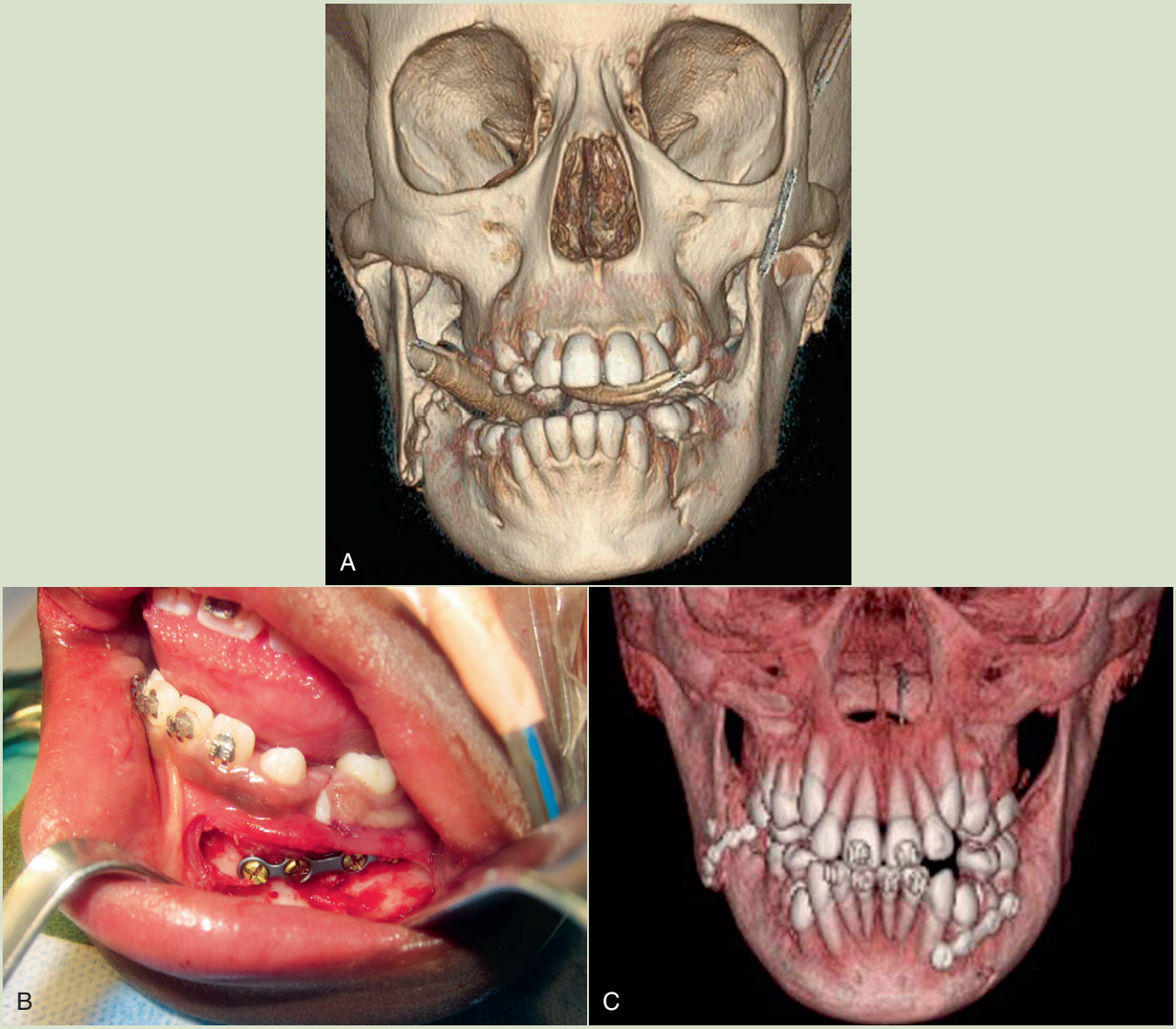


FIGURE 29-6 **A,** Preoperative CT scan demonstrating bilateral mandibular fractures in a 10-year-old child after being struck by a car. **B,** Intraoperative view of ORIF. **C,** Postoperative three-dimensional CT scan.

CASE 29-6

A 12-year-old male, s/p assault sustaining bilateral mandibular body fractures. Because the occlusion was easily reproducible, a decision was made to place the patient into MMF using orthodontic brackets. Fixation was maintained for 2 weeks followed by 2 weeks of elastics and soft diet, with good outcome (Fig. 29-7).

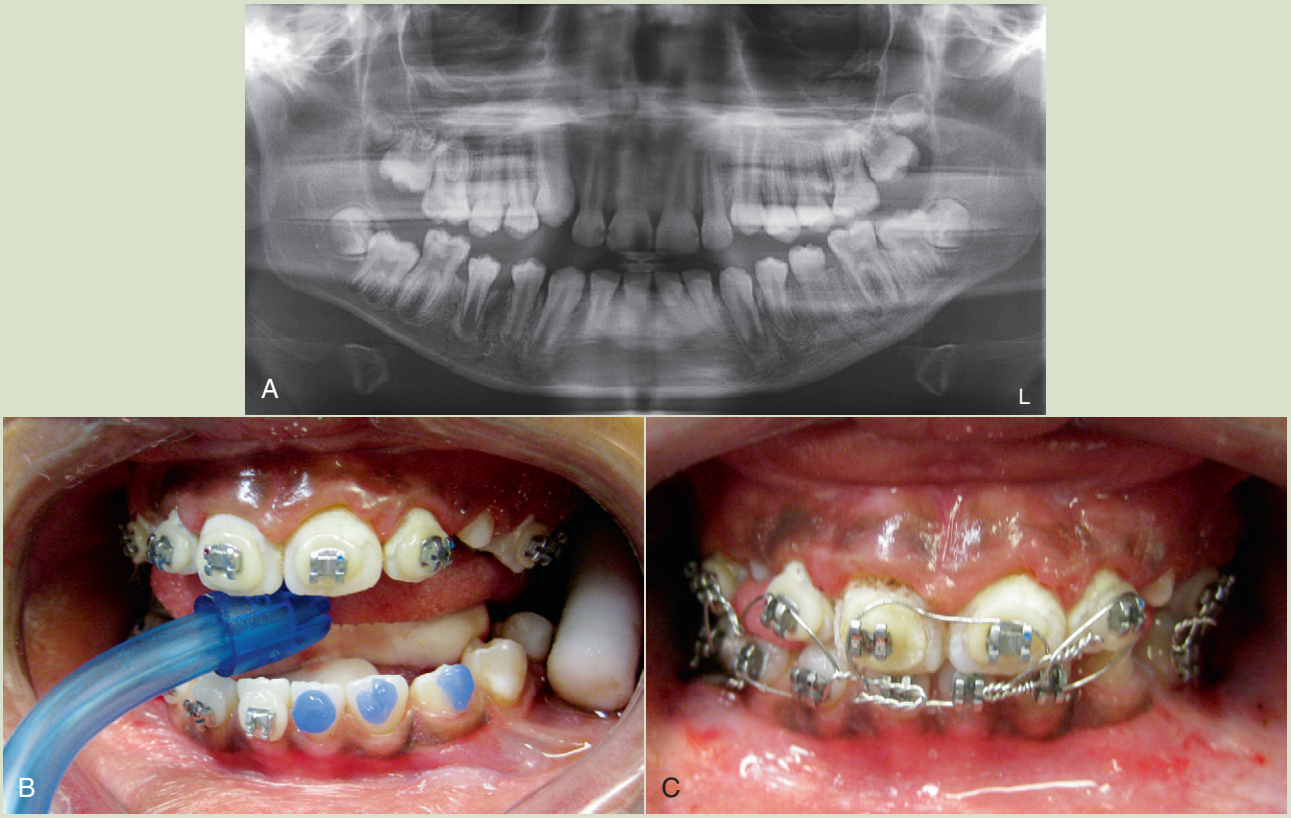


FIGURE 29-7 **A,** Preoperative panoramic radiograph demonstrating minimally displaced mandibular body fractures on a 12-year-old boy. **B,** Application of orthodontic brackets. **C,** Use of orthodontic brackets for MMF.

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CHAPTER

30

Oral and Maxillofacial Trauma in the Geriatric Patient

Brian M. Smith | Dmitry Peysakhov

OUTLINE

The Biology of Aging
System Changes
Cardiovascular System
Respiratory System
Renal System
Nutrition
Psychosocial Issues
Soft Tissue Trauma and Wound Healing
Facial Fracture Management in the Geriatric Patient
Maxillary Fractures
Midface Fractures
Mandibular Fractures
Atrophic Mandibular Fractures
Geriatric Trauma: Outcome and Survivability

The older population in the United States is on the rise. A number of factors have contributed to this trend, including early detection and prevention of life-threatening diseases and a more widespread engagement in exercise and fitness activities among the older. As a result, more than half of the current U.S. population can anticipate living to age 80, with a contemporary life expectancy at age 75 of 11 years and at age 85 of 6 years.[1] From 1990 to 1994, the older population, defined as 65 years and older, increased 11-fold and the segment of the population younger than 65 years only increased threefold. Based on several variables, including fertility rate, mortality rate, immigration activity, and the aging of the baby boomer generation, the U.S. older population is projected to increase by 18% over the next 10 years and by more than 50% within 50 years.[2] It is estimated that by 2050, more than 20% of the population will be 65 years of age or older. The impact of these population trends on the national health care system is significant considering the adjustments that must take place in the delivery of health care and reallocation of resources.

The geriatric population has been an increasing focus of health care providers and therefore is one of the largest consumers of health care in regard to resources and funding. Although older adults make up only 14% of our population, they receive more than 30% of all prescribed medications.[3] Trauma is the seventh leading cause of death in older adults, making up 25% of all trauma admissions. The most common mechanisms of injury among older adults are falls and motor vehicle accidents (MVAs). Most deaths occur in the first 24 hours, usually due to head injury. Late mortality is related to severity of organ failure and existence of a premorbid condition. Approximately one third of long-term survivors require nursing home care. The oral and maxillofacial surgeon, as a specialist involved in the care of trauma patients, will see an increasing number of older patients requiring treatment for trauma-related injuries. Increasing age puts a trauma patient into a higher risk category and older patients sustaining major trauma are known to have higher complication and mortality rates than their younger counterparts.[4] The purpose of this chapter is to review the biologic effects of the aging process on major organ systems and its effects on the psychological status of older adults, and discuss management of traumatic injuries to the maxillofacial region.

THE BIOLOGY OF AGING

Aging is a biologic process that results in a progressive deterioration of structure and function over time that cannot be stopped or reversed. It is a process that is genetically programmed but modified by environmental influences. Kohn[5] has described the aging population as having multiple coexisting diseases that are progressive within a physiologic framework that has a diminished ability to react to stress. This results in increased mortality from injuries and other various insults. Although aging should not be considered a disease, these age-related changes will eventually result in disease and death. Human aging is associated with molecular, cellular, and physiologic changes characterized by a deteriorating homeostatic balance associated with the increasing prevalence of neoplasia and other diseases. At the molecular level, aging has been associated with an increase in DNA point mutations, telomere attrition, and alterations in patterns of methylation.[6-9] Each of these can disrupt the normal expression and/or function of proteins involved in cellular growth, maintenance of genomic integrity, responses to cellular stress, and inflammation.[7,8]

On the cellular level, multiple age-related changes have been observed, including the increase in the number of nucleoli as a result of invagination of nuclear membrane around the clumped nuclear chromatin. Decreased levels of protein production have been attributed to reduction in rough and smooth endoplasmic reticulum. Changes in mitochondrial size and function

have been linked to reduced efficiency of fatty acid usage in the aged cell.[10,11] In 1968, Harman proposed that mitochondria determine life span due to its damage by free radicals.[12] As the cell ages, there is a progressive loss of water from the cytoplasm, causing an increase in the viscosity of the cytoplasm, which reduces the exposure of biologically active cell surfaces.[13] Cell membrane permeability also increases and the sodium-potassium pump becomes less efficient. There is a shift of water out of cells, increasing the extracellular space. The total level of intracellular potassium is also reduced.[14] These alterations in membrane function have to be considered when managing fluid administration and resuscitation in the older patient. All these changes affect the cell's ability to produce energy and maintain vital biochemical functions. In its entirety, these age-related cellular changes are directly responsible for the physiologic aberrations that characterize the older patient.

The age-old question of why we age still has to be answered. Throughout the years, many theories have been proposed to answer this question. The popular programmed theory of aging proposes that aging is similar to other biologic processes controlled by the expression of a specific gene.[15] This theory is supported by observations that every organism has a physiologic life span that is highly characteristic for its species. In this model, so-called senescence genes, when expressed, are responsible for modulating various cellular functions that begin the aging process. The somatic mutation theory focuses on mitotic errors as an important factor in the aging process. It is thought that the gradual accumulation of genetic errors, which has been observed in older cells, results in chromosomal losses that have a negative impact on cellular metabolism. Others have hypothesized that the aging process is directly related to alterations in immune system function characterized by an increase in the levels of circulating autoantibodies.[16] Some have linked aging to the release of a specific hormone that reduces the response of peripheral tissue to the effects of thyroid hormone.[17] Harmon[12] has proposed that free radicals from the diet, environment, or metabolic cellular processes can accumulate in cells and interfere with cellular oxidation. Older cells, therefore, will have a greater exposure to these agents and are more likely to experience these negative effects.

SYSTEM CHANGES

CARDIOVASCULAR SYSTEM

As a person ages, cardiac function is altered in an age-related manner and a risk of cardiovascular disease increases directly proportional to age. These age-related physiologic changes (Table 30-1) do not include common cardiovascular diseases such as coronary artery and cerebrovascular diseases, which increase in frequency with aging. Clinical manifestations and prognosis of these cardiovascular diseases likely become altered in older persons of advanced age because interactions occur between age-associated cardiovascular changes in health and specific pathophysiologic mechanisms that underlie a disease.[18] As a result, these significant changes predispose a person to these diseases and, in many cases, worsen the prognosis, such as a patient with a myocardial infarction (MI). Preexisting age-associated changes in vascular and ventricular properties alter the substrate on which the infarction occurs and probably play a crucial role in the poor prognosis of older patients with acute MI.[19]

TABLE 30-1 Common Age-Related Cardiovascular Changes and Associated Diseases

Age-Related Changes	Associated Cardiovascular Disease
Decreased heart rate response	Sinus pauses
Longer P-R intervals	Second- and third-degree AV block
Right bundle branch block	Left bundle branch block
Increased atrial ectopy	Atrial fibrillation
Increased ventricular ectopy	Sustained ventricular tachycardia
Altered diastolic function	Decreased systolic function (ejection fraction)
Aortic sclerosis	Aortic stenosis, aortic regurgitation
Annular mitral calcification	Mitral regurgitation, stenosis systolic hypertension diastolic hypertension

It is difficult to measure the direct age-related effect on the cardiovascular system because most studies have eliminated asymptomatic individuals with a clinically significant CAD (coronary artery disease). Wenger[20] has found that about 20% of patients older than 80 years of age have clinically manifest CAD, but most older people with significant obstructive CAD are asymptomatic.

When planning appropriate treatment for a trauma patient, it is important to understand the patient's cardiovascular status, based not only on the past medical history but also on age-related changes. Older patients with hypertension are two to three times more likely to have a cardiovascular event than younger patients with similar hypertension.

As the heart and blood vessels age, there is a gradual decline in the elasticity of these structures as a result of a decrease in the content of elastic tissue and increase in the amounts of collagen, calcium, and smooth muscle. The degree of collagen cross-linking increases with age and results in a stiffer, less compliant ventricle. The reduced elastic recoil of the large- and medium-sized vessels, coupled with atherosclerotic changes, culminates in an increase in total peripheral vascular resistance. Fibrosis and calcification of the aortic valve, which adversely affect left ventricular function, are present in 20% of patients older than 65. The combination of these effects increases the preload and afterload of the heart and results in a decrease in cardiac output (1%/year) and stroke volume (0.7%/year) and an increase in systolic and diastolic pressures. The impact of cardiovascular disease on the surgical mortality in older patients is significant. In a large controlled study, the average mortality of 2.4% increased to 6.6% with the presence of

cardiovascular disease.[21,22] The mortality rate increased to 7.2% in patients older than 60 years and 14% in patients older than 70 years.

RESPIRATORY SYSTEM

The respiratory system undergoes anatomic, physiologic, and immunologic changes with age. Due to variability in physiologic respiratory measurements among healthy adults, it is sometimes difficult to differentiate between a diseased state and normal state. Although the overall number of alveoli does not change appreciably with age, structural changes do occur, which affects gas exchange. Alveolar ducts enlarge and septa collapse because of a loss of elastic tissue, resulting in reduced total alveolar surface area.[22] The reduction in elastic lung tissue and increased calcifications at rib articulations results in a stiff, less compliant lung. The effort to expand the lung decreases 30% from 20 to 60 years of age.[23] Diaphragmatic breathing assumes a greater role in ventilation but is less efficient as it flattens with age, decreasing the muscle length and strength. These changes function to reduce the elastic recoil of the lungs and lead to premature airway closure, air trapping, and increased dead space. The functional residual capacity increases approximately 10% by age 60.[24] This will create ventilation and/or perfusion mismatches and shunting, resulting in a lower Pao_2. These changes will decrease an older patient's ability to deliver oxygen to the tissues that are actively involved in respiration. The operative mortality risk associated with symptomatic pulmonary disease is related to the severity of the dysfunction. This can be determined by preoperative quantitative pulmonary function testing. Respiratory system reserve is limited with age and diminished ventilatory response to hypoxia and hypercapnia makes it more vulnerable to ventilatory failure during high-demand states (e.g., heart failure, pneumonia) and possible poor outcomes.[25] There is no evidence that the changes in the respiratory system with aging affect day to day function of older adults, but they may become evident under circumstances when physiologic demand reaches the limits of supply.[26]

RENAL SYSTEM

Age-related renal changes are mainly characterized by decrease in renal mass, renal blood flow, glomerular filtration rate (GFR), tubular secretion and absorption, and creatinine clearance.[27] The decrease in GFR can occur with a normal serum creatinine level and may increase the risk of renal failure and mortality from established renal failure. Older patients with end-stage renal disease (ESRD) undergoing dialysis have a substantial and sustained decline in functional status, and are more likely to die in an acute hospital setting. Along with decreased renal function, a number of significant cellular changes occur as a result of aging, including an increase in some renal membrane transporters and renal membrane protein metabolism; also, Na,K-ATPase protein abundance and activity and renal adaptation to a number of challenges are often diminished.[28] The age-related kidney disease is characterized by phenotypic changes in mesangial cell progenitors and is an entity distinct from all other causes of renal disease.[29] Alternatively, age-related renal changes are accelerated by comorbid conditions such as hypertension, atherosclerosis, and heart failure.[30] Bax et al have assessed the data of 1056 patients to study the effect of atherosclerosis on renal size and function and concluded that atherosclerosis accelerates the decrease of renal size and increase of serum creatinine levels with age.[31]

The extensive pharmacologic treatment commonly seen in older adults, in combination with a decrease in renal clearance, leads to significant pharmacokinetic changes, which ultimately result in reduced receptor sites for drug binding, an increase in volume of distribution of lipid-soluble drugs, and subsequent prolongation of elimination half-life.[32,33] This should be an important consideration when administering anesthetic medications to the geriatric patient or assessing the patient who is on a cardiovascular drug regimen.[34] Failure to excrete anesthetics and analgesics normally by the kidneys can lead to toxic levels of these agents and may lead to complications such as oversedation and apnea. There is often a narrow therapeutic index for opioid analgesics related to hepatic and renal insufficiency and an increased sensitivity to central nervous system (CNS) drugs, and so adverse effects must be carefully monitored.[35] For example, in older patients, a longer duration of action has been observed following morphine administration due to a prolonged elimination of the drug from the plasma.[36] When assessing geriatric patients, dose adjustment should be considered for all medications that are eliminated renally, such as allopurinol, amantadine, most antibiotics, atenolol, carteolol, digoxin, lithium, gabapentin, H2 blockers, procainamide, quinidine, and sotalol.[37]

From a surgical standpoint, older patients with active renal disease, especially dialysis patients, require optimal perioperative management. The perioperative concerns include a high incidence of CAD and myocardial dysfunction and difficulty adjusting fluid and electrolytes in the perioperative period in patients with diminished renal function.[38] Hyperkalemia is the most common complication and may require immediate postoperative dialysis.[39] Other surgical complications include increased perioperative bleeding complications and poor blood pressure control, including hypertension and hypotension. In addition, surgery may need to be deferred to nondialysis days due to heparin coadministration during hemodialysis and the possibility of intraoperative hemorrhage.

NUTRITION

Adequate nutrition is essential for maintaining homeostasis in the normal state and trauma patient, in whom so many physiologic systems can be stressed. This is particularly significant in older patients for whom protein energy and micronutrient deficiencies are common. Energy intake below 30% of estimated needs and a low serum albumin level have been associated with longer hospital stays, higher readmission rates, higher in-hospital mortality rates, and increased mortality at 90 days and 1 year.[40,41] These concerns are compounded by the fact that certain older maxillofacial surgery patients with

TABLE 30-2 Caloric Requirements for the Geriatric Patient

Degree of Stress	Caloric Requirement (kcal/kg/day)
Nonstressful activity	25
Mild stress (mild URI, grade I or II pressure ulcer)	30
Moderate stress (pneumonia, UTI, grade III pressure ulcer)	35
Severe stress (major trauma, sepsis, grade IV pressure ulcer)	40

URI, Upper respiratory tract infection; *UTI,* urinary tract infection.
Adapted from Abbasi A: Nutrition. In Duthie EH, Katz PR, editors: *Practice of geriatrics*, Philadelphia, 1998, WB Saunders.

compromised oral function secondary to postoperative swelling or maxillomandibular fixation (MMF) will not be able to maintain normal oral nutrition. In addition, the stress of trauma and surgery will elicit a series of physiologic and metabolic responses that will markedly alter the nutritional requirements of the trauma patient. The presence of comorbid conditions, such as diabetes, renal disease, and heart failure, will serve to compound this effect. Identifying and correcting nutritional deficiencies preoperatively will have a significant impact on the healing and recovery of the patient. Following a meta-analysis of clinical studies involving hospital patients with malnutrition, Potter et al have reported that patients who began nutritional supplementation on hospital day 3 or sooner have an average length of stay 3 days shorter than those who started later.[42]

The caloric requirement of older adults declines by 22% from the age of 30 to the age of 80 years.[43] This decline is reflective of a decrease in lean body mass and metabolic rate secondary to reduced physical activity. However, in the older trauma patient, caloric needs will be altered according to the clinical setting. An assessment of the caloric requirement must be established before initiating nutritional support (Table 30-2). Although the caloric requirements of older patients are lower than those of younger patients, their protein requirements are the same and may be elevated. The current recommendation for protein intake in older adults is 0.8 g/kg/day. During episodes of physiologic stress (e.g., trauma, surgery) the protein requirement may be as high as 1.0 to 1.5 g/kg body weight.[43] Following the stress of surgery or trauma, glycogen stores are depleted within 36 hours. When glycogen stores are depleted, energy demands are met through the process of gluconeogenesis, in which protein from skeletal muscle is converted to glucose. This increases the demand for protein intake. Surgical patients with a low protein intake have a fourfold increase in medical complications and a sixfold increase in mortality, and are more likely to have an extended hospital stay.[44,45] In older adults, inadequate intake of protein has been reported in 10% of men and 20% of women.[46] Protein-energy malnutrition (PEM), defined as a deficit in energy (in kilocalories [kcal]) and protein, is also common in older patients. In older surgical patients, the incidence of PEM was reported to range from 43% to 50%[47,48] and was higher for patients in extended-care facilities.

Given the prevalence of malnutrition in the older population, a complete preoperative nutritional assessment by a clinical nutritionist is often indicated. Deficits and requirements are established based on an evaluation of anthropometric, biochemical, clinical, and dietary data. Anthropometric measurements are important for determining the weight status of patients. The body mass index (BMI) is a relative measure of weight to height and is used to distinguish patients who are underweight (BMI < 22) from those who are overweight (BMI > 27). Several biochemical markers can be used to assess nutritional status. Levels of albumin, thyroxine-binding albumin, transferrin, insulin growth factor-1, and micronutrients such as vitamin B_{12}, iron, folate, and zinc are evaluated. Deficiencies of any of these elements that are considered clinically significant are indicative of malnutrition and should be corrected. Clinical signs and symptoms of malnutrition are assessed by a thorough review of systems. Evidence of muscle wasting, poor skin turgor, altered mobility, and altered gastrointestinal (GI) function is suggestive of malnutrition. The dietary component of the nutritional assessment involves an evaluation of typical dietary habits and type of food intake. For the older patient, this will often provide an insight into the preoperative nutritional status.

When the nutritional assessment is complete and the specific requirements are calculated, nutritional replacement can commence. Older patients who have sustained facial trauma or have their jaws immobilized for fracture treatment may not be able to sustain their nutritional requirements through a transoral route. In that situation, other routes of nutritional replacement must be considered. Enteral feeding with a feeding tube is the most effective physiologic approach. Nasoenteric feeding tubes are easily placed at the bedside and can be used in patients who require enteral support for less than 30 days. Enteral feeding through a nasogastric suction tube should be avoided because these tubes are not well tolerated by patients and can cause mucosal irritation and ulceration. Gastrostomy tubes are indicated for patients who require prolonged enteral nutritional support. The tubes are inserted percutaneously through the anterior stomach wall under endoscopic guidance. Regardless of the type of feeding tube used, the proper position of the tube in the GI tract must be confirmed radiographically before initiating tube feedings. Nutritionists determine the content, character, concentration, and rate of delivery of the nutritional supplement. Nutritional support should also be part of the discharge planning process in older patients with compromised maxillomandibular function.

PSYCHOSOCIAL ISSUES

The older patient will often exhibit cognitive, emotional, and behavioral changes that may significantly alter the surgical outcome and recovery. These changes are usually secondary to the normal aging process. Dementia caused by Alzheimer's disease is the leading cause of disability in the older population[49] and represents the most

common reason for institutionalization.[50] Such patients demonstrate a compromised ability to recall, comprehend, and conceptualize issues of daily life. These limitations are magnified in the older trauma patient who has additional physiologic reasons to be confused and disoriented. The most common cognitive difficulties displayed by older patients are reduced attention span, poor short-term memory, and difficulty processing complex information.[51] This will result in poor compliance with preoperative and postoperative management strategies, including compliance with prescribed medications and obtaining an informed consent for treatment. In addition, age-related cognitive and behavioral changes increase the incidence of depression and anxiety and may directly contribute to the cause of the traumatic injury.

Postoperative mental status changes have been associated with a significant negative effect on outcome. Marcantonio et al[51] have reported a sevenfold increase in mortality, a sevenfold increase in major complications, and a notable increase in the length of hospital stay. Data on surgical outcome from a multicenter Veterans Administration hospital study[52] have demonstrated an association between postoperative delirium and complications such as postoperative pneumonia, unplanned intubation, and failure to wean from the ventilator. More significantly, the death rate in this study was eight times higher in patients with postoperative delirium. Identifying older patients who are at increased risk of having postoperative changes in cognitive function is therefore important. Various preoperative, intraoperative, and postoperative factors have been studied in an effort to identify variables related to postoperative delirium. Preexisting cognitive impairment, poor functional status, and polypharmacy are factors commonly associated with postoperative mental status changes. In a large prospective study of noncardiac surgery patients, age older than 70 years, alcohol abuse, poor preoperative cognitive function, poor functional status, and abnormal serum electrolyte levels were found to be independent correlates for postoperative delirium.[51] Operative blood losses requiring a transfusion and a postoperative hematocrit less than 30% have also been associated with a higher likelihood of developing postoperative delirium.[53]

Completing an objective assessment of the patient's cognitive abilities via direct interaction with the patient or family members will enable the clinician to identify psychosocial issues that might compromise surgical care and modify treatment accordingly. In patients with severe cognitive impairments, proper discharge planning with the assistance of a social worker and caretakers is essential for ensuring that appropriate postoperative care will occur.

SOFT TISSUE TRAUMA AND WOUND HEALING

Soft tissue injuries in the geriatric population present a unique treatment challenge due to multiple intrinsic structural changes in the skin and underlying tissues and age-associated comorbidities that affect healing. As the aging population grows, it is not uncommon for them to be more physically active and successfully employed well into their 60s, 70s and even 80s. This in turn reflects in increase in the number of traumatic injuries (Fig. 30-1). In 2009, there were an estimated 210,830 nonfatal work injuries and illnesses among workers 55 years and older, with 94% being the result of trauma. Regardless of the patient's age, the importance of proper tissue management with careful attention to detail cannot be overemphasized because the face is considered the most conspicuous and significant portion of a person's identity.[54]

Wound healing is a complex and dynamic process of restoring cellular structures and tissue layers. Wounds

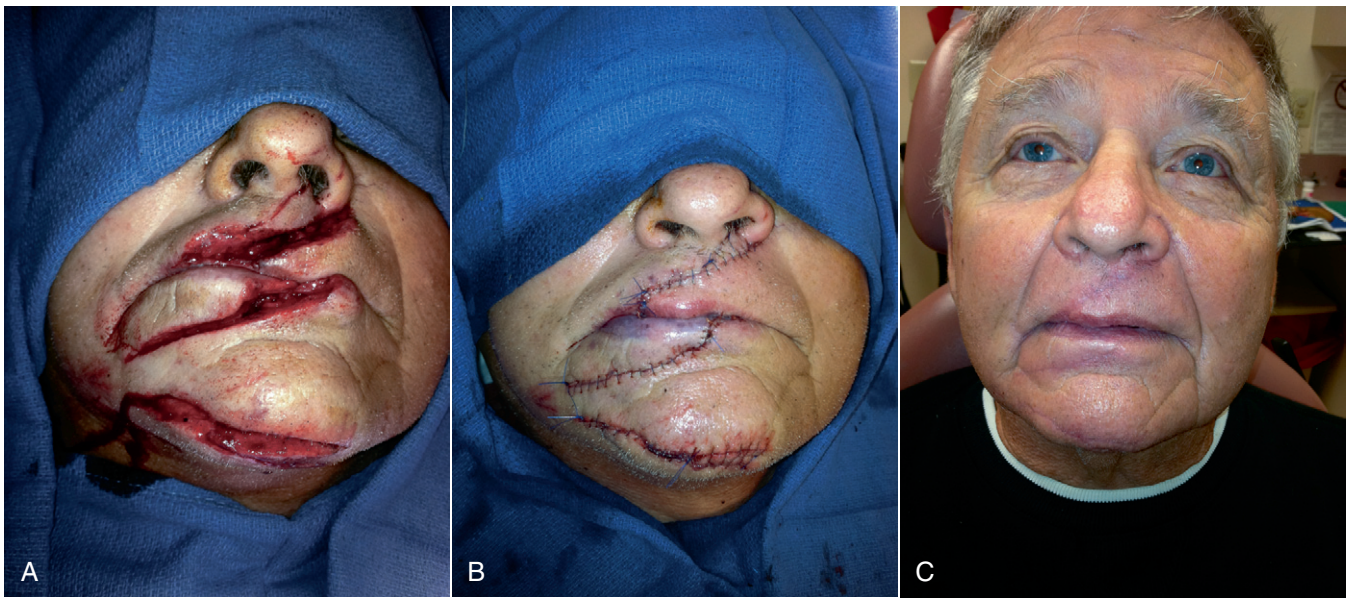


FIGURE 30-1 This 71-year-old man suffered multiple facial lacerations as a result of electric grinder injury at work **(A),** immediately following repair **(B),** and 5 weeks after injury **(C).**

heal significantly more slowly in older adults compared with younger adults regardless of gender and independent of demographic factors such as ethnicity, alcohol or nicotine use, or BMI.[55] In addition, older adults are more prone to injury and have a decreased healing ability due to multiple changes that occur in the skin and underlying tissues. Aging affects all stages of wound healing. A decreased rate of cell proliferation is a result of reduction in collagen synthesis and slower epithelization, mainly due to a decline in the number of mast cells and compromised macrophage function, which leads to a decreased inflammatory response and a limited mitogenic response of keratinocytes and fibroblasts.[54-60] These cellular changes decrease wound tensile strength, collagen deposition, and wound contraction.[61]

In addition to these alterations, concomitant medical illness, pharmaceutical intake, and dietary changes further decrease the rate of wound healing and increase rates of wound dehiscence, ecchymosis, tape strip injuries, infection, and persistent contact dermatitis.[62,63] The pH of the skin surface increases with age, increasing its susceptibility to infection. Neurosensory perception of superficial pain is diminished in intensity and speed of perception, thus increasing the risk of thermal injury; deep tissue pain, however, may be enhanced. A decline in lipid content as the skin ages inhibits the permeability of nonlipophilic compounds, reducing the efficacy of some topical medications. Allergic and irritant reactions are blunted, as is the inflammatory response, compromising the ability of aged skin to affect wound repair. These functional impairments, although a predictable consequence of intrinsic structural changes, have the potential to cause significant morbidity in the older patient and may also be greatly exacerbated by extrinsic factors, such as photodamage.

FACIAL FRACTURE MANAGEMENT IN THE GERIATRIC PATIENT

In the geriatric patient, treatment planning decisions for facial fractures are frequently influenced by risks of prolonged or invasive surgery, the impact on function from intermaxillary fixation or recuperation from open surgery, and even the psychological implications. Preexisting medical conditions are frequently encountered, along with diminished cardiac, pulmonary, and wound-healing abilities that occur with aging. Techniques such as closed reduction with intermaxillary fixation may challenge the patient's respiratory function or the ability to obtain adequate nutrition. Fractures that are not optimally reduced and would normally heal uneventfully in a younger patient run a greater risk of fibrous union, nonunion, or prolonged healing time in the older patient. Open reductions with greater surgical insult and longer time in anesthesia are a greater challenge to the patient's cardiac, pulmonary, and wound-healing abilities.

Management of fractures can also be more challenging because of partial or complete edentulism. The patient's use of partial or full dentures and their fit and condition affect treatment options. Knowledge about the fit and relining of dentures, taking impressions, and vertical dimension may be necessary to use some reduction techniques successfully.

Bony changes in the geriatric population also affect treatment choices. The bone in the maxilla is often thin and comminution at the fracture areas is more common. Suspension techniques that depend on stable bone in the fracture area may not be as useful in older adults. Alveolar bone atrophy is common in areas of tooth loss. In severe cases, there is atrophy of the basal bone as well. Among the most challenging fractures to manage are bilateral body fractures in the severely atrophic mandible.

Historically, many maxillary and mandibular fractures have been managed with closed reduction techniques. These techniques often did not fully reduce fractures and the immobilization of the fracture was not complete. The past decade has seen a shift to open reduction, with good reduction of the fractures and rigid fixation of the fractures in the general population and in older adults. Often, however, weighing the risks and benefits in the geriatric patient is more complex.

MAXILLARY FRACTURES

Although the treatment techniques are not unique to the geriatric population, patients with partial or full dentures are more common in this population. The patient with an edentulous maxilla and maxillary alveolar atrophy often has treatment compromised by thin bone and diminished bony volume in areas that might normally be used to secure a denture or splint. The most common maxillary fracture involves the junction of the horizontal plate of the palatine bone and posterior part of the maxilla.[64] The nature of the fractures also presents the risk of collapse of vertical or anteroposterior positioning as suspension wires are tightened. Poor bone quality and the high likelihood of fracture comminution also increase the complexity of using rigid fixation in this patient population.

For patients with existing suitable dentures, the patient's denture is altered to be useful in positioning and immobilizing the maxilla.[65-69] Holes are drilled in the easily repaired pink acrylic area of the denture to aid in securing arch bars to the denture, making stabilization to the underlying bone easier. If the denture is not available or not usable, a Gunning's splint can be fabricated[70-72] (Fig. 30-2). Impressions of the maxillary and mandibular arches are taken to make study models. These models may have to be cut and repositioned to simulate the planned fracture reduction. They are then mounted on an articulator to ensure the correct jaw relationship. Capturing the correct jaw relationship or creating the correct jaw relationship on the articulator can be a challenge when the fracture(s) or injuries make testing the splint impossible preoperatively.

The denture or Gunning's splint can be secured to the maxilla in several ways. The amount of alveolar atrophy, location of the fractures, and thickness of the bone influence the decision about which method to choose. Transalveolar wiring, transalveolar pin placement or suspension wires from the malar buttress, piriform rims, nasal spine, or zygomas are all commonly used and are

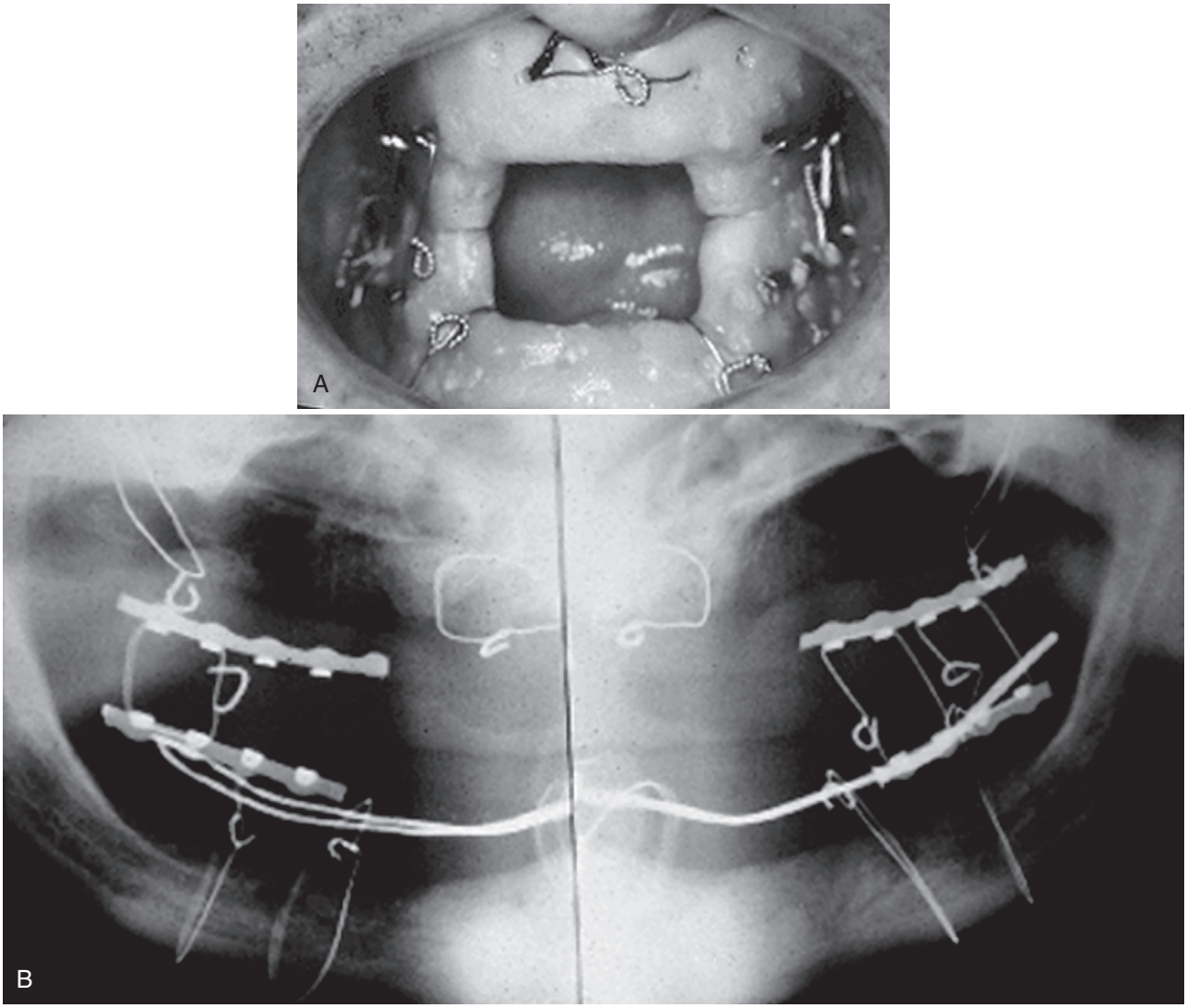


FIGURE 30-2 **A,** A Gunning splint is used to treat this edentulous patient with a mandible fracture. Impressions of the patient's maxillary and mandibular ridges were taken. A bite registration was used to mount the study models on an articulator before fabrication of the splint components. **B,** The Gunning splint is secured to the maxilla via suspension wires in the buttress and piriform areas. It is secured to the mandible with circummandibular wires. In this case, arch bars have been directly luted into the acrylic during the splint fabrication. The arch bars are used to secure the maxillary and mandibular splints together.

suitable methods. Circummandibular wires are the method used for securing a mandibular prosthesis or full mandibular dentures. Partial dentures may be secured by ligation to the remaining teeth with the aid of an arch bar that is also secured to the partial dentures.

Circumpalatal wiring has also been used to secure the prosthesis to the nonfractured maxilla.[73-75] These wires are passed from the anterior maxilla through the nose to the junction of the hard and soft palate. They are brought into the mouth again at the junction of the hard and soft palates by perforating the tissue in this region with the wire-passing awl. They are then brought anteriorly under the prosthesis and secured to the free ends in the anterior vestibule.

The use of craniomaxillary suspension in maxillary fractures should be used with some caution in the geriatric patient. Complications have been reported with craniomaxillary suspension when compared with MMF alone.[76] The principal complication was palatal tipping, presumably from unequal tightening of the suspension wires in unstable fracture areas.

MIDFACE FRACTURES

The comprehensive treatment of midface fractures is covered in the earlier chapters in this text. As with any surgical treatment concerning the aging patient, an assessment has to be made regarding the surgical and anesthetic risk factors versus the necessity and benefits of open or closed treatment. Midface fractures in the geriatric population that do not involve occlusion are managed similarly to those in the general population. However, due to age-related soft and hard tissue changes, some surgical approaches are more suited for a geriatric patient. The aging face has more rhytids, which can be an advantage for some approaches. The infraorbital approach provides direct and easy access to the



FIGURE 30-3 **A,** 83-year-old woman with a left orbital blowout fracture after a fall. **B,** Transconjuctival incision with lateral canthotomy allows good visualization of the defect. **C,** Reconstruction of the orbital floor using a Silastic-impregnated titanium plate.

infraorbital rim and orbital floor. This approach is less popular in younger people because it is unaesthetic, but is usually satisfactory aesthetically in the geriatric population. In contrast, aging causes laxity and downward shift of eyelid tissues and atrophy of the orbital fat. These changes contribute to the cause of several eyelid disorders such as ectropion, entropion, dermatochalasis, and ptosis. The higher eyelid skin crease and ptosis may be due to age-related disinsertion of the levator muscle aponeurosis and involutional atrophy of the orbital fat. The horizontal eyelid fissure shortens by about 10% with aging.[77] Thus, supraorbital approaches including midlid crease and subciliary incisions should be used with caution due to their inherently higher risk of ectropion.[78,79] A transconjuctival approach, with and without lateral canthal extension, may be successfully used for reconstruction of both zygomaticomaxillary complex and isolated orbital fractures (Fig. 30-3). It results in minimal postoperative complications, is cosmetically acceptable, and has a minimal incidence of ectropion compared with cutaneous approaches.[80-82]

MANDIBULAR FRACTURES

As in the maxilla, the treatment techniques for mandibular fractures are not unique to the geriatric population. Weighing the risks and benefits of closed versus open reduction has traditionally resulted in a preference for closed reduction in this population. However, like the general trends in fracture management, open reductions have become more common over the last decade.[83-85]

Closed reductions are most frequently accomplished with intermaxillary fixation using the teeth. Missing teeth or the presence of crown and bridgework may complicate this standard technique. Partial or full dentures or extensive fixed bridgework are more common in this population. For patients with dentures, the dentures may be altered to aid in immobilization of the fracture. The mandibular full denture is stabilized to the mandible with circummandibular wires. These wires are placed using an awl or Keith needle. Care is taken to avoid the mental nerve and facial artery and vein on the inferior and lateral surface. Placement close to the lingual surface avoids the submandibular duct and lingual nerve. Partial dentures may also be secured in this manner with arch bars attached to the remaining teeth and secured to the partial denture.

Another method of obtaining immobilization of the jaw is with intermaxillary fixation screws.[86,87] These are self-tapping bicortical screws that are placed in the alveolus. The screw is placed between the roots of the teeth, if teeth are present. Placing the screw deep in the vestibule and below the tooth roots will often result in local mucosal ulceration that may be difficult for the patient to tolerate. One screw is placed in each quadrant. The intermaxillary fixation wire is passed through holes in the end of the screw or wrapped around the screw end. This method has the advantage of being a rapid method for the application of intermaxillary fixation. Placement can be performed with local anesthetic. It is also effective when crown and bridgework make the application of traditional arch bars difficult. These screws must be positioned properly because damage to tooth roots and the mental nerve has been reported.[88]

Mandibular fractures have also been managed with a closed technique that uses external fixation[89-91] (Fig. 30-4). The early instrumentation in the Joe Hall Morris and biphasic reduction kits used special screws that were placed into the mandible, usually two on either side of the fracture through stab incisions and holes drilled in the mandible. Steinmann pins or Kirschner wires can also be used as external pins and do not have to have a hole drilled before insertion. Once external pins are in position, the fracture segments are manipulated to achieve reduction and then the pins are locked in this position by application of an acrylic mix placed over the ends of the pins that are protruding out of the skin. The acrylic is allowed to harden while the mandible is held in the reduced position. The biphasic kit has armamentarium that allows three-dimensional adjustment of the segments as an intermediate step before application of the acrylic bar. For difficult or unstable fractures, obtaining or maintaining an excellent reduction with this technique can be challenging. This technique can be used in combination with an open technique, with or without internal fixation, to aid in obtaining a good alignment of the fracture segments.[92]

Open reduction techniques have a prominent role in geriatric mandible fracture management. The advantages of direct visualization and achieving excellent reduction and immobilization can be significant. This can be particularly important with unstable fractures or fractures in areas with diminished healing capacity.



FIGURE 30-4 Demonstration of the application of a biphasic external pin appliance in the management of fractures of edentulous mandibles. **A,** At least two pins are placed on either side of the fracture site. Note that the pins are not parallel but diverge slightly from one another. **B,** Following reduction, first-phase stabilization is obtained with the application of a connecting bar and universal connectors. **C,** Second-phase stabilization proceeds with cold-cured acrylic applied while the connector bar is in place. Once the acrylic has cured, the universal connectors and bar are removed, as seen here. **D,** Clinical application.

Fractures of the atrophic edentulous mandibular body in older adults represent one of the more challenging injuries to manage successfully.

ATROPHIC MANDIBULAR FRACTURES

The incidence of atrophic edentulous mandibular fractures, although rare in the past, is continuing to rise due to the improved quality of life for many older adults, correlating with more physical mobility and engagement in more leisure activities.[93] Although atrophic mandibles present a surgical as well as a restorative challenge, mandibles that have 5 mm or less of bone height at their most resorbed point (Cawood type VI) are at high risk for a spontaneous fracture. As a person ages, with progressive loss of teeth, atrophy of the alveolar bony apparatus ensues, resulting in contributing to this fracture potential.[94] This change in bony volume results in a decreased vascular supply from periosteum and endosteum and a decreased potential for osteogenesis, which can compromise bony healing in this patient population. The inherent instability of edentulous, atrophic, mandibular body fractures makes maintaining an adequate reduction and immobilization without direction stabilization unpredictable. The challenge is made greater by the position of the inferior alveolar nerve, which can make internal fixation very difficult.

Throughout history, various treatment modalities have been used to treat the fractures of edentulous and atrophic mandible. Although the earliest description of MMF was by Hippocrates in 460 BC, it was not until 1894 that Gunning splints were first introduced, and are still used today (see Fig. 30-2).[87-99] Open reduction and internal fixation (ORIF) with a silver wire was first performed in 1869, but was largely abandoned for almost a century in favor of other procedures, such as external pin fixation and biphasic pin placement, which came into use in the late 1930s and 1940s. Although both techniques have been successfully used, they were occasionally plagued by poor fracture reduction due to relatively small surface area at the fracture site. To achieve better reduction, open techniques have been used in conjunction with external pin fixation and biphasic pin placement. Placement of transosseous wires and eventually the use of small bone plates have enhanced the stability of the reduction. As the use of plating systems advanced, the reliance on the use of open reductions with the application of rigid internal plate fixation increased. By the early 1970s, Bradley demonstrated that the body region of the edentulous atrophic mandible was primarily dependent on a periosteal vascular supply.[95] This led to a cautious approach, which resulted in minimizing periosteal stripping in the management of these fractures.

The current treatment of edentulous atrophic mandibles is generally divided into closed reduction and stabilization with gunning splints, closed reduction with external fixation, and ORIF (Fig. 30-5). The treatment

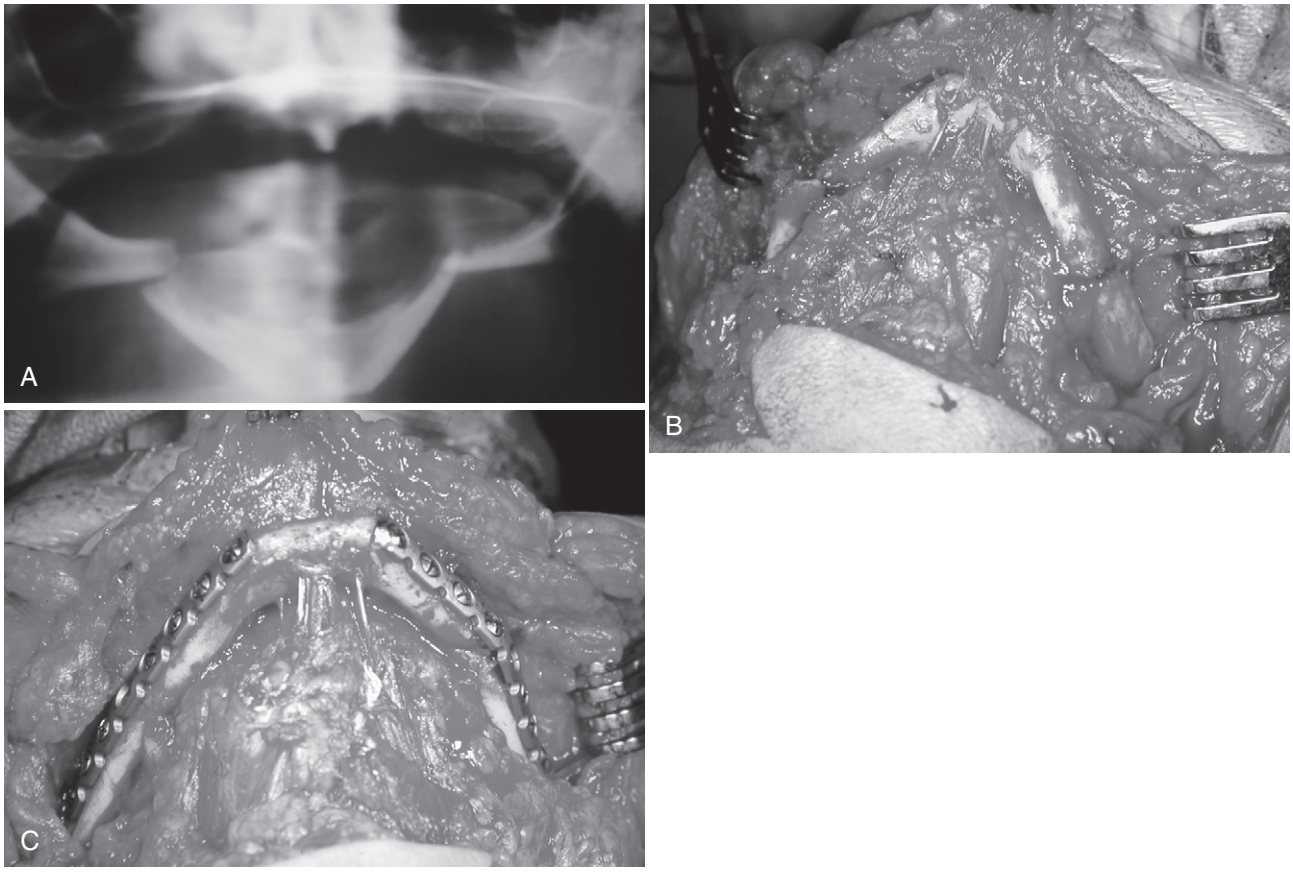


FIGURE 30-5 **A,** Bilateral fracture in an atrophic edentulous mandible. **B,** Wide access is obtained in preparation for placing large plates. **C,** 2.4-mm plates are used to provide prolonged stability from the plating system.

of atrophic mandible fractures should be based on the degree of atrophy. The edentulous mandible with an adequate ridge is usually amenable to closed reduction with Gunning splints. More rigid fixation may be necessary in mandibles with less than 15 mm in bone height.[96] To minimize subperiosteal stripping, many surgeons prefer to treat these fractures with closed reduction techniques or open techniques that involve less periosteal elevation, such as wiring or miniplate fixation.[95-97] Sugiura et al[99] have reported no noticeable differences in compressive stress levels in the bone around screws in the single- or double-miniplate models with bone contact.

On the other end of the spectrum, some surgeons have advocated bone grafting at the time of fracture management as a means of increasing the likelihood of bony union. Although these techniques have often been successful, poor reductions, fibrous unions, and nonunions were not uncommon with this method.

Techniques directed at attaining excellent reduction and providing longer periods of fracture stability through a more rigid internal plating system have gained in popularity, in spite of the need for significantly more periosteal stripping.[100-105] The use of larger plates, 2.4-mm mandibular fracture or reconstruction plates, and bicortical screws applied to an atrophic mandibular body fracture has gained acceptance as a technique that can best prevent the common problem of nonunion and fibrous union.[106] Ellis and Price[107] have evaluated 32 patients with unilateral or bilateral atrophic mandibular fractures who were treated through an extraoral approach with rigid internal fixation. They reported complete healing in all patients with no incidence of any complications, and concluded that the use of a seemingly more aggressive approach for managing fractures through the atrophic mandible, in the end, may be more conservative than a less aggressive approach.

In addition to spontaneous fracture potential, severely atrophic mandibles, in which the maximum bone height is 5 mm or less, may be associated with neurosensory loss and fistula formation. In addition, poor retention of the dental prosthesis may seriously compromise the nutritional status of an already medically compromised individual. Various attempts have been described for reconstruction of severely atrophic mandibles. A highly successful technique to augment a severely atrophic mandible involves the use of a freeze-dried cadaveric mandible packed with autogenous cancellous iliac bone (Fig. 30-6). Recently, there has been success in reconstruction with implants and simultaneous bone graft placement. Lopes et al[108] have reported successful treatment of a severely atrophic mandible with four short dental implants at the symphysis with an immediate bone graft and a 2.0-mm large-profile bone plate for reinforcement. Marx et al[109] have presented a long-term follow-up of 64 patients with initial mandible height ranging from 3 to 6 mm who were treated with dental implants as tent poles to consolidate and maintain the volume of simultaneously placed bone graft. At 3-year follow-up, they reported a mean bone height gain of 10.2 mm, no permanent neurosensory disturbance, and successful osseointegration of 354 of 356 dental implants placed (99.5%).

GERIATRIC TRAUMA: OUTCOME AND SURVIVABILITY

Advanced age has long been considered a risk factor for poor outcomes following major trauma. Compared with younger patients, older trauma patients have higher mortality and morbidity rates. This has been attributed to various factors (see earlier), which include the presence of preexisting diseases and a poor level of physiologic reserve. Identifying older trauma patients who will benefit from expensive and aggressive resuscitative techniques is important in addressing the ethical question of who should and should not receive care. In an effort to establish an evidence-based guideline in the decision algorithm of trauma care for older patients, Jacobs et al reviewed the existing literature for an answer to various care-related problems.[110] There were several limitations to this approach, including the paucity of prospective randomized studies, lack of consensus regarding how to define the geriatric trauma patient, and the fact that most of the studies reviewed were at least 10 years old and did not reflect current trends and improvement in trauma care. Despite these limitations, certain recommendations about the triage and resuscitation of older trauma patients developed from this review, including the following:

1. Advanced age should lower the threshold for triage directly to a trauma center.
2. Age alone is not a predictor of poor outcome and should not be the determining factor in limiting care.
3. The presence of preexisting medical conditions adversely affects the outcome in older trauma patients.
4. Older trauma patients (≥65 years) with a Glasgow Coma Scale score less than 8 have a very poor prognosis. If improvement is not possible within 72 hours, limitation of continued aggressive therapy should be considered.
5. Low survival rates and longer lengths of stay are associated with postinjury complications.
6. Given that 85% of older trauma patients will return to independent function, resuscitation should be pursued aggressively on patients who are not moribund on arrival.
7. Older patients with an admission base deficit less than −6 have a high mortality rate (66%) and may benefit from inpatient acute care nursing.
8. Patients 65 years of age or older with a trauma score less than 7 or a respiratory rate less than 10 have a 100% mortality rate; in these patients, limitation of continued aggressive therapy should be considered.
9. Despite meeting the criteria for triage to a trauma center, trauma patients who are 55 years and older are much more likely to be improperly triaged.
10. Invasive hemodynamic monitoring is indicated for geriatric trauma patients with uncertain cardiovascular or renal disease.
11. Effort should made to optimize the cardiac index (>4 liter/min/m^2) and oxygen consumption index (170 mL/min/m^2).

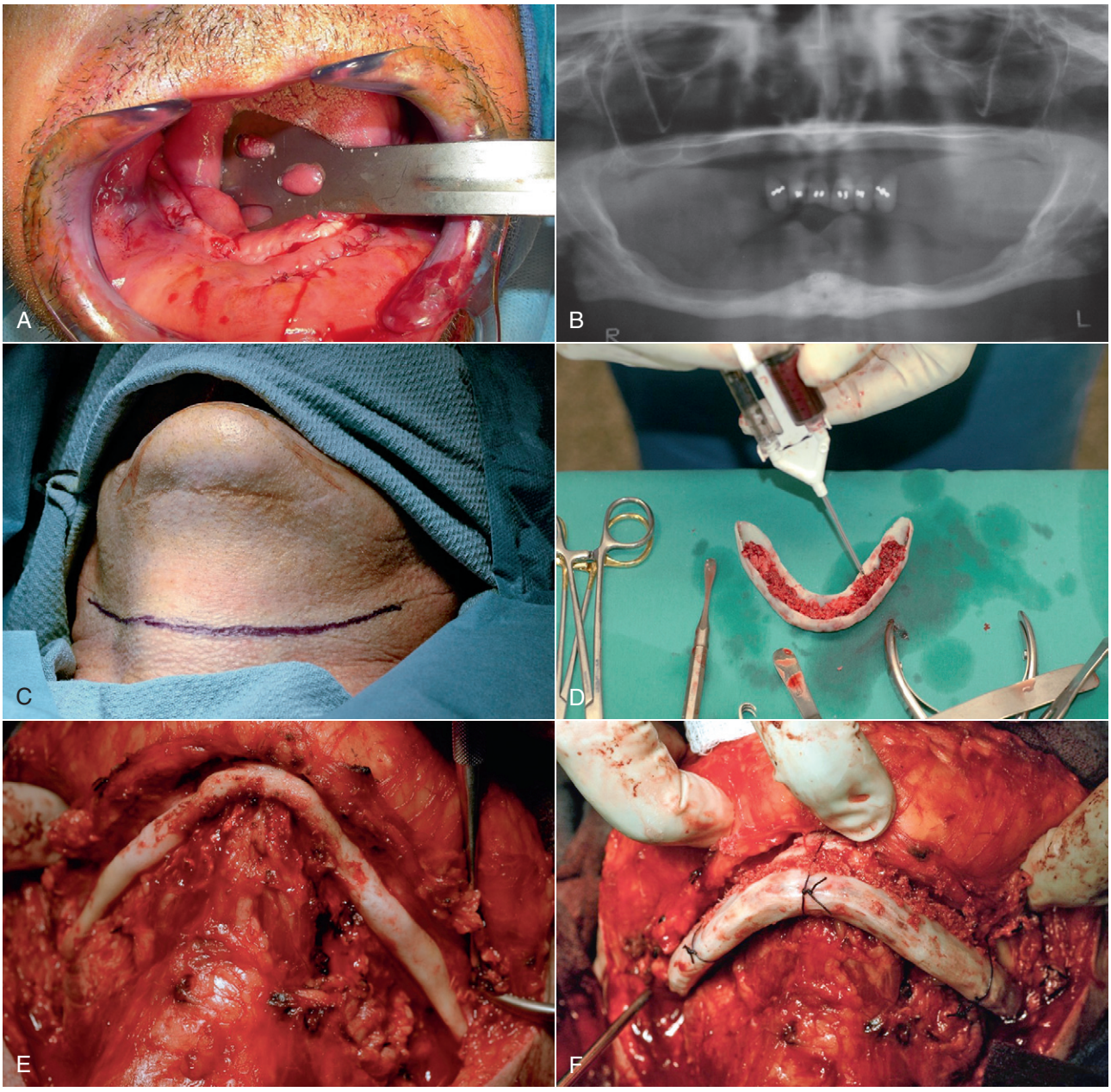


FIGURE 30-6 Reconstruction of severely atrophic mandible (6 mm) using freeze-dried cadaveric mandible packed with autogenous cancellous iliac bone. **A,** Intraoral view demonstrating a severely resorbed alveolar ridge. **B,** Panoramic view of generalized severe mandibular atrophy. **C,** Outline of incision using an existing skin crease. **D,** Hollowed-out, freeze-dried cadaver mandible filled with cancellous autograft from anterior iliac crest. **E,** Exposed view of the inferior border. **F,** Cadaver mandible is fitted to the inferior border of the patient's mandible and secured with circummandibular wires.

Because these recommendations are based on retrospective data collection, they should be applied cautiously in individual patients. Prospective, randomized controlled trials are necessary so that valid evidence-based guidelines for geriatric trauma care can be established and uniformly implemented.

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CHAPTER

31 Reconstruction of Avulsive Defects of the Maxillofacial Complex

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OUTLINE

Causes of Avulsive Injuries of the Maxillofacial Complex
Firearm Injuries
Occupational and Farm Injuries
Tearing Injuries
Abrasion Injuries
Incidence
Assessment and Initial Medical Management
Classification
Avulsive Soft Tissue Maxillofacial Injuries
Avulsive Hard Tissue Maxillofacial Injuries
Initial Surgical Management of Avulsive Maxillofacial Injuries
Reconstruction of Avulsive Soft Tissue Injuries
Ear Avulsions
Scalp and Nasal Avulsions
Anterolateral Thigh Flap
Adjunctive Therapy for Complete and Partial Soft Tissue Avulsions of the Maxillofacial Region
Hyperbaric Oxygen Therapy
Medicinal Leech Therapy
Reconstruction of Avulsive Hard Tissue Injuries
Mandibular Reconstruction
Simple Alveolar Defects
Small Mandibular Defects Requiring No Cutaneous or Mucosal Grafting
Mandibular Condyle Defects
Large Mandibular Defects and/or Those Requiring Soft Tissue Coverage
Midfacial and Upper Facial Third Reconstruction
Special Concerns
Combined Mandibular and Midfacial and Upper Facial Hard Tissue Reconstruction
Osseointegrated Implants
Custom-Fabricated, Patient-Specific Implants and Endocultivation
Computerized Surgical Stents
Composite Tissue Allograft Transplant

A man's face as a rule says more, and more interesting things, than his mouth, for it is a compendium of everything his mouth will ever say, in that it is the monogram of all this man's thoughts and aspirations.

Arthur Schopenhauer, German philosopher

Trauma, including unintentional and violent injury, remains the leading cause of death and disability in the United States for individuals from the age of 1 year to the mid-40s (Fig. 31-1).[1] Arguably, no other physical deformity is as psychologically devastating as a traumatic defect to the face, exceeded only by those defects involving avulsive loss of hard and soft tissue.[2] Although the art and science of prosthetic limb replacement has advanced tremendously during the past 2 decades, with current research investigating true brain-machine integration, no satisfactory component has been developed to serve as a replacement for the facial unit.[3,4] Imagine an individual living a normal life, such as eating in a restaurant, shopping, or sitting at the local coffee house while using their laptop computer to browse the Internet. And, in one cruel twist of fate, the focal point of their self-image, and the source of their engagement with society, is permanently mutilated. Avulsed hard and soft facial tissues leave enormous psychological, interpersonal, functional, and physical disabilities, well beyond those encountered by those with congenital deformities or traumatic amputation of the extremities; integration back into society is slightly easier if you have never known life without your congenital defect, or your lower extremity amputation has been replaced by a state of the art prosthetic limb. The comprehensive management of avulsive facial injuries rarely has been the focus of any epidemiologic, demographic, or other therapeutic studies. A complete review of the potential treatment modalities, soft and hard tissue reconstruction options, and management principles of avulsive hard and soft tissue wounds to the craniomaxillofacial unit is beyond the scope of this chapter, and would take volumes of data and literature to address the topic adequately. Instead, the focus here is to provide the practicing facial trauma surgeon with an overview of the causes, incidence, diagnosis, classification, and surgical management of these devastating injuries.

CAUSES OF AVULSIVE INJURIES OF THE MAXILLOFACIAL COMPLEX

The description of injury can be summarized simply as the effects of energy overcoming inertia. Our study of physics, and the work of Sir Isaac Newton, has shown that the velocity of a body remains constant unless the body is acted on by an external force. To quote Haug, "In the

10 Leading Causes of Death, United States
2007, All Races, Both Sexes

	Age Groups											
Rank	<1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65+	All ages
1	Congenital anomalies 5,785	Unintentional injury 1,588	Unintentional injury 965	Unintentional injury 965	Unintentional injury 6,493	Unintentional injury 9,404	Unintentional injury 14,977	Unintentional injury 16,931	Malignant neoplasms 50,167	Malignant neoplasms 103,171	Heart disease 496,095	Heart disease 616,067
2	Short gestation 4,857	Congenital anomalies 546	Malignant neoplasms 480	Malignant neoplasms 479	Homicide 2,224	Homicide 3,327	Suicide 5,278	Malignant neoplasms 13,288	Heart disease 37,434	Heart disease 65,527	Malignant neoplasms 389,730	Malignant neoplasms 562,872
3	SIDS 2,453	Homicide 398	Congenital anomalies 196	Homicide 213	Suicide 1,481	Suicide 2,659	Homicide 4,758	Heart disease 11,839	Unintentional injury 20,315	Chronic low. Respiratory disease 12,777	Cerebro-vascular 115,961	Cerebro-vascular 135,952
4	Maternal pregnancy comp. 1,769	Malignant neoplasms 364	Homicide 133	Suicide 180	Malignant neoplasms 673	Malignant neoplasms 980	Malignant neoplasms 3,463	Suicide 6,722	Liver disease 8,212	Unintentional injury 12,193	Chronic low. Respiratory disease 109,562	Chronic low. Respiratory disease 127,924
5	Unintentional injury 1,285	Heart disease 173	Heart disease 110	Congenital anomalies 178	Heart disease 346	Heart disease 738	Heart disease 3,223	HIV 3,572	Suicide 7,778	Diabetes mellitus 11,304	Alzheimer's disease 73,797	Unintentional injury 123,706
6	Placenta cord membranes 1,135	Influenza and pneumonia 109	Chronic low. Respiratory disease 54	Heart disease 131	Congenital anomalies 191	Congenital anomalies 211	HIV 1,091	Homicide 3,052	Cerebro-vascular 6,385	Cerebro-vascular 10,500	Diabetes mellitus 51,528	Alzheimer's disease 74,632
7	Bacterial sepsis 820	Septicemia 78	Influenza and pneumonia 48	Chronic low. Respiratory disease 64	Cerebro-vascular 71	HIV 127	Diabetes mellitus 610	Liver disease 2,570	Diabetes mellitus 5,753	Liver disease 8,004	Influenza and pneumonia 45,941	Diabetes mellitus 71,382
8	Respiratory distress 789	Perinatal period 70	Benign neoplasms 41	Influenza and pneumonia 55	Chronic low. Respiratory disease 71	Cerebro-vascular 124	Cerebro-vascular 505	Cerebro-vascular 2,133	HIV 4,156	Suicide 5,069	Nephritis 38,484	Influenza and pneumonia 52,717
9	Circulatory system disease 624	Benign neoplasms 59	Cerebro-vascular 38	Cerebro-vascular 45	Diabetes mellitus 55	Complicated pregnancy 116	Congenital anomalies 417	Diabetes mellitus 1,984	Chronic low. Respiratory disease 4,153	Nephritis 4,440	Unintentional injury 38,292	Nephritis 46,448
10	Neonatal hemorrhage 597	Chronic low. Respiratory disease 57	Septicemia 36	Benign neoplasms 43	Septicemia 53	Diabetes mellitus 113	Liver disease 384	Septicemia 910	Viral herpatitis 2,815	Septicemia 4,231	Septicemia 26,362	Septicemia 34,828

FIGURE 31-1 Statistics for leading causes of death in the United States, 2007. (From National Center for Injury Prevention and Control: WISQARS leading causes of death reports, 1999-2007, 2010 [http://webappa.cdc.gov/sasweb/ncipc/leadcaus10.html].)

case of maxillofacial trauma, the object is the specific maxillofacial body part, and kinetic energy is provided by the wounding agent, *either* bullet, bludgeon, or motor vehicle.[5] When considering avulsive defects of the maxillofacial complex, a sufficient quantity of energy must be delivered to the body part to remove it from its native bed. Kinetic energy (KE) is related as ½ mv^2 (where m = mass and v = velocity), but Cunningham has hypothesized that softer tissues, such as brain and muscle, are associated with a lower exponent of injury (0.5) than harder tissues, such as bone, which would have a higher exponent (2.5) and therefore a higher likelihood of permanent injury. Thus, the corrected formula for estimating wounding capacity by kinetic energy would be as follows:

$$KE = \frac{1}{2}mv^{0.5} \text{ to } KE = \frac{1}{2}mv^{2.5}$$

depending on the location of the trauma.[6]

FIREARM INJURIES

Firearm injuries have been described in detail in another chapter of this text and are acknowledged as possibly the most common source of avulsive tissue loss of the craniomaxillofacial components currently seen by facial trauma specialists. Whether sustained as a the result of self-inflicted wound, accidental hunting or recreational incident, criminal activity, or of military or professional service to the community, high-energy avulsive ballistic wounds present a vexing treatment dilemma to restore form, function, and cosmesis adequately to the facial unit.[6-8]

OCCUPATIONAL AND FARM INJURIES

Industrial or agricultural accidents possess the potential to create severe crush or avulsive wounds of the soft and hard tissues of the maxillofacial complex. Whether low or high velocity, the sheer mass of the injury platform associated with industrial machinery create tissue injury patterns characteristics of a high-energy impact.[9-11] As seen in firearm and ballistic injuries, if the injury pattern is consistent with a high-energy impact, the degree of tissue disruption occurring in the hard and/or soft tissues may result in separation from the native tissue bed and avulsion. If the wounding mechanism is a crush injury, the soft tissue overlying the bone can sustain catastrophic compromise to the vascular pedicle, resulting in soft and/or hard tissue necrosis. This necrosis, resulting in partial loss of tissue or the physical structure or complete loss of the anatomic component, has the same physical outcome as avulsion.

TEARING INJURIES

Shearing or cutting injuries created by blades or saws, tearing wounds caused by industrial machinery, and bites inflicted by animals are capable of creating avulsive defects and contaminated crush injuries of the craniomaxillofacial unit.[12-16] Whereas large domestic animals can bite with forces as great as 450 lb/inch, animals in the wild, such as bears or jaguars, can have bite forces in excess of 1700 lb/inch[2,17] (Fig. 31-2). This form of crush injury can be contaminated by a wide range of microorganisms, including viruses, bacteria, and mycobacteria. Significant areas of soft tissue overlying the facial skeleton may be avulsed, crushed, or bruised, compromising vascular supply and delaying initiation of the body's host defense mechanism. Gross contaminants on machinery parts, including industrial residue and/or microbial sources from many sites of origin, serve as infectious reservoirs, further complicating the normal healing cascade.



FIGURE 31-2 Victim of a mauling by a jaguar *(Panthera onca).* Note the bite wounds to the scalp as the jaguar was attempting to crush the patient's skull.



FIGURE 31-3 MVA victim who suffered severe abrasion and total avulsion of the external ear and upper eyelid. The victim's automobile flipped on the driver's side door, breaking the window. The victim was restrained by his seatbelt and was held against the roadway as the vehicle continued to slide along the pavement.

ABRASION INJURIES

Abrasion injuries are another source of avulsive tissue loss in the craniomaxillofacial region, which have the potential to create horrific defects. Although relatively unusual for a victim to be actively dragged by a moving object, when it does occur, as seen in a motor vehicle accident (MVA) or a motorcyclist sliding along the road, the overlying soft tissues will bear the brunt of the injury[18-20] (Fig. 31-3). Protruding anatomic structures,

skin, muscles, and neurovascular components will be lost before the bone is potentially injured or avulsed. The result is an injury that is grossly contaminated and devoid of any healthy soft tissue for local flap reconstruction, and that will require adjacent tissue recruitment or remote transfer for adequate rehabilitation.[21]

INCIDENCE

Historically underreported in the craniomaxillofacial trauma literature, or noted primarily in case reports from periods of military conflict, avulsive defects to the maxillofacial region have been recognized to occur infrequently and are perceived to have a poor prognosis in regard to an acceptable cosmetic and functional repair.[5,20,21-30] In the civilian literature, the decade-long experiences of Clark et al at one of the busiest and premier level I trauma centers in the United States, have identified approximately 1.5 cases/year of avulsive tissue loss to the craniomaxillofacial region.[24] Considering the reported volume of over 6700 unique trauma patient identifiers at the shock trauma center annually, the relative infrequency of this occurrence becomes apparent.[31] Clark et al noted that approximately 46.6% of these injuries involve the mandible, maxilla, and orbit; 26.6% involve the mandible and maxilla; and 26.6% involve the mandible alone. Haug et al have reported a 1% occurrence of avulsive maxillofacial injuries (5 of 475patients) during a review of experiences at a civilian trauma center in Cleveland, with most cases involving self-inflicted gunshot wounds with shotguns in a suicide attempt.[27] In a survey of over 9400 patients treated during the Vietnam conflict, Osbon had previously noted that 9.4% exhibit an avulsion of a notable portion of their mandible.[25] Because Vietnam represented the first armed modern conflict against nonconventional military forces, the avulsive loss of mandibular anatomy was not previously experienced to the degree as seen in this conflict.[29] Dobson outlined classic forms of warfare to include: minor conventional warfare, major conventional warfare, rural attack, and terrorist attack.[32]

Rural attacks, minor conventional warfare, and major conventional warfare displayed remarkably similar historical incidences of head and neck injury, with 16%, 16%, and 15% for British, Commonwealth, and military personnel, respectively, since 1914. Terrorist attacks, however, displayed a statistically higher incidence of 21%. In the only known paper published during Operation Iraqi Freedom–Operation Enduring Freedom (OIF-OEF) with reported battlefield conditions consistent with classic military combat, Montgomery et al noted the incidence of head and neck casualties as 25% for U.S. military personnel, which is consistent with the previously reported historic norms for U.S. conflicts.[33] Dobson et al[32] have described terrorist attacks as highlighted by the unconventional use of improvised explosive devices (IEDs), which correlates with the injury pattern of the IED as used by insurgents in OIF-OEF. Most hard tissue facial injuries were sustained in the mandible due to its prominence from the facial skeleton. This was also noted in Lew et al's study of maxillomandibular fixation (MMF) and open reduction and internal fixation (ORIF) of mandible fractures being the second and fourth most commonly reported surgical procedures.[29] The placement of IEDs, usually well below the level of the head and neck, would obviously place the anterior-inferior aspect of the mandible at increased risk as the explosive force and associated shrapnel would proceed in a superior vector, striking the lower face. Modern body armor plays a significant role in the overall survivability of wounded military personnel, but Dobson et al's observations offer a unique perspective and potential additional cause for the increased craniomaxillofacial injuries seen in OIF-OEF.

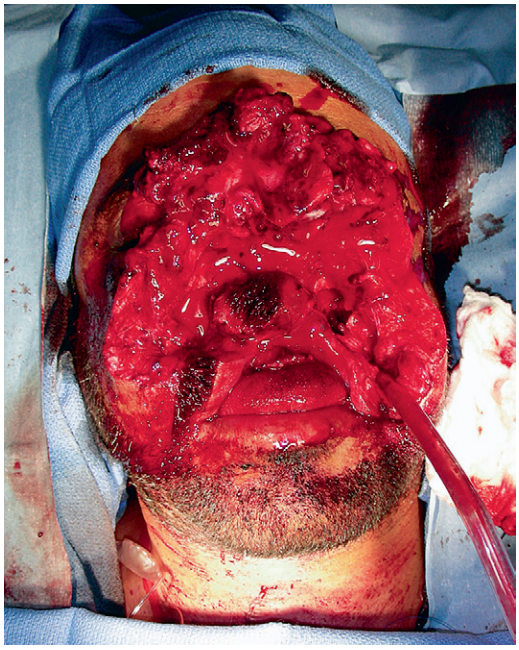


FIGURE 31-4 Avulsive injuries to the face are potentially lethal. Strict attention should be paid to establishing an airway, ventilating the patient, and then controlling hemorrhage while protecting the cervical spine.

ASSESSMENT AND INITIAL MEDICAL MANAGEMENT

Obviously, avulsive injuries to the craniomaxillofacial complex are potentially life-threatening (Fig. 31-4). Compromise of the airway through anatomic collapse, hemorrhage, foreign body obstruction, or aspiration are acknowledged as known risks, requiring immediate intervention to provide for continued oxygenation of the patient. As prescribed in the current Advanced Trauma Life Support (ATLS) protocols, securing the airway definitively should be accomplished by conventional oral or nasal endotracheal intubation, intubation of a visible component of a traumatically exposed trachea, or cricothyroidotomy or surgical tracheostomy.[21] After the airway has been established, attention should be directed to a thorough analysis of the patient for potentially lethal injuries and appropriate interventions initiated. Control of hemorrhage should be established with simple

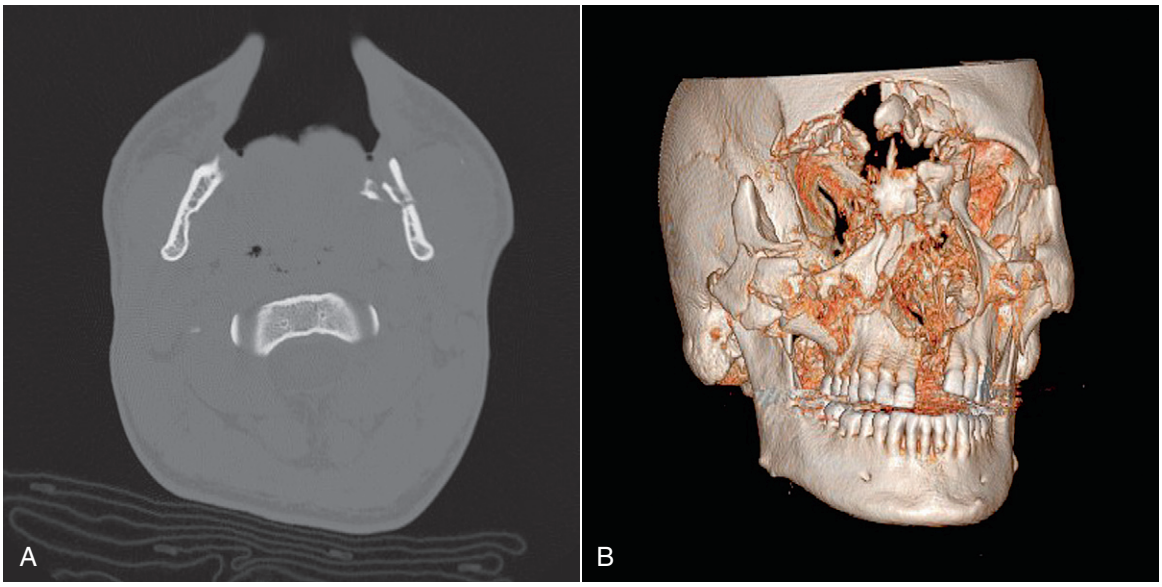


FIGURE 31-5 **A,** Note the devastating avulsive injury to the anterior mandible in this two-dimensional CT scan, indicating significant loss of bone and soft tissue. **B,** Note the clarity and identification of anatomic injuries and the presence of an avulsive loss of the anterior cranial vault floor in this three-dimensional reconstruction of a CT scan of a patient involved in a motorcycle accident.

pressure to the site by compression dressings or manual palpation, ligation of visible sources of active bleeding, or use of hemostatic dressings impregnated with components such as chitosan or similar compounds.[33] Injuries to the deep vessels of the head or neck, or those not visible from routine surgical access, may require interventional approaches to gain control of the hemorrhage.[34] During the course of fluid resuscitation, particular attention should be paid to the head and neck region because seemingly benign scalp lacerations, or those not recognized due to lack of hemorrhage secondary to hypovolemia, have been associated with exsanguinating hemorrhage as the patient's mean arterial pressure is increased.[35] If the head and neck region is covered under surgical drapes due to ongoing therapy in other areas of the body, this unrecognized source of bleeding can confound anesthesia providers and surgeons, or potentially be lethal.

After all life-threatening injuries have been recognized and successfully managed, identification of the avulsive facial defects can be accomplished. Computed tomography (CT) scans with three-dimensional reconstruction are the minimum baseline studies required for the treatment planning of avulsive craniomaxillofacial defects (Fig. 31-5). Most avulsive soft tissue injuries, and all avulsive hard tissue injuries, will be definitively treated in an operating room environment. After stabilization, medical management should commence with appropriate early nutritional and psychiatric support for the patient, with antibiotic therapy directed toward cutaneous, oral, pharyngeal, nasal, and sinus contaminants.[33,36] Because these wounds are typically contaminated, and may exhibit vascular compromise, consideration should be given to the administration of the appropriate tetanus prophylaxis (Table 31-1) and, in the case of animal bites, rabies prophylaxis (Fig. 31-6).

CLASSIFICATION

AVULSIVE SOFT TISSUE MAXILLOFACIAL INJURIES

Avulsive injuries of the soft tissue of the craniomaxillofacial region should be classified as either complete avulsion or partial avulsion. Complete avulsion should be defined as an injury in which there is complete loss or detachment of a portion of the craniomaxillofacial structures, without the ability to regenerate the missing anatomic entity (Fig. 31-7). The ability to regenerate the missing component is directly related to the magnitude of the avulsion and the vascular status of the remaining soft tissue bed. A partial avulsion is defined as one in which the segment is still attached to the remaining native tissue bed, but presents with obvious concerns for viability (Fig. 31-8). The same classification holds true for scalp injuries. Complete avulsion would be one in which a portion of the scalp is lost and the magnitude of the loss is significant enough to prevent complete regeneration from the surrounding soft tissue bed (Fig. 31-9). A partial avulsion would be one in which loss of the scalp has occurred, with resultant vascular compromise, with or without the necessity for tissue replacement.

AVULSIVE HARD TISSUE MAXILLOFACIAL INJURIES

Currently, no universal classification system exists for avulsive defects of the craniomaxillofacial complex. The injuries are usually described in anatomic terms, such as mandibular, midfacial and/or upper facial third, or full thickness or partial thickness for skin, or by the wounding mechanism (e.g., low energy, thermal, high energy).[27,28] With the advent of composite facial allografts, and the various descriptions used by the transplant community to describe these surgeries, a uniform system for the description of avulsive loss of craniomaxillofacial components will likely be forthcoming in the near future.[37-40]

TABLE 31-1 Rabies Postexposure Prophylaxis (PEP) Schedule

Vaccination Status	Intervention	Regimen*
Not previously vaccinated	Wound cleansing	PEP should begin with immediate thorough cleansing of all wounds with soap and water. If available, a virucidal agent (e.g., providine-iodine solution) should be used to irrigate the wounds.
	Human rabies immune globulin (HRIG)	Administer 20 IU/kg body weight. If anatomically feasible, the full dose should be infiltrated around and into the wound(s), and any remaining volume should be administered at an anatomic site (IM) distant from vaccination administration. Also, HRIG should not be administered in the same syringe as vaccine. Because HRIG might partially suppress active production of rabies virus antibody, no more than the recommended dose should be administered.
	Vaccine	Human diploid cell vaccine (HDCV) or purified chick embryo cell vaccine (PCECV) 1.9 mL, IM (deltoid area†), one each on days 0,‡ 3, 7, and 14§
Previously vaccinated¶	Wound cleansing	PEP should begin with immediate thorough cleansing of all wounds with soap and water. If available, a virucidal agent (e.g., providine-iodine solution) should be used to irrigate the wounds.
	HRIG	HRIG should not be administered.
	Vaccine	HDCV or PCECV, 1.0 mL (deltoid area†), one each on days 0‡ and 3.

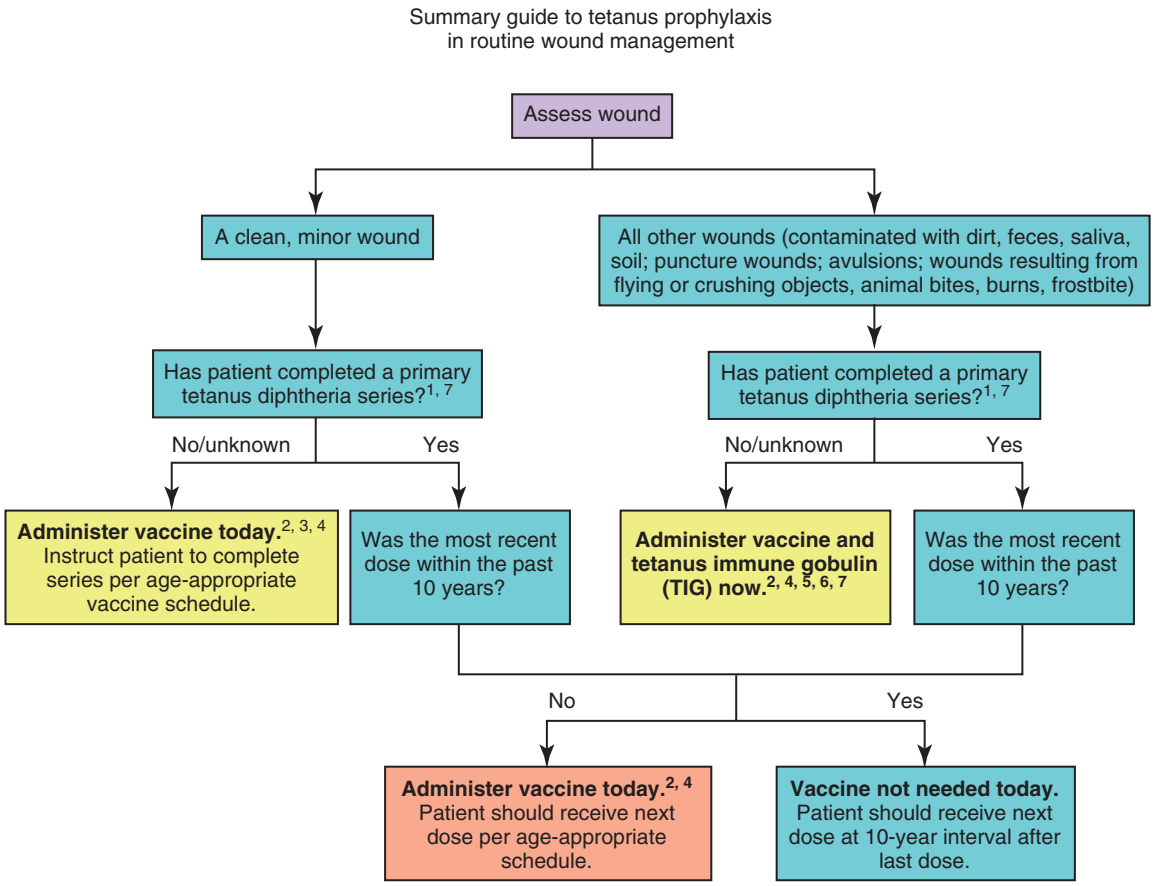
*These regimens are applicable for persons in all age groups, including children.
†The deltoid area is the only acceptable site of vaccination for adults and older children. For younger children, the outer aspect of the thigh may be used. Vaccine should never be administered in the gluteal area.
‡Day 0 is the day that dose 1 of vaccine is administered.
§For persons with immunosuppression, rabies PEP should be administered, using all five doses of vaccine on days 0, 3, 7, 14, and 28.
¶Any person with a history of preexposure vaccination with HDCV, PCECV, or rabies vaccine absorbed (RVA); prior to PEP with HDCV, PCECV or RVA; or previous vaccination with any other types of rabies vaccine and a documented history of antibody response to the prior vaccination.
Adapted from (Centers for Disease Control and Prevention: ACIP recommendations: Use of a reduced (4-dose) vaccine schedule for postexposure prophylaxis to prevent human rabies, 2011[http://www.cdc.gov/rabies/resources/acip_recommendations.html]).

INITIAL SURGICAL MANAGEMENT OF AVULSIVE MAXILLOFACIAL INJURIES

After initial stabilization and resuscitation, the patient may be transported to the operating room for their first surgical procedure. The goals of the initial operation are as follows:

1. Secure a definitive airway.
2. Control hemorrhage.
3. Explore and identify remaining native tissue and documentation of avulsed components.
4. Identify, document, and primarily repair, if possible, the presence of significant soft tissue injuries:
   a. Neurovascular structures
   b. Facial nerve
   c. Salivary structures
   d. Muscle disinsertion, detachment
   e. Globe injury
5. Salvage and maintain remaining hard and soft tissues.
6. Establish anatomic landmarks and an osseous foundation for definitive reconstruction.
7. Prevent infection and subsequent exacerbation of soft tissue contracture.

Because of the likely multitude of surgical interventions and concurrent soft tissue edema that the patient will experience, not only due to the primary injury but as a tissue response to initial and subsequent fluid resuscitations, strong consideration should be given to performing an elective surgical tracheostomy if not done previously. Wound exploration for transected vessels should be performed with identification and definitive control established through ligation or cautery. Efforts should be directed to preservation of all hard and soft tissues, including those with questionable vitality, because they may be able to be preserved or surgically reattached through conventional or microsurgical techniques. Soft tissue is generally repaired from inside-out, with stable reapproximation of the deep tissues. Identification of clinically significant soft tissue injuries, including transection of the facial nerve or salivary ducts, should be performed under magnification with surgical loupes or microscope, and primary repair attempted, if feasible. The recognition of facial nerve injury is a critical finding for the rehabilitation of the patient because the determination for facial reanimation surgery and the timing for repair options have a finite period for intervention.[41,42] Hard tissue repair can be accomplished by beginning with the mandible and reestablishment of the occlusion, followed by the midface and upper facial third. The frame of the midface and upper face should be established from the supraorbital bar, lateral orbital rims, malar buttresses, and arch projection first, followed by the central midface of the naso-orbital-ethmoid (NOE) region, with final corrections of the dentoalveolar unit at the Le Fort I level accomplished last. Although the mandible-first protocol generally is applied most often, if the midface or upper facial third is minimally injured, and a more accurate reconstruction for the surgical foundation can be accomplished in those areas, reconstruction should commence in those regions.



[1] A primary series consists of a minimum of 3 doses of tetanus- and diphtheria-containing vaccine (DTaP/DTP/Tdap/DT/Td).

[2] Age-appropriate vaccine:
- DTaP for infants and children 6 weeks up to 7 years of age (or DT pediatric if pertussis vaccine is contraindicated);
- Tetanus-diphtheria (Td) toxoid for persons 7 through 9 years of age; and ≥65 years of age;
- Tdap for persons 10 through 64 years, unless the person has received a prior dose of Tdap.*

[3] No vaccine or TIG is recommended for infants <6 weeks of age with clean, minor wounds. (And no vaccine is licensed for infants <6 weeks of age.)

[4] **Tdap** is preferred for persons age 10 through 64 who have never received Tdap. Td is preferred to tetanus toxoid (TT) for persons 7 through 9 years, or ≥65 years, or those who have received a Tdap previously. If TT is administered, an adsorbed TT product is preferred to fluid TT. (All DTaP/DTP/Tdap/DT/Td products contain adsorbed tetanus toxoid.)

[5] Give TIG 250 U IM for all ages. It can and should be given simultaneously with the tetanus-containing vaccine.

[6] For infants <6 weeks of age, TIG (without vaccine) is recommended for "dirty" wounds (wounds other than clean, minor).

[7] Persons who are HIV positive should receive TIG regardless of tetanus immunization history.

FIGURE 31-6 Summary guide to tetanus prophylaxis in routine wound management. (Centers for Disease Control and Prevention: ACIP recommendations: Use of a reduced (4-dose) vaccine schedule for postexposure prophylaxis to prevent human rabies, 2011[http://www.cdc. gov/rabies/resources/acip_recommendations.html]).

The initial surgical procedure should not be considered as a definitive reconstruction, but as an examination under anesthesia, with the primary focus being to identify which structures have been lost and which remain, and to prevent further progressive or iatrogenic loss of additional hard and/or soft tissue. The existing identifiable bone should be located, identified, and rigidly fixed in an anatomic position with surgical reconstruction plates, with the reestablishment of a functional occlusion being the ultimate goal. The remaining soft tissue wound edges are arranged in as close an anatomic approximation as possible, even if tension at points of closure or blanching of the tissue occurs, and a comprehensive evaluation under anesthesia should commence, identifying as accurately as possible the avulsed or injured soft and hard tissue components.

After this primary surgery, the patient should undergo a complete CT imaging survey to evaluate anatomic reduction or fixation, better identify tissue loss, and assist with treatment planning for an early reconstruction. As described by Powers and Robertson and others, the patient will likely require frequent returns to the operating room at 2- to 3-day intervals to control infection, decontaminate and/or débride necrotic tissue, and reassess vitality in the remaining tissues.[33,43] Good mucosal coverage with adequate vascularity of the osseous structures internally, and similar cutaneous coverage externally, will help prevent infection and mitigate hard tissue necrosis.



FIGURE 31-7 Complete avulsion of external ear as a consequence of being an ejected passenger from an MVA and sliding across the pavement of the highway.



FIGURE 31-8 Partial avulsion of the ear. Note that only a small pedicle is maintaining vascularity to the entire ear. This patient underwent immediate closure of the partial ear avulsion and had an uneventful postoperative course.

RECONSTRUCTION OF AVULSIVE SOFT TISSUE INJURIES

Initial determinations of reconstructive options for avulsive soft tissue injuries are aligned under the decision to proceed with prosthetic reconstruction, or native soft tissue utilization. As previously described by Haug and Carlson, the necessity for adjunctive therapy of native soft tissue rehabilitation is determined by the following parameters: temperature, color, oximetry, and capillary refill.[5]

Unimpeded vascular supply provides nutrients and warmth to a body part, so comparing the temperature of the reapproximated avulsed segment with its surrounding tissue allows for a qualitative assessment of adequate arterial circulation. Color is an acceptable evaluator of blood flow—venous congestion will appear blue or purple and arterial insufficiency will appear pale or white



FIGURE 31-9 Complete avulsion of the scalp as a consequence of an industrial accident. The patient had long hair, which was maintained in a tight pony tail, and a portion of the hair became entrapped in a machine driven fan belt.

when compared with surrounding soft tissue. Pulse oximetry may provide another measure for determining the adequacy of vascularity to a reconstructed segment. Although no specific guidelines are found in the literature, the Sao_2 of a repaired component of soft tissue should probably be at least 80% for predictable survivability. Capillary refill is a useful clinical tool to assess venous congestion versus arterial compromise of partial avulsions of facial soft tissue. Capillary refill of facial soft tissue should be about 2 to 3 seconds. Prolonged capillary refills longer than 3 seconds suggest arterial insufficiency and rapid capillary refills shorter than 1 to 2 seconds indicate venous congestion. Appropriate identification of the need for adjunctive procedures to assist with maintenance of vascularity can be achieved by following these recommendations.

EAR AVULSIONS

Débridement of the wound begins the process of repair in treating complete and partial avulsions of the ear. The concept of débridement is well established in the literature and is a critical component in the management of avulsive injuries to the craniomaxillofacial complex.[33,43] Serial washouts and débridement of tissue have become mainstays for craniomaxillofacial trauma surgeons in the treatment of these injuries. Unfortunately for many surgeons, the term *débridement* is synonymous with the absolute removal of tissue in the operating room. Although devitalized necrotic tissue does require excision, a more accurate definition of the goal of serial washouts should be the term *decontamination.* Preservation of all viable tissues is a critical component in the management of gunshot wounds to the maxillofacial region. Once tissues

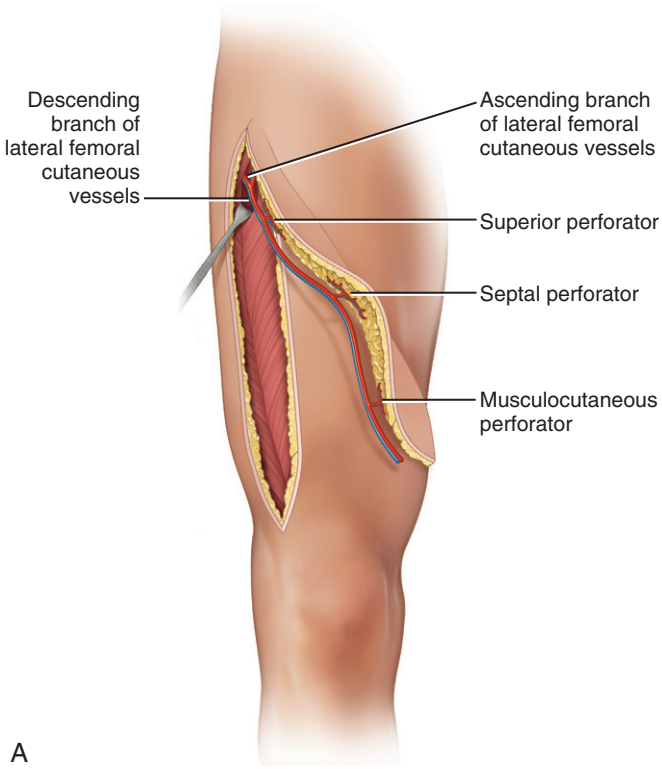


FIGURE 31-10 **A,** Anatomy for anterolateral thigh flap. **B,** Anterolateral thigh flap ready to be delivered from the donor bed.

are lost, the surgeon is faced with two choices—compromise the anterior-posterior projection to allow for primary closure of native tissue or transfer additional tissue to the region via pedicled or microvascular grafts. Avoidance of the need for tissue transfer should be the goal, and judicious use of the practice of decontamination will assist in achieving the desired results.

Pulsatile jet irrigation with normal saline may be indicated for the débridement of avulsive soft tissue wounds. It is important, however, to prevent iatrogenic trauma with overexuberant use of jet irrigation. In the case of complete avulsion of an ear segment, in which the segment has been salvaged and accompanies the patient to the hospital, the component should be cleansed with antiseptic solution and a salvage procedure attempted with microvascular anastomosis, if possible.[44] When microvascular anastomosis of an avulsed segment is not possible, the surgeon may elect to proceed with primary reattachment of the ear and adjunctive use of hyperbaric oxygen or leech therapy to address the resultant venous congestion and vascular concerns.[45] If the ear has not been stored or transported in a cool environment, excessive contamination exists, or perceived excessive ischemia time with obvious tissue necrosis is present, the wound may be primarily closed and future reconstruction planned. Partial ear avulsions are those in which the traumatized segment of the ear is still attached but there is questionable vascularity to the partially avulsed segment. Partial ear avulsions should be treated with primary closure of the partially avulsed segment as soon as possible to prevent tissue necrosis and loss.

SCALP AND NASAL AVULSIONS

Partial scalp avulsions are those in which significant lacerations have occurred in the otherwise intact and preserved scalp, thereby compromising its blood supply and impairing healing. Although most surgeons are inclined to provide primary closure of such wounds, it is advisable also to consider adjunctive therapy. The completely avulsed scalp injury requires local flap surgery for reconstruction of the avulsed segment or, in the case of larger magnitude avulsions, reconstruction with skin grafts or possibly microvascular flaps. As with ear avulsions, it is always advisable to attempt to locate vessels in the completely avulsed scalp for microvascular reconstruction in the event that it is brought to the hospital with the patient. If not possible, it may be appropriate simply to reattach the avulsed segment and resort to adjunctive therapy to salvage the avulsion. Complete and partial avulsions of the nasal complex require a management approach identical to that of the ear and scalp.

ANTEROLATERAL THIGH FLAP

Avulsive loss of soft tissue exclusively is seen in the injury patterns described, such as tearing, shearing, abrasion, and some forms of ballistic damage. Although rotational soft tissue techniques, including pedicled myocutaneous flaps, present a viable treatment option in some scenarios, the transfer of tissue is limited by the attachments of the vascular base or available size of tissue to be transported. When a large volume of soft tissue only is required at a remote site, the anterolateral thigh (ALT) flap is a proven choice, easily delivering skin pedicles 8 to 10 cm in width and 25 cm in length, still allowing for a cosmetic primary closure of the donor site[46,47] (Fig. 31-10). Larger skin paddles may be taken, but may require other forms of closure, such as skin grafting. The ALT flap requires a microvascular craniomaxillofacial surgeon for its application in clinical scenarios. Microvascular reconstruction has the advantage of providing greater quantities of tissue of varying types for replacement of the defect, with large volumes of bone and associated soft tissue transported to the recipient site, depending on the donor source chosen. Microvascular transfer allows for the immediate restoration of contour with a reliable vascular supply ensuring osseous incorporation and healing, and also permitting the transfer of soft tissue to replace lost

or damaged skin or mucosa. Microvascular reconstruction requires additional training and expertise in addition to the added expense of specialized surgical equipment necessary for clinical use. Due to the length of added surgical time associated with this treatment modality, two surgical teams are typically used, with one team working at the recipient site and the other team harvesting the surgical graft.

The ALT flap is marked with the patient supine on the operating table. Readily palpable bony landmarks, the anterior superior iliac spine and superolateral patella, are identified and a line connecting the two is drawn.[46,47] A Doppler signal identifies the cutaneous perforator vessels from the lateral circumflex femoral artery and the skin paddle is designed to the dimensions required. The initial exploratory incision is performed along the medial aspect of the proposed flap. The ALT flap may be raised in a suprafascial or subfascial plane, depending on the desired thickness. Once adequate perforators have been identified, the flap is elevated by retrograde dissection of the selected perforators to the source vessels in the intermuscular septum. Meticulous intramuscular dissection, with strict adherence to hemostasis and maintenance of perforator integrity, is of paramount importance and continues proximally until the desired length of the pedicle is obtained. The vascular pedicle is transected and layered closure of the harvest site is accomplished, generally without the need for drains. The advantages to the ALT flap include its versatility as a cutaneous or fasciocutaneous flap for tissue resurfacing or as a musculocutaneous flap for oromandibular or complex maxillary defects. Coupled with the large volume of soft tissue obtained with essentially minimal donor site morbidity, the ALT flap has become a versatile first choice for soft tissue reconstruction of avulsive craniomaxillofacial defects.

ADJUNCTIVE THERAPY FOR COMPLETE AND PARTIAL SOFT TISSUE AVULSIONS OF THE MAXILLOFACIAL REGION

HYPERBARIC OXYGEN THERAPY

Although not indicated for routine management of soft or hard tissue injuries, in the setting of vascular compromise to the craniomaxillofacial subunit, hyperbaric oxygen therapy may have an important role in maintaining the viability of an injured anatomic region. Normal atmospheric sea level pressure is referred to as 1 atmosphere absolute (1 ATA). An increase in atmospheric pressure to 2 to 3 ATA results in an increase in the oxygen-carrying capacity of the blood by hemoglobin saturation and dissolving oxygen in plasma to meet all the requirements for normal maintenance and physiologic activity, even if there is insufficient hemoglobin to bind and transport oxygen. This may occur in an avulsive tissue loss or compromised vascular pedicle.[5,48-51] As hyperbaric oxygen saturation increases the oxygen supply to all body tissues, including those that have sustained partial or complete avulsion, hyperoxygenation can minimize or eliminate hypoxic and ischemic conditions and can be used for temporization of such injuries. This adjunctive therapy, especially in the acute traumatic injury, can maintain the viability of those severely compromised wound areas considered to be questionable or hopeless, thereby preventing clinically significant tissue loss.



FIGURE 31-11 The medicinal leech, *Hirudo medicinalis*.

MEDICINAL LEECH THERAPY

In complete or partial avulsions of facial structures, significant venous congestion may be encountered in the postoperative period. If left untreated, the reconstructed tissue may undergo necrosis secondary to vascular compromise and be lost, necessitating free tissue transfer or rotational coverage with adjacent native tissue. Incision and drainage of the venous congestion may not be successful in releasing the pressure. Although hyperbaric oxygen may assist with delivery of oxygen to injured tissues, it will not be effective in relieving the congestion from venous stasis. In clinical situations such as this, medicinal leeches may be considered.[52-54] The medicinal leech, *Hirudo medicinalis,* acts by removing excess blood and secreting hirudin, a long-acting anticoagulant that promotes prolonged venous drainage from the puncture site, into the tissues, resulting in a lowering of venous pressure until the injured tissues can reestablish normal blood flow (Fig. 31-11). During active use of medicinal leech therapy, consideration for protection of the body cavities, such as the external auditory canal, nasal aperture, or oral cavity, should be performed with obliteration of dead space with gauze dressings, packings, or surgical drapes to prevent migration and loss of the leech. While therapy is ongoing, antibiotic prophylaxis should be directed against *Aeromonas* spp.[5] The leeches spontaneously fall off when engorged and therapy may need to be repeated several times to achieve the desired result.

RECONSTRUCTION OF AVULSIVE HARD TISSUE INJURIES

MANDIBULAR RECONSTRUCTION

After the patient has been medically stabilized, the following steps are needed prior to scheduling the primary reconstruction:

TABLE 31-2 Advantages and Disadvantages of Various Osseous Graft Types for Craniofacial Reconstruction

Type of Graft	Available Bone	Soft Tissue Pedicle	Simultaneous Harvest	Advantages	Disadvantages
Fibula free flap	25 cm	24 × 9 cm	Yes	Mandibular reconstruction, Any length, versatile, supports implants, blood supply permits contouring in osteotomies	May require skin graft for closure, edema, ankle instability, questionably reliable skin paddle
Scapula free flap	14 cm	12 × 20 cm	No	Wide latitude in placement, relative to skin or muscle, versatile orbit, base of skull, maxilla, zygoma, palate, mandibular defect with massive intraoral/extraoral soft tissue defect	Shoulder debilitation, widened scar if oriented transversely and closed primarily, thin bone often inadequate for implants
Iliac crest free flap	15 cm	10 × 20 cm	Yes	Reconstruction of large mandibular defects, curve parallels mandible, mass permits contouring osteotomies, supports implants	Technically difficult, especially in obese patients, abdominal weakness and hernia, hemorrhage, thigh pain, +/– sensory deficits, gait disturbance, cutaneous portion of flap bulky
Radial forearm free flap	12 cm	Any portion of forearm skin	Yes	Intermediate mandibular defect requiring bone and soft tissue coverage	Radius fracture, poor cosmesis, sensory deficit, bone often inadequate for implants, unicortical bone intolerant of osteotomies
Ilium cancellous or corticocancellous graft	30-50 mL (anterior) 80-100 mL (posterior)	None	Yes (anterior) No (posterior)	Mandibular defects, orbital reconstruction, augmentation of small midfacial defects	Hernia, hemorrhage, seroma, thigh pain +/– sensory deficits, gait disturbance, intraperitoneal communication (ileus, perforated viscus)
Split calvarium	Extensive	None	No	Orbital reconstruction, nasal dorsum, zygoma, maxilla, mandible	Palpable or cosmetic defect if outer table used dural tear
Rib	12-18 cm	None	Yes	Mandibular condyle, ramus, anterior mandible, midface (split), contourable	Pneumothorax

From Haug RH, Bradrick JP, Billy ML: Avulsive hard tissue facial injuries. In Fonseca RJ, editor: *Oral and maxillofacial surgery*, Philadelphia, 2000, WB Saunders.

1. Appropriate clinical examination with identification of hard and soft tissue deficits
   - Quantity of hard and soft tissue replacement
   - Potential nerve grafting: Reanimation surgery
   - Prosthetic rehabilitation plans: Auricular, ocular, nasal; dental
2. Adequate radiologic studies
   - Three-dimensional reconstruction
   - Fabrication of stereolithography models
3. Soft tissue decontamination and débridement accomplished
   - Vitality of remaining soft and hard tissues established
   - Sites free of potential infection: White blood cell count
4. Viability of native vascular or recipient bed confirmed

The patient's needs are then assessed and matched with the appropriate osseous and soft tissue donor sites, considering the advantages and disadvantages of each modality (Table 31-2).

SIMPLE ALVEOLAR DEFECTS

Minor alveolar defects of the maxilla or the mandible may be reconstructed by using soft tissue local flaps or tissue expansion, followed by cancellous bone augmentation from the lateral anterior iliac crest or tibial bone harvest. This harvest can be performed with a minimally invasive trephine or the traditional open approach. For the anterior iliac crest harvest, the determination for local anesthesia, conscious sedation, or general anesthesia will be the choice of the surgeon. In a minimally invasive trephine approach, the anterior ilium should be palpated, determining the position of the crest and

anterior superior iliac spine.[55-59] After placement of subcutaneous or subperiosteal local anesthesia, the crest of the ilium is secured, the overlying skin tensed, and a stab incision with a sterile scalpel blade is accomplished that is slightly larger than the diameter of the trephine. After incision, the subcutaneous fat is divided and Scarpa's fascia is identified. The fascia is incised down through the periosteum with the scalpel blade and is laterally displaced from the bone. The trephine is introduced and cancellous bone is removed with each pass until a sufficient amount has been harvested to overfill the defect.

In the tibial harvest technique, Gerdy's tubercle is located and identified on the anterior surface of the lateral proximal tibia where the iliotibial tract attaches.[60] Palpation of the tubercle is essential to avoid violation of the articular surface of the tibial plateau and head of the fibula. A stab incision is accomplished through the skin to the layer of the fascia of the iliotibial tract and periosteum, which is laterally displaced, allowing direct access to the surface of Gerdy's tubercle and underlying cancellous bone. A layer of subcutaneous fat may be readily encountered during the dissection, based on the body habitus of the patient. Care should be taken to verify correct angulation and depth of the trephine to prevent potential iatrogenic violation of the joint space of the knee. Direct pressure, electrocautery, or microfibrillar collagen is useful in controlling hemorrhage. The graft may be stored in normal saline-soaked gauze until placement and then a layered closure is performed. The defect in the alveolus is repaired by first creating an incision through the mucosa along the crest of the remaining ridge adjacent to the defect, much like that created for repair of secondary cleft alveolus deformities. After incision and subperiosteal dissection, the defect is examined, curetted free of fibrous tissue, and the harvested bone placed. Consideration for the use of platelet-rich plasma or collagen-based barrier membranes to prevent fibrous ingrowth into the bone graft is at the discretion of the surgeon.

SMALL MANDIBULAR DEFECTS REQUIRING NO CUTANEOUS OR MUCOSAL GRAFTING

Anterior Ilium

Mandibular defects of the body, angle, ramus, or symphysis that are 5 cm or smaller and require no soft tissue augmentation may be reconstructed with cancellous or corticocancellous bone grafts from the anterior ilium[55-59] This technique has the advantage of having the patient remain in the supine position, facilitating the option for a simultaneous two-team approach. The disadvantages are that simultaneous soft tissue grafting is impossible with the free tissue transfer method and only relatively small osseous defects may be repaired. For this reconstructive procedure, the patient is maintained in a supine position under general anesthesia and a hip roll is placed under the patient's hip to be harvested to make the ilium more prominent. If two teams are used for the reconstruction, the first approaches the mandibular defect and the second harvests the bone from the ilium. If this is a dentate procedure, the patient should be placed into MMF by whatever fashion is preferred. The various methods for surgical access to the mandible are covered elsewhere in this text in greater detail, and will be deferred to the prerogative of the individual operative surgeon. Regardless of the method for chosen access, residual fibrous tissue must be removed, along with avascular bone, from the defect, to provide adequate recipient site viability. If cancellous bone is the reconstruction choice, a titanium reconstruction plate may be placed to maintain proper positioning of the recipient hard tissue sites and serve as a foundation for bone graft support. If a corticocancellous graft is used, the reconstruction plate should be coupled with a mitered recipient site and modified with a reciprocating or sagittal saw in a stepped fashion to create butt joints.[5]

As the recipient site is being prepared, the graft harvest team should identify the iliac crest and anterior superior iliac spine and then manually reposition the overlying skin in a medial direction. This alteration of tissue planes in a medial vector will prevent the incision from lying immediately over the anterior iliac spine, increasing cosmesis and preventing trauma from wearing of pants or a belt postoperatively. A 5- to 6-cm long incision should be created through skin, which when not tensed will lay 3 cm lateral to the crest. The sensory nerves that are most commonly encountered in this field are the lateral cutaneous branches of the subcostal and iliohypogastric nerves. Approximately one third of patients will experience transient sensory deficits of the skin over the gluteus medius and gluteus minimus subsequent to this approach.[5] The dissection is carried out as described earlier for the cancellous-only retrieval technique.

For adolescents and growing children treated before ossification of the iliac crest, a lateral cortical window is opened with saws or chisels below the crest to eliminate the risk of growth disturbances by damaging the cartilaginous cap, or the cap can be carefully split in a clamshell manner during the dissection to prevent an iatrogenic avulsion. The cancellous marrow or corticocancellous blocks are then harvested. A medial osteotomy is made with saws or chisels, and then the iliac crest is fractured medially while still attached to the abdominal muscles. Cancellous bone may then be curetted from the donor site.[55-59]

To obtain a corticocancellous block graft, a more extensive dissection will be required medially under the iliacus muscle. Saws and chisels may be used to separate the medial table and corticocancellous block from the ilium, taking care during the outfracture of the medial wall to guard against excessive lateral forces being applied to the body of the remaining ilium and subsequent iatrogenic fracture. Hemorrhage is best controlled with electrocautery and/or microfibrillar collagen. The wound is closed in layers and a pressure dressing may be used to eliminate potential dead space. Postoperative infusion pumps, elastomeric or mechanical, and administering controlled doses of local anesthesia have proven beneficial in controlling the patient's pain, reducing the need for postoperative narcotics and expediting postsurgical ambulation.[61-64] Postoperative antibiotics can be administered based on the surgeon's preference.

Posterior Ilium

For bone defects larger than 5 cm, without the need for simultaneous soft tissue coverage, the posterior ilium is the preferred donor site choice. The primary disadvantage of this technique is the need to have the patient in the prone position for harvest, and then turned intraoperatively back to the supine position for reconstruction and stabilization of the graft. After stabilizing the patient in the prone jackknife position, the sacroiliac articulation, dorsal lumbar spines, and posterior iliac crest should be identified by palpation and marked with a surgical marking pen.[56,57,64] A 6- to 7-cm semilunar incision is created over the posterior iliac crest through the skin and subcutaneous fat from the posterosuperior iliac spine in a vector superiorly and laterally. The middle and superior cluneal nerves can be avoided by placing the incision along the posterior iliac crest between the sacroiliac junction and the region superior and lateral to the posterior gluteal ridge. Identification of the lumbodorsal fascia ensures the location of a proper depth of dissection. The periosteum should be incised in the intermuscular plane over the posterior iliac crest. Subperiosteal dissection of the gluteus maximus and gluteus medius at the posterior gluteal line will expose the lateral cortex. Injury to the sciatic nerve and superior gluteal vessels within the greater sciatic notch may be prevented by carefully restricting exposure cephalad to an imaginary line drawn perpendicular to the operating table from the posterosuperior iliac spine. Caution should be exercised when harvesting cancellous marrow because the medial cortical table is thin and friable and is easily perforated into the sacroiliac articulation. Microfibrillar collagen, gelatin foam, or other hemostatic agents may be applied to the donor site before closure, decreasing postoperative hemorrhage. Postoperative local anesthesia infusion pumps may be considered as an adjunct in controlling the patient's pain, reducing the need for postoperative narcotics or inpatient physical therapy consults, and expediting postsurgical ambulation.[61-63]

MANDIBULAR CONDYLE DEFECTS

Avulsive loss of the mandibular condylar apparatus presents a unique challenge to the craniomaxillofacial trauma surgeon, primarily due to the limited availability of reconstructive options. Although exotic choices can be found in the literature, two basic choices predominate in current treatment philosophy: prosthetic joint reconstruction and costochondral rib graft.[65-71] The costochondral graft can be readily harvested in a two-team approach and fashioned morphologically to re-create a condyle. The cartilaginous cap is an appropriate reconstructive choice for communication with the glenoid fossa or temporomandibular joint (TMJ) disc, minimizing the risk for bony or fibrous ankylosis and restricted mandibular function. Surgical risks and drawbacks include second surgical site morbidity, potential for iatrogenic pneumothorax, and postoperative pain with inspiration, leading to atelectasis or decreased pulmonary function. Prosthetic joint reconstruction alleviates the need for an additional surgical site with its associated morbidity, but the patient runs the risk of eventual replacement during their lifetime, as well as the potential for a foreign body response by the patient's immune system.

Detailed surgical approaches to the TMJ region and mandibular condyle are covered in other chapters of this text and will not be reviewed here. To harvest a costochondral graft, the skin adjacent to the fourth, fifth, or sixth rib is identified and an incision is made through the skin and subcutaneous tissue directly over the rib to be harvested for males; for females, the incision is created in the inframammary crease for aesthetic concerns. The pectoralis major and then the intercostal muscles are identified, along with the rib and junction to the sternum. The incision is created along the rib, through periosteum from the midaxillary to midclavicular line.[5,70,71] Periosteum is carefully dissected free, with care taken not to perforate the pleura, and a rib cutter is used to section the rib once an appropriate length has been determined (Fig. 31-12*A*). A scalpel blade may be used to carve the cartilaginous component of the rib from the costosternal junction so as not to dislodge the cartilaginous cap. The pleura is evaluated for perforation and the determination for potential thoracostomy tube placement is made. The wound is closed in layers.

Once harvested, the cartilaginous component of the rib may be carved with a blade to resemble the contours of the head of a condyle (see Fig. 31-12*B*). Care must be taken not to fracture off the costochondral junction during modification and placement into the joint space. The rib is inserted through the inferior incision and its position within the glenoid fossa verified (see Fig. 31-12*C*). The rib can then be secured to the mandible with titanium screws using a lag screw technique or by using a small locking plate, which aids in retention while preventing an inadvertent split of the rib. If concerns remain regarding potential ankylosis development, a biologic spacer can be introduced through the use of a temporalis muscle or temporoparietal fascia flap.[72,73]

For prosthetic joint reconstruction, surgical access to the glenoid fossa and inferior border of the mandible is achieved in a similar manner to the costochondral graft. Current prosthetic reconstructive options fall under two broad categories, temporary-transitional and definitive. Temporary-transitional TMJ reconstruction consists of condylar attachments applied to existing titanium reconstruction plates, used as an interim management protocol for TMJ rehabilitation (Fig. 31-13). Documentation of the temporary nature of this form of reconstruction, patient understanding, and definitive reconstruction plans must be present. Definitive reconstruction uses a TMJ prosthesis that is anticipated to provide the final reconstructive option for TMJ replacement surgery, although by nature this appliance is also temporary in that the functional life of the appliance generally is less than the lifespan of the patient. Definitive TMJ replacement can use a stock condylar prosthesis and variable types of glenoid fossa prostheses, allowing treatment to be altered intraoperatively because of the selection and diversity of implants, or computer-aided diagnosis and computer-aided manufacture (CAD-CAM) prosthesis manufactured from a chromium cobalt alloy is also available, providing a custom-fit prosthesis[68,69] (Fig. 31-14). The advantages and disadvantages of each design are

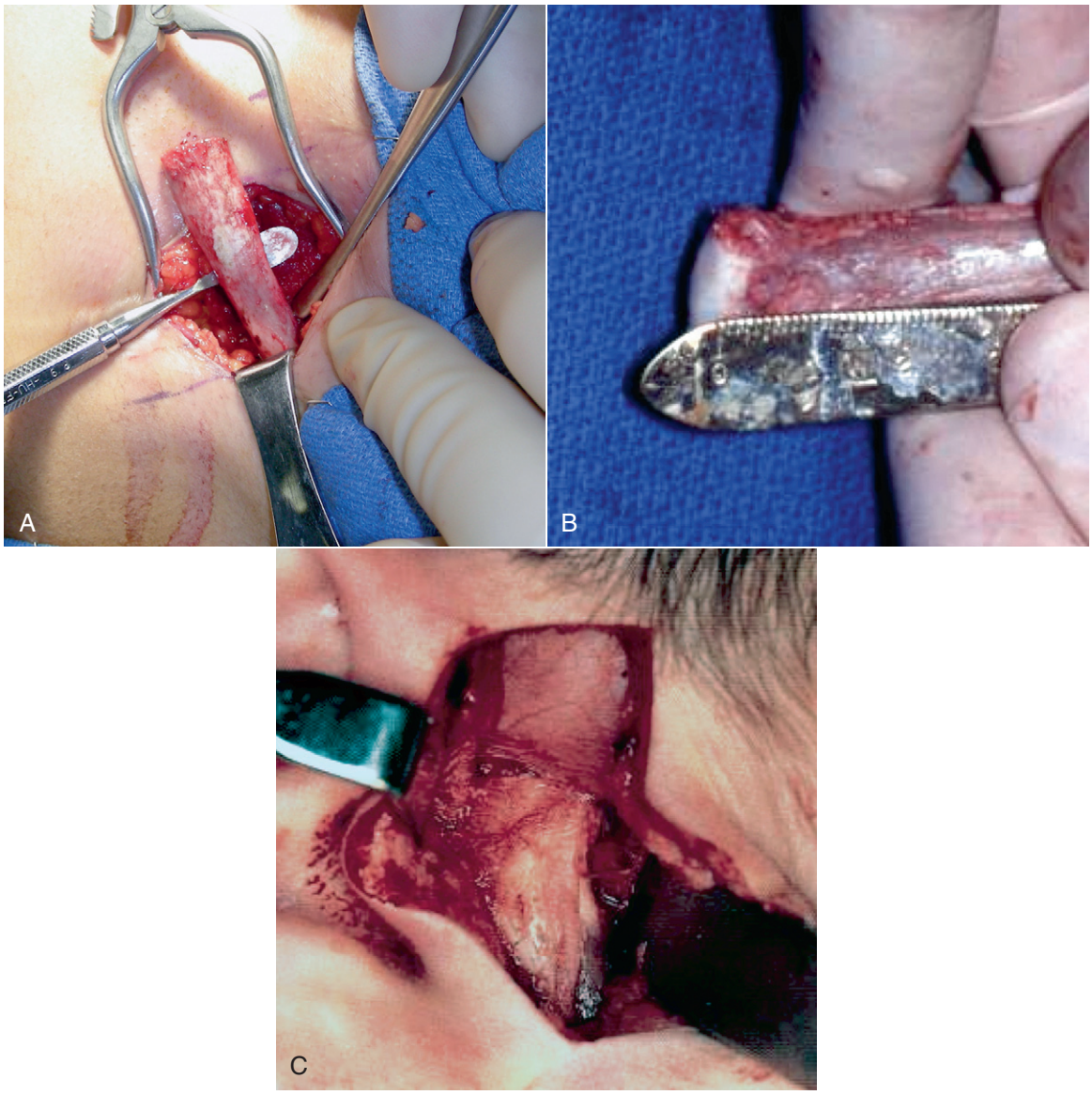


FIGURE 31-12 **A,** Delivery of a rib prior to sectioning with a rib cutter. Note the placement of the incision in the inframammary crease on this female patient. **B,** The rib is contoured with a scalpel blade maintaining several millimeters of cartilaginous cap on the distal extent of the graft, contacting the glenoid fossa to prevent ankylosis. **C,** Verifying positioning of the costochondral graft in the temporomandibular joint. Note the presence of a temporoparietal fascia flap in the superior aspect of the surgical exposure.

determined by patient needs, with the size and variability of the reconstructive options dependent on the system chosen. The stock set will have greater intraoperative flexibility but limited reconstruction size, whereas the custom-designed appliance allows for a more detailed and extensive reconstruction of the TMJ, but without the ability to alter the original treatment plan.

LARGE MANDIBULAR DEFECTS AND/OR THOSE REQUIRING SOFT TISSUE COVERAGE

Osseous defects larger than 5 cm, and those requiring concurrent replacement of soft tissue, necessitate microvascular surgical repair. The choice of the reconstructive option is dependent on patient needs and clinical benefits of each modality (see Table 31-2). Comprehensive presurgical planning is critical to achieve the desired result, with identification of the volume of hard and soft tissue requirements and the available stock achievable from the chosen donor site. The choice of recipient vessels for anastomosis will be determined by the degree of injury at the site and resultant tissue injury, diameter and viability of the native vessels, and length of the vascular pedicle for the graft.

Free Fibula Flap

The free fibula flap has many advantages for use in mandibular reconstruction, including the ability to be easily contoured to re-create mandibular shape, simultaneous transfer of an impressive stock of soft tissue to assist with mucosal reconstruction, and adequate bone stock to be reconstructed with dental implants (Fig. 31-15). The chief drawback to its use is the inability to re-create the normal height and width of the anterior mandible

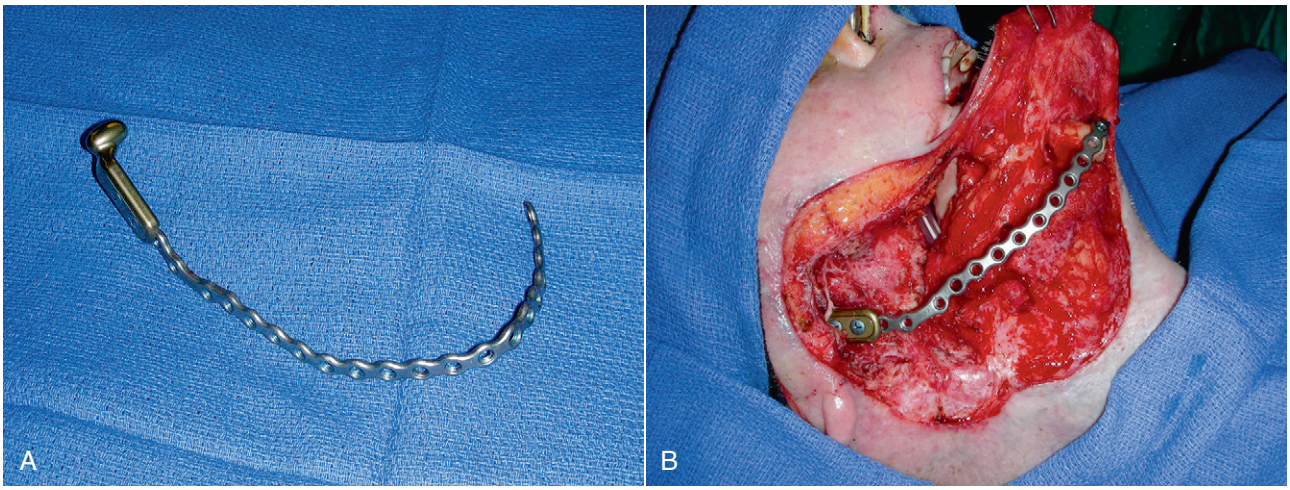


FIGURE 31-13 **A,** Temporary condylar head prosthesis attached to a prebent titanium reconstruction bar. **B,** Temporary condylar prosthesis in position after stabilization of the reconstruction plate. Proper documentation, and patient concurrence, should be present in the medical records regarding the long-term plans for replacement of this temporary reconstruction.

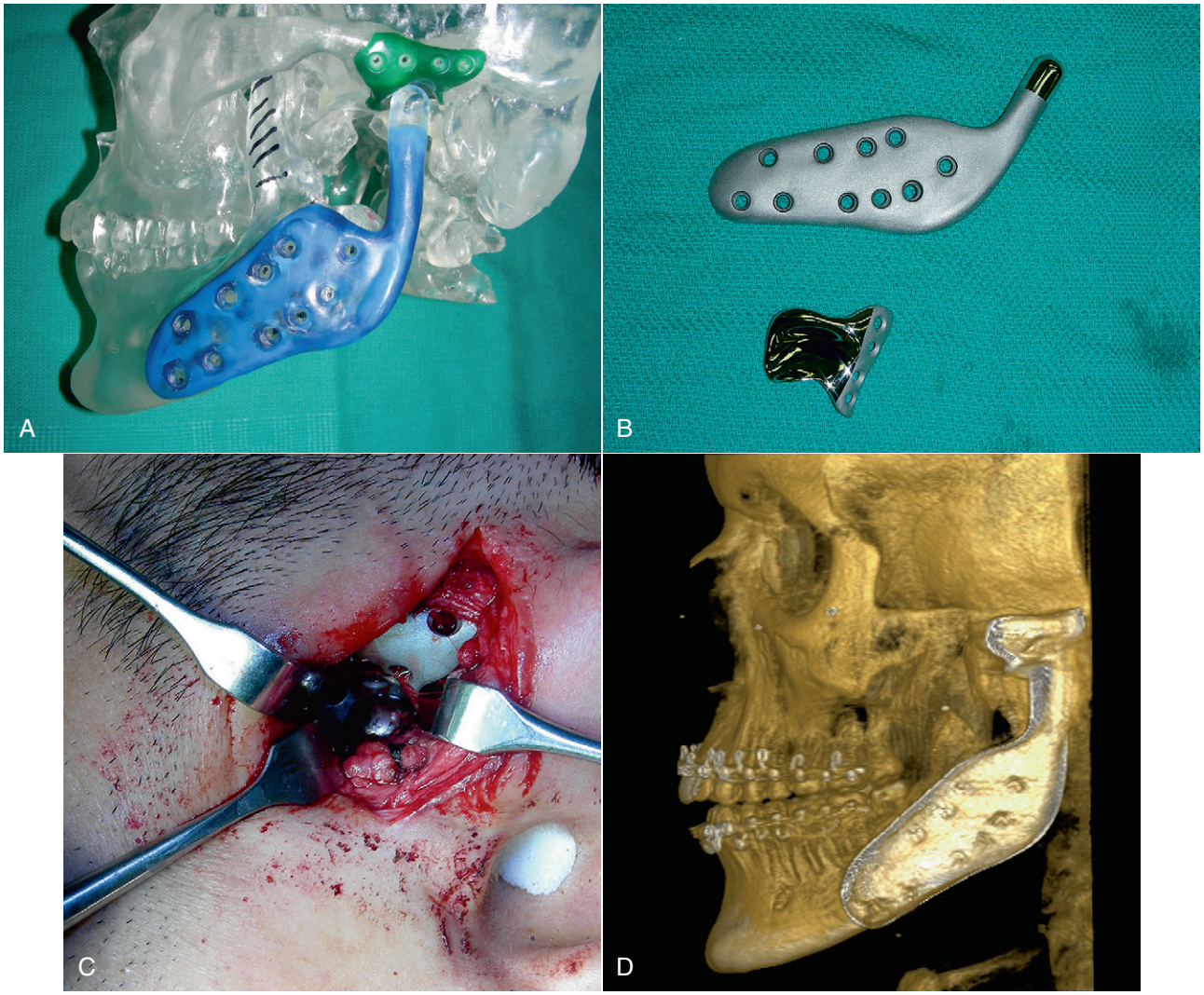


FIGURE 31-14 **A.** Wax-up of the planned definitive reconstruction of the TMJ with a custom-manufactured chromium cobalt alloy manufactured from a CAD-CAM stereolithographic model. **B,** The manufactured custom fossa implant and condylar apparatus after casting. **C,** Intraoperative view indicating proper positioning of the fossa implant and condylar head. This patient suffered a ballistic injury to the left TMJ during military service in Iraq, resulting in complete destruction of the normal joint anatomy. **D**, Postoperative CT scan verifying correct positioning of the appliances.

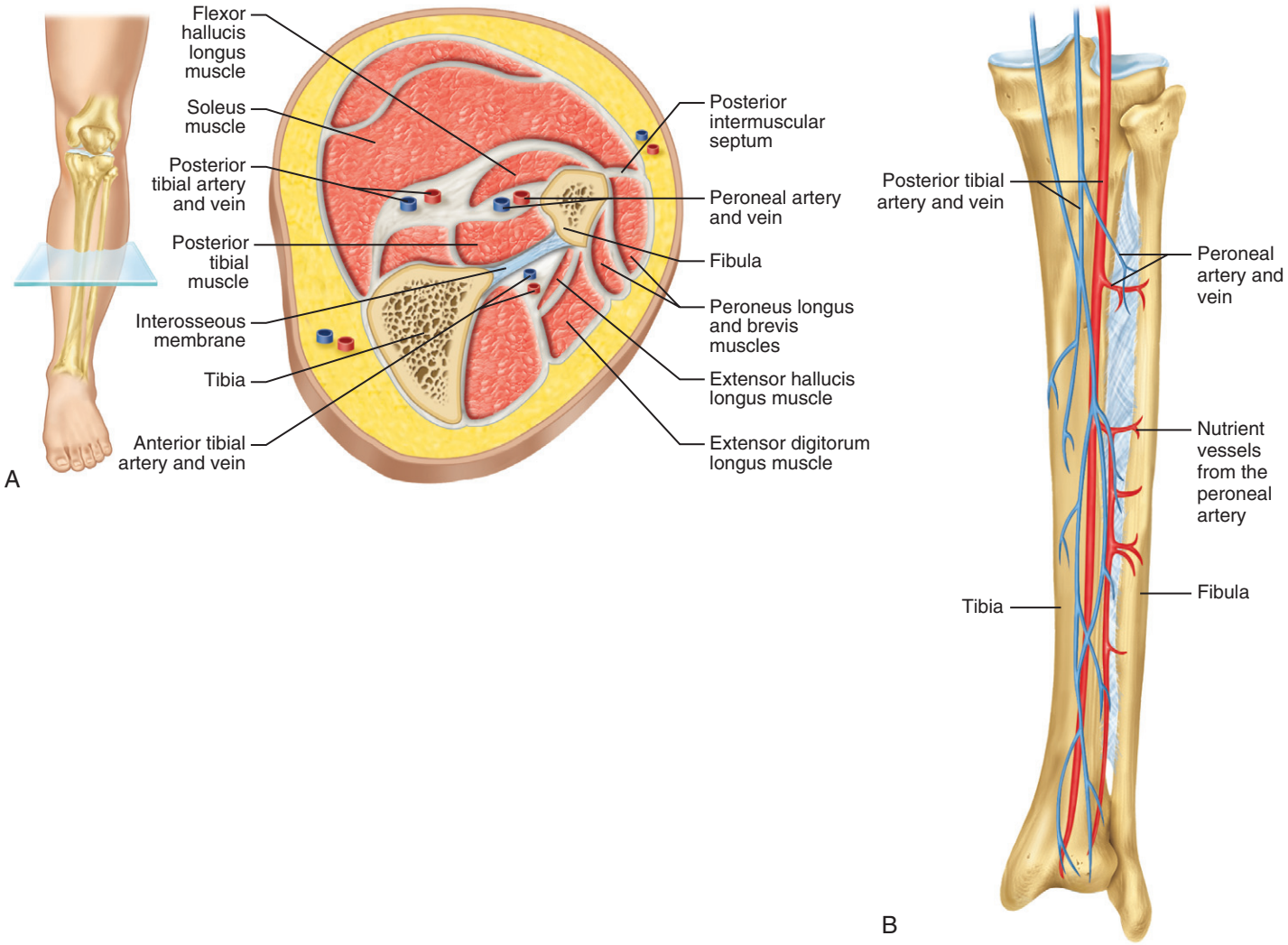
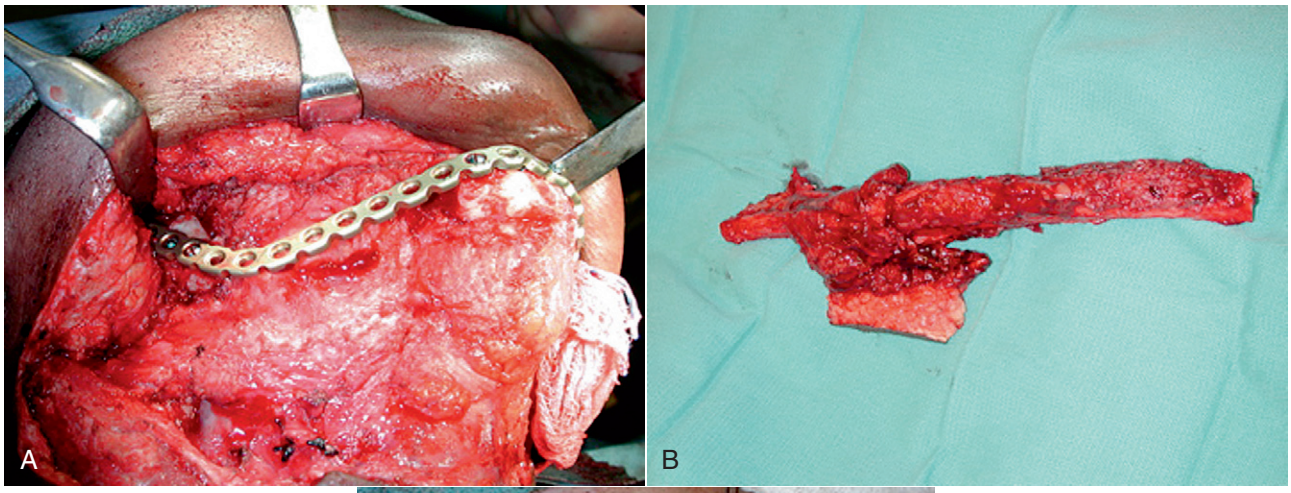
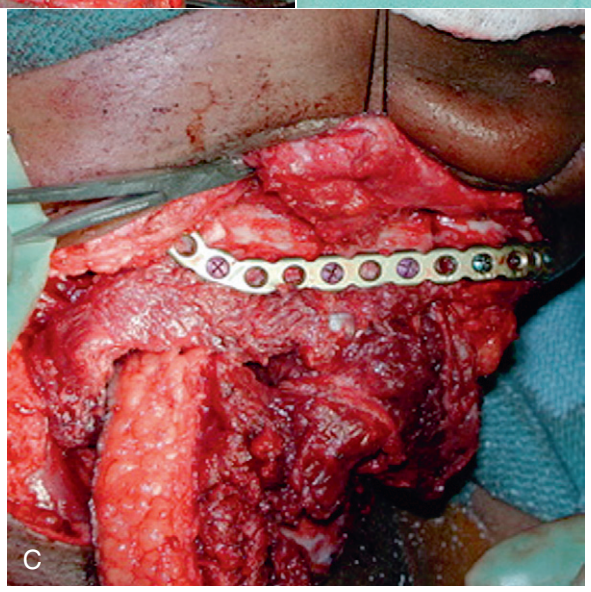


FIGURE 31-15 **A, B,** Anatomy of the fibula for free grafting.

anatomically.[74,75] The fibula ipsilateral to the site of the recipient neck vessels is generally chosen, depending on prior history of lower extremity trauma and vascular supply concerns. The proposed skin paddle is outlined over the fibula to ensure adequate al blood supply.[5]After elevation of the leg and application of an operative tourniquet, the skin is incised anteriorly through the deep muscle fascia and the lateral compartment muscles are removed from the graft, with only a thin cuff of muscle left attached to the bone. The septum between the lateral and anterior compartments is then incised.

The posterior skin is transected and a cleft between the soleus and flexor hallucis muscles is developed, allowing the soleus to be separated. Proximal and distal osteotomies are created and the peroneal vessels and flexor hallucis muscle are identified, exposed, and divided. Traction will expose the tibialis posterior muscle, which is also divided, and the posterior tibial vessels, which are identified and divided. After delivery from the donor site, the graft can be removed and presented to the recipient site surgical team for modification, shaping, and inset to the mandible in standard fashion with titanium plates and screws (Fig. 31-16). The vascular pedicle of the fibula graft is connected to the appropriate recipient vessels, providing necessary blood flow and oxygen transport to maintain vitality (Fig. 31-17). The skin paddle is secured to the appropriate location, providing immediate soft tissue reconstruction. The donor site is closed primarily in layers. Customary monitoring of graft viability is accomplished in an intensive care, monitored setting, with scheduled Doppler and clinical evaluations, with particular attention directed in the immediate postsurgical period for potential occlusion and/or compromise of the vascular anastomosis. Gait disturbances are the feared consequence of fibula grafts, but fortunately the incidence of true ambulatory disability is low, with most patients exhibiting no long-term functional consequence to surgery.[76-78] Concerns regarding inadequacy of appropriate height for mandibular reconstruction with free fibula transfers have been addressed by use of the double-barrel technique.[79] Simply by placing two lengths of the fibula on top of each other, and securing the sites with titanium plates and/or osseointegrated dental implants, a significant increase in form and volume of bony reconstruction can be obtained.





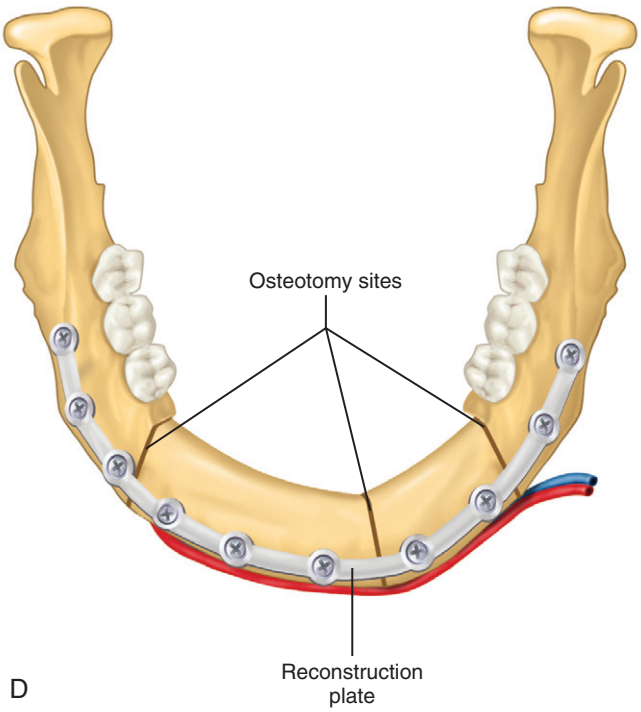


FIGURE 31-16 **A,** The anticipated fibula recipient site replacing the right body of the mandible. **B,** Fibula graft with a vascular pedicle and small monitor skin panel **C,** Fibula graft inset and secured to titanium reconstruction bar. **D,** Graphic representation of the classic handheld osteotomy of the fibula prior to inset into the craniofacial defect.

Iliac Crest Free Flap

The iliac crest free flap is an excellent choice for mandibular reconstruction because it can simulate the mandible in shape, height, and width, providing an impressive bone stock and a generous soft tissue paddle.[80-82] Due to its width and height, the iliac crest flap is an ideal recipient for osseointegrated implants. The anatomic position in the anterior aspect of the pelvis allows for a simultaneous two-team surgical approach (Fig. 31-18). Significant morbidity, including hernia, ileus, hemorrhage, and gait disturbances, are reported consequences of surgical intervention.[5]An incision is made along the upper border of the inguinal ligament and iliac crest, roughly the length of the anticipated bone graft. The deep circumflex iliac artery and vein are then identified, dissected free, isolated, and traced to their origin. The external oblique aponeurosis is dissected free, and the ascending branch of the deep iliac circumflex artery is identified, which is then traced downward, making dissection of the vascular pedicle easier and safer.

The dimensions of the skin, bone, and muscle for harvest are planned and diagrammed on the skin. An elliptical skin paddle may be dissected with subcutaneous tissue and fascia capturing the perforating vessels. This flap is included with a cuff of external oblique and deep abdominal muscles. The iliac crest is identified, periosteum divided, and medial and lateral osteotomies created with a reciprocating or sagittal saw to begin to free up the graft. The iliac muscle is divided and anterior-posterior osteotomies are performed. The composite graft is mobilized and the vascular pedicle dissected free, exposed, and ligated (Fig. 31-19). After delivery of the graft to the recipient site surgical team, meticulous layered closure is performed, with consideration given to placement of a suction device to prevent hematoma formation. Contouring, inset, stabilization, and reestablishment of vascular supply to the graft is accomplished, as described earlier.

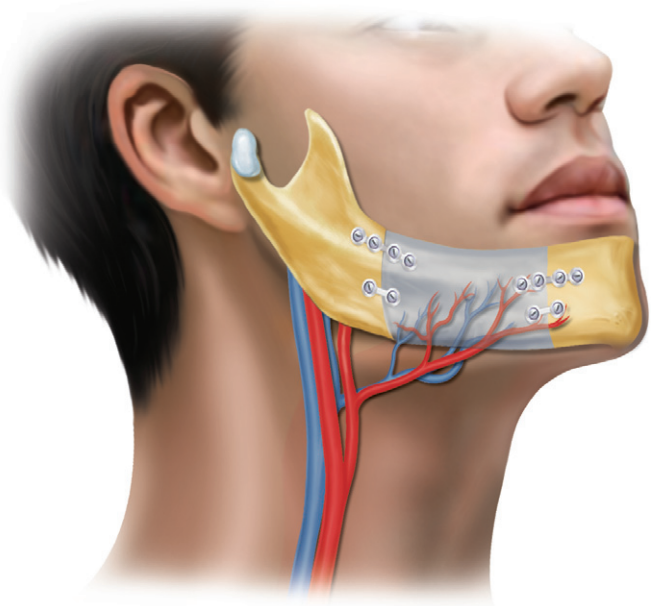


FIGURE 31-17 The graft is inset and secured with titanium miniplates. Subsequently, the arterial supply and venous drainage are anastomosed to the external carotid artery (or facial or superior thyroid arteries) and jugular vein.

Free Radial Forearm Flap

As with several other reconstructive options, the radial forearm flap has the advantage of providing bone and soft tissue for reconstruction and can be harvested by a second team without turning the patient.[83-85] The disadvantages are deficient bone stock in regard to morphology and size, which generally is unsuitable for dental implant rehabilitation, and a smaller soft tissue paddle than other microvascular sources.[5] Although some controversy exists, an Allen's test should be performed to ensure adequate circulation to the hand. In the rare case of an exclusive radial artery vascular supply to the hand, a radial forearm flap would have catastrophic consequences. After application of a tourniquet, an incision is created through the skin, parallel to the radius, and continued distally to the wrist, curving medially. The radial artery and cephalic veins are then identified, dissected

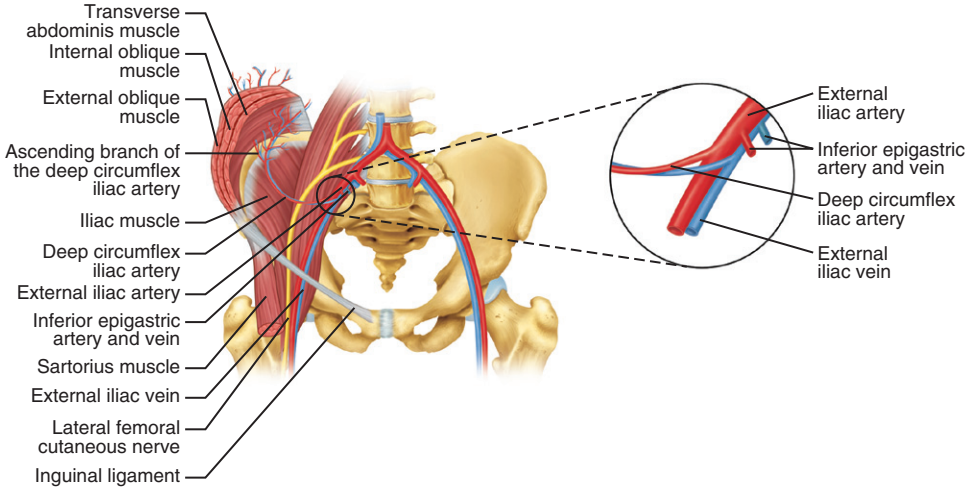


FIGURE 31-18 Anatomy of the ilium for microvascular grafting.

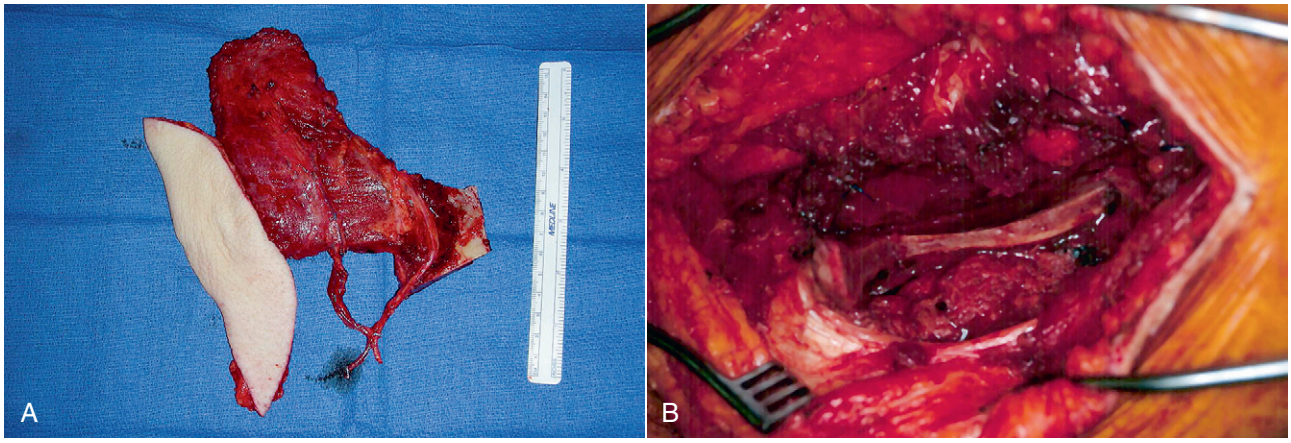


FIGURE 31-19 **A,** Iliac crest free flap. Note that the vascular pedicle, bone, muscle and skin all contained as a composite graft for inset into an avulsive maxillary defect secondary to a self-inflicted gunshot wound. **B,** Iliac crest free flap donor site. Note full-thickness removal of iliac bone incorporated into the flap.

free, and isolated (Fig. 31-20*A*). The radial nerve should be identified, protected, and maintained. An attempt should be made to avoid the lateral and medial antebrachial cutaneous nerves of the forearm. The flexor carpi radialis and brachioradialis muscles are then identified and separated. The radius is identified and care taken to preserve the perforator vessels from the radialis artery through the intramuscular fascia to the bone. Oblique, proximal, and distal osteotomies are first performed in the appropriate length for harvest, and then the bone is sectioned. Swanson et al have recommended a harvest of only 33% or less of the diameter of the radius because greater amounts will compromise the integrity of the wrist and may render the wrist susceptible to fracture during rotation; any level of bone harvest from the radius reduces the breaking strength by 76%[86] (see Fig. 31-20*B* and *C*). A split-thickness skin graft may be used to cover the cutaneous defect caused by the harvest. The wound is closed in layers while the graft is inset, stabilized, and anastomosed. Physical therapy of the wrist may be necessary and some suggest postoperative casting to prevent fracture. Standard postoperative microvascular graft evaluation protocols apply.

Free Scapula Flap

The free scapula graft may provide significant amounts of bone and skin of multiple shapes, but is difficult to contour and generally does not permit placement of osseointegrated implants because of inadequate osseous thickness (Fig. 31-21*A*). Harvesting the graft will require the patient to be positioned in a lateral decubitus position, or essentially prone, preventing simultaneous two-team surgical site preparation.[5,87-90] The head and torso must be protected with padding to minimize the possibility of the development of decubitus ulcers. The flap design is then planned and an incision is created from the posterior border of the deltoid muscle lateral and parallel to the lateral border of the scapula. The cutaneous flap is created by incision and careful dissection, maintaining the subcutaneous vascular plexus of the scapular and parascapular skin. The thoracodorsal fascia should also be maintained as the dissection is continued deep to the medial and lateral border of the scapula. The circumflex scapular artery and veins are identified, dissected free, and isolated. Finally, the osseous flap is designed and osteotomies created so that scapular and parascapular bone harvest may be performed. The patient is repositioned allowing access to the recipient site, and contouring, inset, stabilization, and re-establishment of vascular supply to the graft is accomplished as previously described (see Fig. 31-21*B* and *C*). The harvest site is closed in layers and standard postoperative microvascular graft evaluation protocols will apply.

MIDFACIAL AND UPPER FACIAL THIRD RECONSTRUCTION

Undoubtedly, middle and upper facial third reconstruction is of critical importance in regard to the management of avulsive craniomaxillofacial injuries. Serving as the focal point of facial anatomy and characteristics, accurate reconstruction of this region of the craniomaxillofacial unit is necessary to allow the patient to reintegrate into society as seamlessly as possible. Slight deviation from anatomic norms can result in the patient having an unacceptable clinical result and potential long-term psychological disorders.[91-93] Reconstruction of maxillofacial avulsive defects for the midfacial and upper facial thirds can be categorized into injuries that require simultaneous skin or mucosal replacement and those that do not.

Defects Not Requiring Soft Tissue Replacement

Extensive access to the upper and middle third of the craniomaxillofacial complex can be obtained with relatively minor surgical incisions. While it is beyond the scope of this chapter to detail the intricacies behind each of these techniques, the approaches are covered elsewhere in this text, or within other excellent photographic and graphic sources.[94] The incisions that are most versatile in providing access to defects of the midfacial and upper face are as follows:[4]

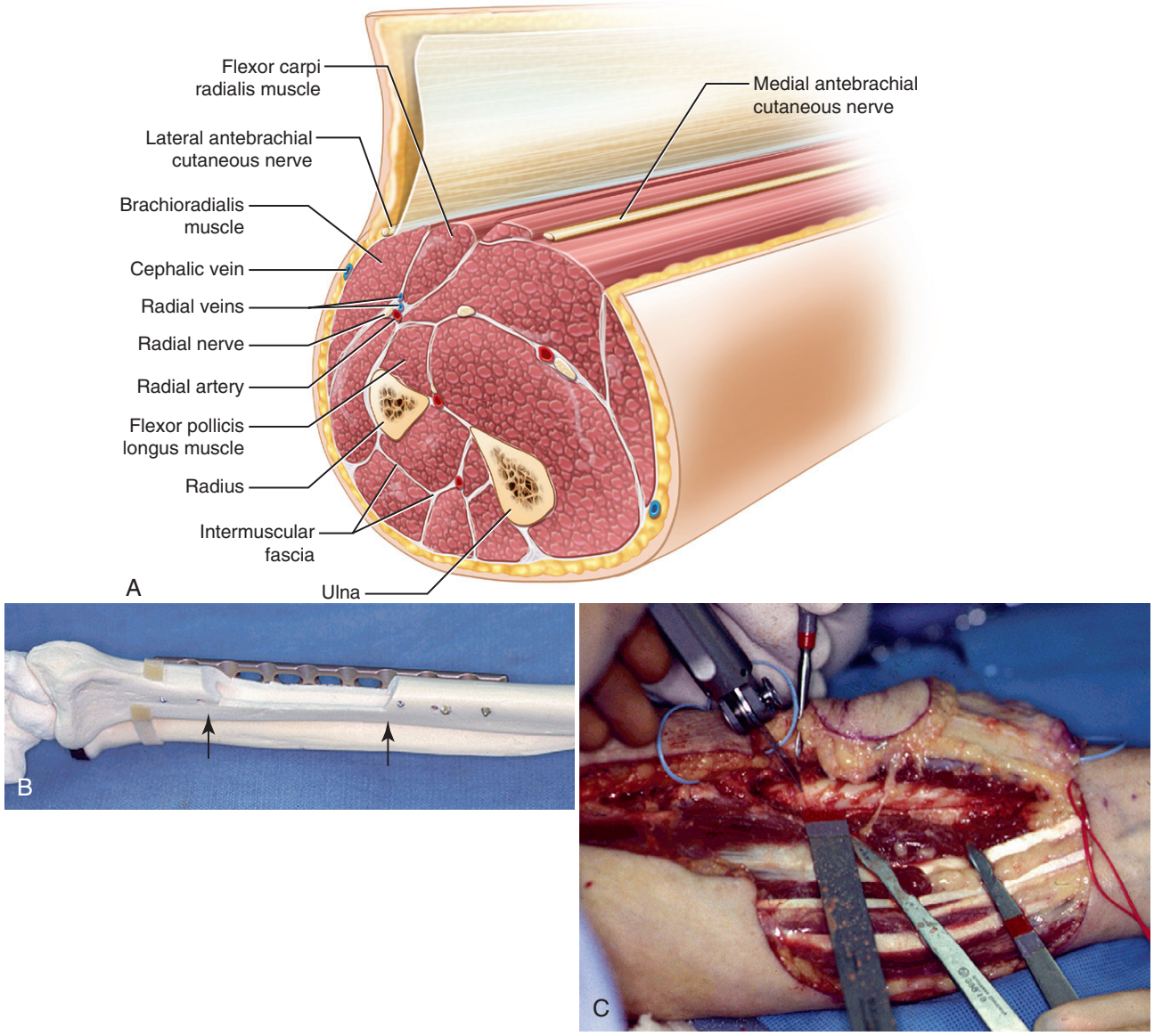


FIGURE 31-20 **A.** Anatomy of the radius for microvascular reconstruction. **B,** Model representation of stabilization of the radius with titanium reconstruction bar at the completion of graft harvest. **C,** Radius osteocutaneous flap procedure with sagittal saw performing osteotomy. (From Schmelzeisen R, Neukam RW, Hausamen JE: *Atlas der mikrochirurgie im kopf-halsbereich*, Munich, Germany, 1996, Carl Hanser Verlag.)

1. Coronal: Extended inferiorly to the zygomatic arch
2. Transconjunctival: Transcaruncular extension
3. Circumvestibular: Nasal extension, intercartilaginous incisions
   - Complete transfixion incision: Midfacial degloving approach—will require columellar sutures; nasal strut graft at closure necessary to support the nasal tip
4. Extension of existing facial lacerations

Properly locating and performing these incisions will provide dramatic access to the facial skeleton, accomplished with minimal to no visible scarring. With the calvarium visualized via the coronal incision and the avulsive defect exposed, in the absence of a requirement for soft tissue transfer, cranial bone is an excellent choice for reconstructive surgical procedures to the midface and upper facial third.[95-97] Identification of the coronal and sagittal sutures and then the temporal line are critical first steps in the harvest; these landmarks are necessary references to avoid potentially lethal complications with obtaining the graft. A large volume of bone can safely be harvested from the entire parietal area, at least 1.0 to 1.5 cm lateral to the sagittal suture, mitigating exposure of the sagittal sinus and possible exsanguination, and superior to the temporal line, avoiding the thin squamous region and potential intracranial violation. A sagittal saw or fissure burr is used to outline the particular shape of the graft desired. An

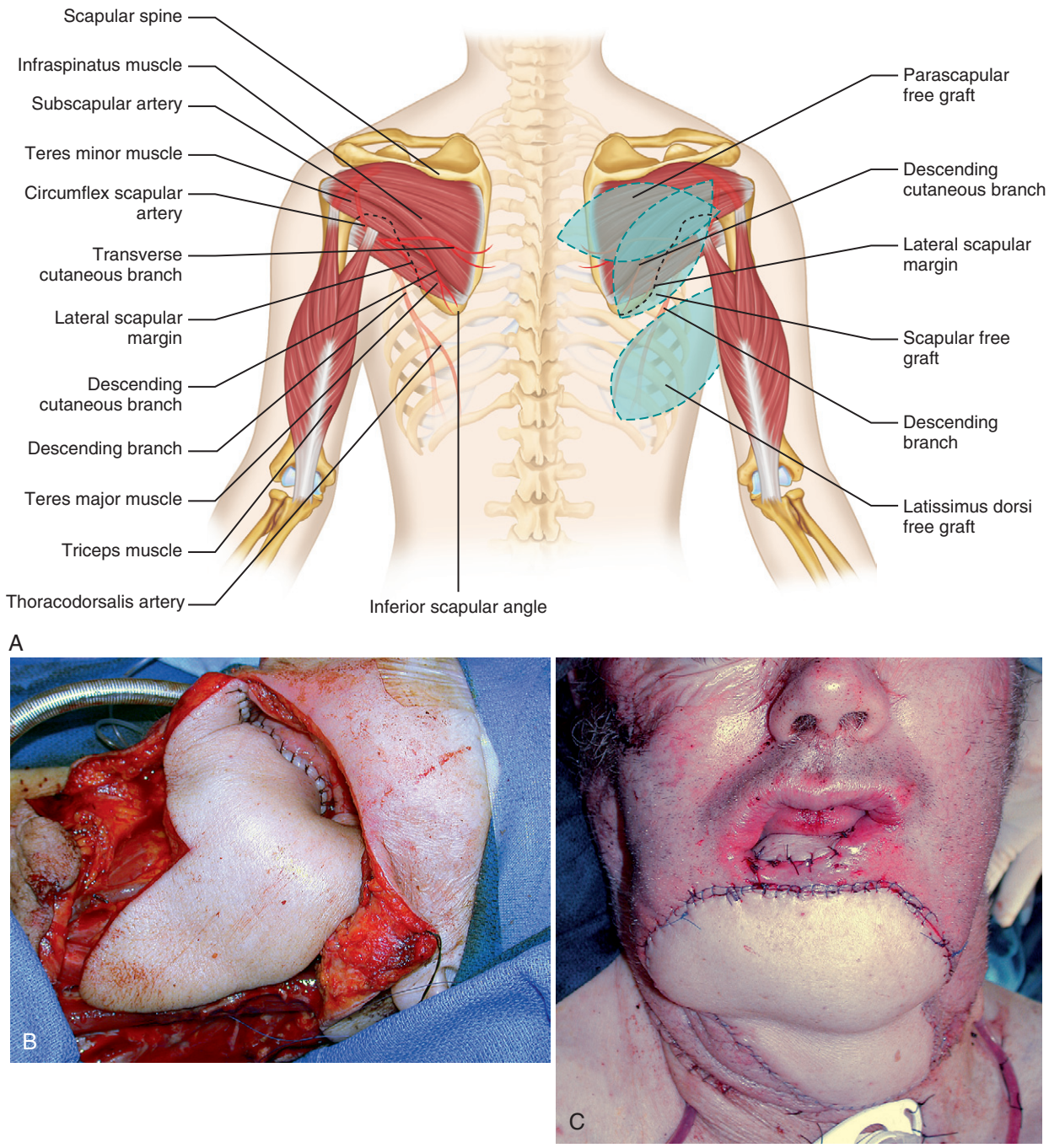


FIGURE 31-21 **A,** Anatomy of the scapula for microvascular reconstruction. **B,** Scapula inset into anterior mandibular soft or hard tissue defect **C,** Completed free scapula flap reconstructing the soft or hard tissue deficits of the anterior mandible. Note the presence of the skin paddle reconstructing the floor of the mouth.

osteotomy is created through the outer table only into the diploe, which is easily identified by the presence of bleeding.

A pear-shaped acrylic burr is then used to create a bevel for assistance with continuing the osteotomy and elevation of the graft adjacent to the osteotomy site. A pronounced bevel will allow access by an osteotome or oscillating saw at a 90-degree angle to the graft, decreasing the likelihood of directing the osteotome or saw intracranially. Undermining the graft with the sagittal saw on all four sides, followed by use of the osteotome to lever the cortical graft from the cranium, generally yields a structurally intact graft without microfractures. Electrocautery, bone wax, and microfibrillar collagen may be helpful in controlling hemorrhage from the donor site. Use of the outer table as the graft can create irregularities in surface contours of the donor site that are especially visible with male pattern baldness. If additional volume of bone is required, or concerns with donor site surface irregularities persist, coordination with the neurosurgeon to perform a full-thickness calvarial harvest or a split calvarium technique is indicated (Fig. 31-22*A*).

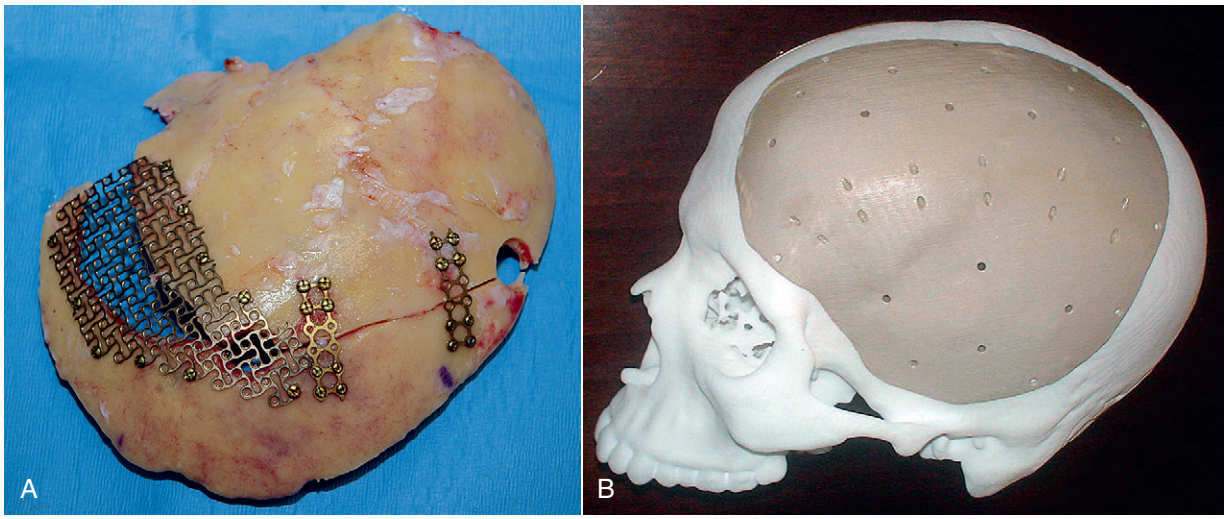


FIGURE 31-22 **A,** For avulsive defects of the maxillofacial region, cranial grafts are useful. This was a full-thickness graft taken by the neurosurgeon to allow for a split calvarial technique in a ballistic trauma case. Note the presence of the titanium mesh reconstructing a portion of the posterior skull and the presence of the burr holes used by the neurosurgeon for access. **B,** PEEK custom calvarial implant. Note that the contours of the implant perfectly reconstruct the defect of the skull.

After the full-thickness calvarial graft is harvested, the inner table is sectioned free for use as the donor graft and the outer table is replaced, maintaining normal cephalic projection. Contourable titanium mesh may be used as an alloplastic bone substitute. This mesh can be adapted to a broad range of contours and form, is biocompatible, and can be used as bone replacement for the midface and upper face. Continued technologic advances in biomaterials have provided a number of bone substitutes projected to replace autogenous grafting—ceramic-based (calcium phosphate), polyetheretherketone (PEEK), polymer-based, and allograft-based substitutes[98-102] (see Fig. 31-22*B*). When addressing small residual defects or voids in the facial skeleton that are not in intimate contact with the dura, paranasal sinuses, or mucosa, acceptable cosmetic results may be achieved with some of these materials, such as calcium phosphate or polymer-based pastes. Foreign body reactions and infections have been reported when calcium phosphate pastes have been placed with direct communication to sinus tissue.

Defects Requiring Soft Tissue Replacement

A significant challenge in the reconstruction of avulsed midfacial and upper facial hard tissue defects is the subset of patients who have additionally lost soft tissue. It is imperative that the neurocranium be isolated from the nasopharyngeal and oronasal compartments, with the obvious microbiologic flora and environmental contaminants present. It is also desirable for the nose and oropharynx to be physically separated. The free fibula, free scapula, and in some cases free radius forearm grafts are acceptable choices for midfacial injury. Each provides soft tissue to substitute for oral and nasal mucosal lining and orbital osseous support. Each can be cabled to provide a palatal, zygomatic, and/or nasal or cranial segment, although the fibula graft has more flexibility in contouring and reconstruction of the various osseous components of the midface.[103] If obliteration of the defect, and not osseous reconstruction is the goal, an ALT flap would be the preferred microvascular surgical approach. Depending on the size and location of the defect, a rotational pectoralis myocutaneous or temporalis flap could be considered.

SPECIAL CONCERNS

COMBINED MANDIBULAR AND MIDFACIAL AND UPPER FACIAL HARD TISSUE RECONSTRUCTION

Considerable amounts of hard and soft tissues may be required if multiple facial units are lost, especially if the adjacent or overlying soft tissue is compromised in quantity or quality.[104] In this situation, multiple segmental free osteocutaneous flaps or sequentially linked free flaps have a role.[5,105,106] When planning treatment and executing the surgical procedure, it is important to keep in focus the individual form for the functional units of the mandible, maxilla, orbit, nose, and cranium.

OSSEOINTEGRATED IMPLANTS

Osseointegrated implants are an integral component in facial reconstruction, including patients with avulsive defects of the craniomaxillofacial complex. In addition to the routine dentoalveolar applications, prosthetic noses, auricles, and orbits can be secured through transcutaneous osseointegrated implants and can complement the reconstruction of the total patient.[107-109] Osteocutaneous flaps, especially free fibula and free ilium, have been found to offer enough bony substance in quality, width, and height to ensure osseointegration.[5,110] Implant fixtures may be placed at the time of the delayed primary reconstruction or during subsequent operations (Fig. 31-23).



FIGURE 31-23 **A,** Complete avulsion of external ear as a consequence of being ejected passenger from an MVA and sliding across the pavement of the highway. **B,** Placement of auricular implants. **C,** Final appearance of patient after completion of prosthetic ear replacement.

CUSTOM-FABRICATED, PATIENT-SPECIFIC IMPLANTS AND ENDOCULTIVATION

With technologic advances, the ability to create CAD-CAM customized, patient-specific implants, which perfectly re-create the missing or avulsed hard tissues, will become more prevalent. Depending on the location, extent, and features of the injury, custom-fabricated implants are now a reality (see Fig. 31-22*B*). Ongoing efforts in the realm of regenerative medicine have succeeded with the endocultivation of hard tissue components from biologic frameworks in Japan and Europe, and will likely see development of patient-specific, soft tissue regenerative processes in the future, as indicated by the recent completion of the first total mandible replacement with a patient-specific custom titanium implant.[111-113]

COMPUTERIZED SURGICAL STENTS

Preoperative planning with computer-based technology allows for the fabrication of acrylic resin surgical stents for complex reconstructive craniomaxillofacial microvascular surgical cases. Via CT analysis of native donor and recipient sites, virtual surgery can take place with extremely accurate placement of the osseous components in the correct anatomic position. By transferring these data to a CAD-CAM–generated acrylic resin stent, highly accurate surgical guides can be brought to the operating room with the desired surgical angles for osteotomies programmed into their manufacture. The result is a faster, more accurate shaping and inset of the microvascular transfer, decreasing ischemia time and potentially improving patient outcomes (Fig. 31-24).

COMPOSITE TISSUE ALLOGRAFT TRANSPLANT

Patients with devastating burn wounds or massive disfigurement to the craniomaxillofacial complex due to avulsive trauma, will unfortunately experience suboptimal functional, cosmetic, and physiologic outcomes with conventional surgical reconstruction. The likely result will be a multitude of psychological and emotional issues, only complicating the already arduous recovery from injury.[2,91-93] For this unique subset of patients, consideration should be given to their suitability for composite tissue facial transplantation.[37-40] A comprehensive physical and psychological evaluation will be a necessary component in the presurgical period at the transplantation center and the patient will need to be fully cognizant of the risks associated with this intervention. Although this patient pool is extremely limited, composite facial transplant surgery for a select few may provide the only avenue for attaining a quality of life previously thought to be lost forever (Fig. 31-25).

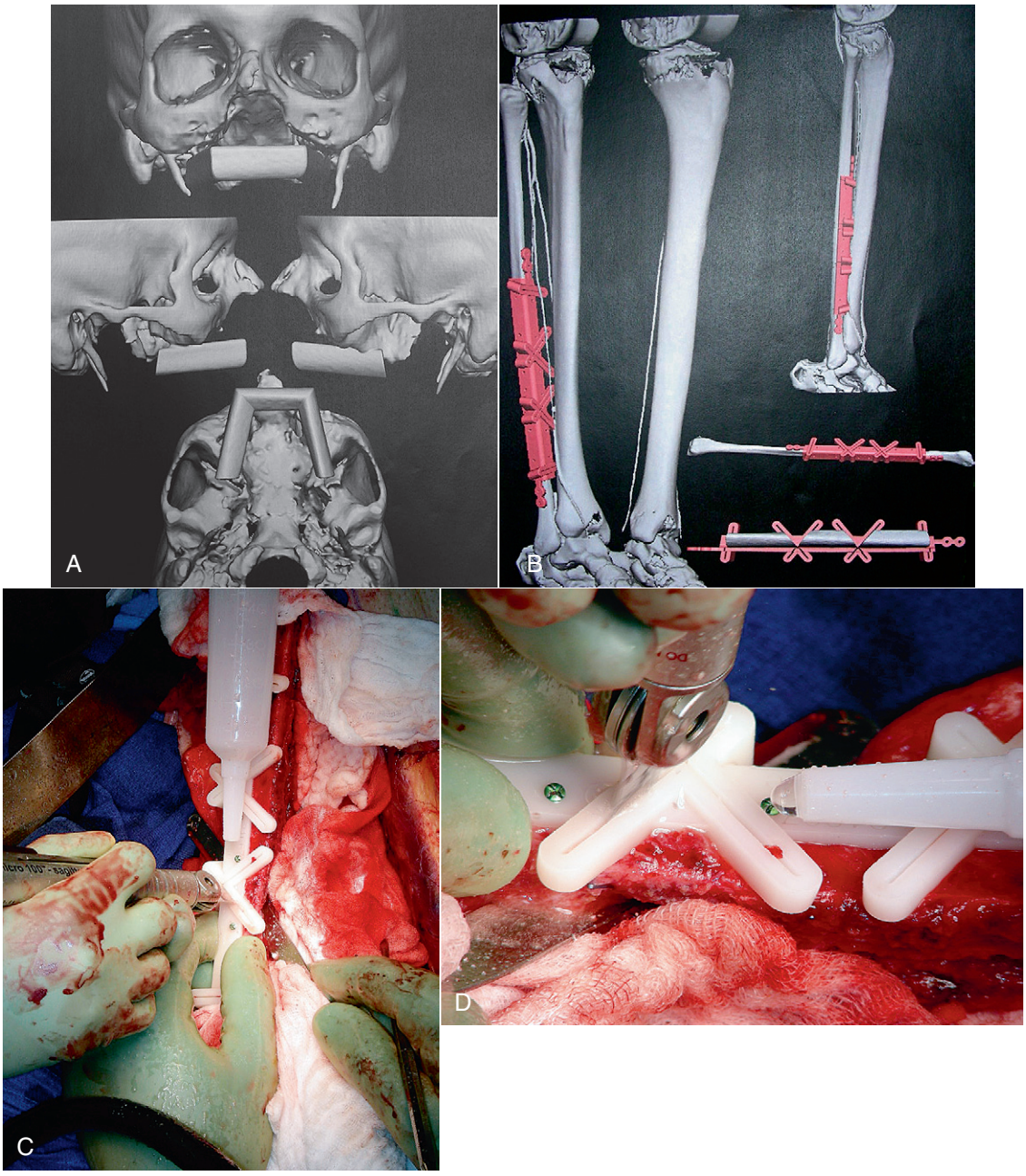


FIGURE 31-24 **A,** Virtual surgery performed on a patient with avulsive loss of her anterior maxilla. She is anticipated for a free fibula graft replacing the missing soft and hard tissue. An exact osseous reconstruction, with ideal osteotomy angles, can be created on the computer program, and subsequently the native fibula can be aligned perfectly with the mandible, assisting with eventual dental rehabilitation and incorporating osseointegrated implants. **B,** Based on the previous information gathered, a surgical guide stent is created in a CAD-CAM format and is positioned on the fibula with precise angle cuts already determined to obtain the planned surgical outcome. **C,** The stent is positioned passively on the fibula. Due to the high degree of accuracy in manufacture, the stent will align on specific anatomic variables of the fibula, ensuring proper positioning. Once the positioning is verified, the stent is stabilized with titanium screws. **D,** Sagittal saw blades are placed through the guide slots and the osteotomies are performed expeditiously.

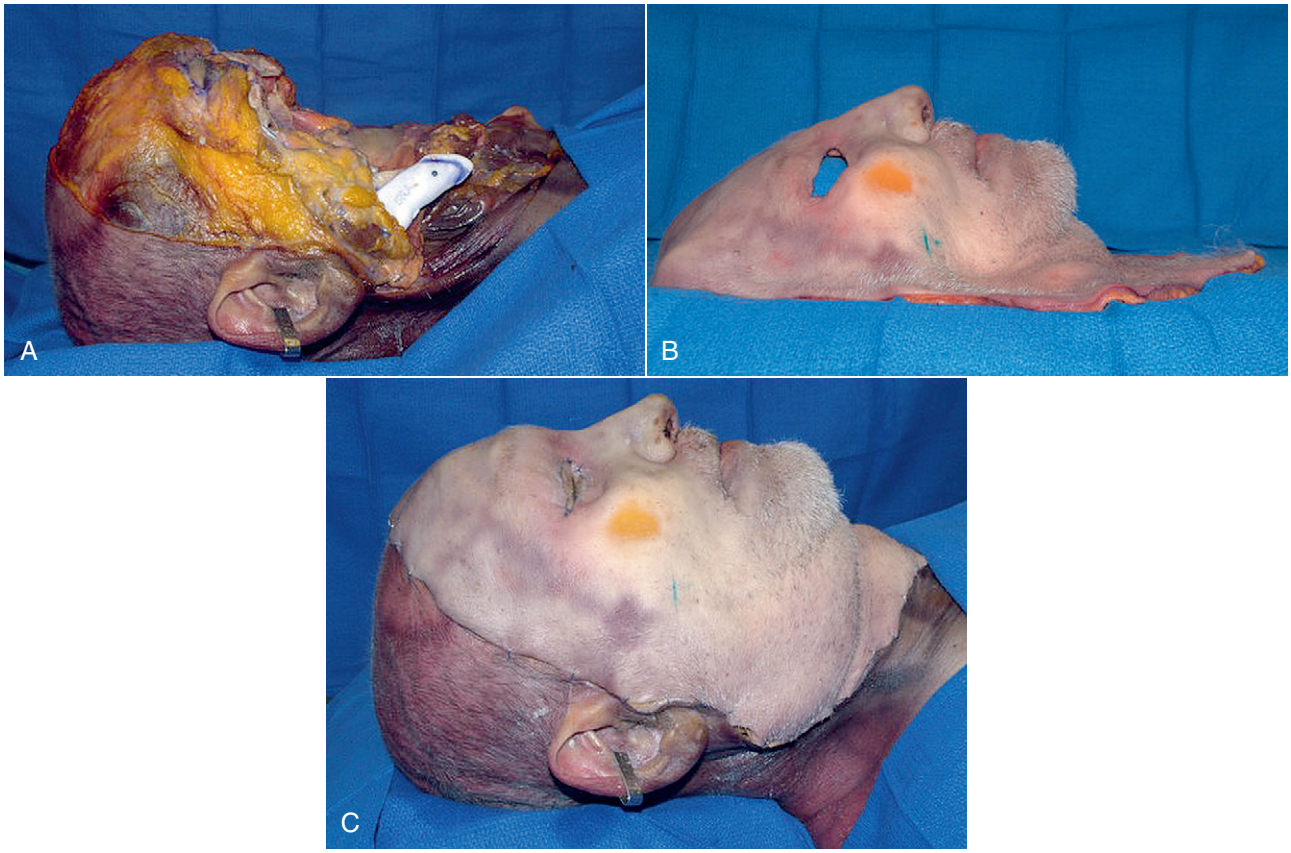


FIGURE 31-25 **A,** Cadaveric specimen with catastrophic middle and lower facial injuries, displaying significant avulsive loss of hard and soft tissue in all regions of the face. Conventional reconstructive techniques would offer this patient a compromised final product in regard to function and aesthetics. **B,** Composite tissue allograft harvested from a donor cadaver, replacing lost bony architecture and soft tissue envelope. **C,** Inset of the facial transplant. Note reconstruction of hard and soft tissues, with adaptation of the graft to the underlying facial skeleton providing superior soft tissue projection and facial aesthetics.

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CHAPTER

32 Infection in the Patient with Maxillofacial Trauma

Stuart E. Lieblich

OUTLINE

Causes of Infection
Local Factors
Systemic Factors
Anergy
Management of Wounds
Preparation of the Patient for Surgery
Prophylactic Antibiotics
Early Detection of Infection
Treatment of Wound Infection
Infections Caused by Oral and Maxillofacial Trauma
Soft Tissue Lacerations
Animal Bites
Human Bites
Facial Bone Fractures
Antibiotic Therapy
Management of Teeth Associated With Mandibular Fracture
Infections Associated With Fractures
Midfacial Fractures
Nosocomial Infections
Tetanus
Treating the Patient With Viral Infection

Infection following a traumatic injury continues to be a major problem, despite many advances in the management of the trauma patient. Local infections occurring at the site of injury, such as osteomyelitis from a jaw fracture, may result in the loss of teeth and bone structure. The individual may survive the initial injury but may be left with significant residual problems, such as a facial deformity or malocclusion and joint dysfunction.

Systemic infections are likely to occur in the traumatized patient through two avenues. First, bacteria may gain direct entry to the host's systemic circulation through the site of injury, IV line, or urinary catheter. Second, invasive manipulations, such as endotracheal intubation, may bypass previously competent host defense mechanisms, causing pneumonia. Thus, although the maintenance of life is addressed during the initial treatment, early interventions are necessary to prevent potentially disastrous infections. In fact, sepsis is the most frequent cause of death following trauma.[1]

This chapter deals with the causes of infection from traumatic injuries, suggests early interceptive methods that are effective at reducing the potential for infection, and describes the treatment of established infections associated with trauma to the maxillofacial structures.

CAUSES OF INFECTION

LOCAL FACTORS

After sustaining a traumatic injury, the patient has a vastly increased risk of infection. At the local level, the injury may disrupt the skin and mucous membranes, which are the body's initial and perhaps most important defenses against bacterial invasion. Intact skin and mucous membranes provide a mechanical barrier to bacterial invasion. Once bacteria have penetrated this barrier through areas of abrasion, laceration, or avulsion of tissue, nonspecific and specific host defense mechanisms are necessary to control the invasion. If colonization and growth of invading organisms are checked, infection will not develop. This is the usual course of events after an injury involving a break in surface integrity.

It is known that a sufficiently large number of bacteria are necessary to produce an infection. Studies have shown that an initial inoculum must contain at least 10^5 bacteria/g of tissue for a clinical infection to occur.[2,3] In a traumatic injury, however, far fewer microorganisms may cause infection owing to the presence of devitalized tissue and foreign bodies in the wound.

Normally, surface flora are kept to a minimum by skin appendages that secrete various antimicrobial substances. Sweat gland production of lactic acid, amino acids, uric acids, and ammonia is bacteriostatic.[4] Secretory immunoglobulin A (IgA) in the oral mucosa is also an important component of the host defense mechanism for controlling bacterial colonization or overgrowth on the mucosal surfaces. Thus, a break in the skin or mucosal surface will not in itself always provide a large enough inoculum to produce an infection.

Vascularity is also an important local factor in the control of invading organisms. If a wound is compromised by vessel trauma, contusion, or edema, the transport of immunologic host defense products to the site of injury is impaired. The decrease in circulation to the tissue provides a more anaerobic environment, which

may permit the growth of certain pathogenic organisms that are inhibited by normal tissue oxygen tension. Local compromise in vascularity is sometimes caused iatrogenically by the injection of epinephrine-containing solutions. For example, a subinfective inoculum of *Staphylococcus aureus* will occasionally produce an infection if placed into tissues that have been injected with epinephrine-containing solutions.[5]

Foreign bodies within a wound also permit infection to occur with much lower numbers of organisms. Infection-potentiating factors have been identified in soil. These factors are highly charged anionic particles that interfere with leukocyte functions and inactivate antibiotics.[6] Inoculation with only 100 bacteria/g of tissue may cause an infection in the presence of soil. Even the placement of a suture will reduce the number of bacteria necessary to cause infection by a factor of 10,000.[7] Thus, vigorous débridement and irrigation are necessary components of wound management, along with the use of the fewest number of sutures acceptable.

Certain mechanisms of injury are more associated with the presence of foreign bodies within a wound. With a gunshot wound, the missile itself is sterilized on explosion and therefore can be left if imbedded in deeper structures. Tooth or bone fragments may be carried into deeper tissue by the primary or secondary missile and do require removal. Other mechanisms, such as motor vehicle accidents (MVAs), should lead the surgeon to search for broken glass embedded in the wound. Small, puncture-type wounds in these cases may harbor substantial foreign bodies that should be removed during primary wound treatment. These are not often apparent on routine radiographs but may show up on computed tomography (CT) scans (Fig. 32-1).

Nonvital tissue within a wound provides a reservoir and culture medium for invading organisms, allowing proliferation to the extent that a clinical infection will result. Dead space in a closed wound is filled with serous exudate and cellular debris. This area is isolated from the systemic circulation, depriving it of host defense products that enter to phagocytose the bacteria.

Local wound management is therefore critical in the prevention of infection. The goal of initial wound therapy is to reduce the number of bacteria to levels that can be readily managed by the host. Débridement of foreign bodies and devitalized tissue should be accomplished as early as possible. Local anesthetic solutions containing epinephrine should not be used because they may further compromise the local blood supply, significantly increasing the potential for infection.

Irrigation under pressure is recommended to remove foreign bodies and reduce the local concentration of bacteria. Gross et al[8] have shown that wounds contaminated with bacteria and sterilized soil were more likely to be rendered sterile if irrigation was accomplished with jet lavage instead of a bulb syringe. The use of large volumes of irrigation will not be effective unless the pressure equals or exceeds 8 psi. Mechanical irrigation devices are available, but the amount of pressure necessary can also be achieved using a 50-mL syringe and a 19-gauge needle. Concerns that the use of a high-pressure irrigation in a wound will further inoculate the site by forcing bacteria into the injured tissue are not well founded. Experimental studies have shown that even with high-pressure irrigation, bacteria and foreign bodies will not be forced more deeply into traumatically injured tissue.[9] However, a reduction in particulate material and bacteria within open bone wounds is not improved with the use of high-pressure lavage versus conventional bulb syringes.[10]

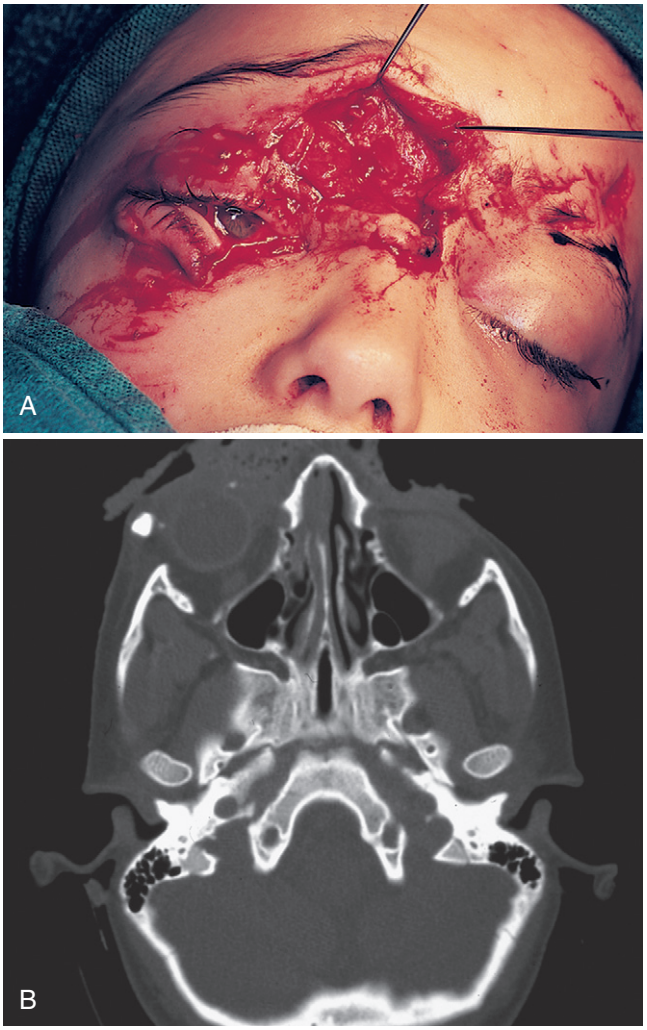


FIGURE 32-1 **A,** Significant laceration of frontonasal region following an MVA. No fractures could be palpated in the wound. **B,** CT scan shows a foreign body fragment, medial surface, near the right globe. The benefit of CT is that it can depict a foreign body that may lead to secondary infection if not removed.

The type of solution used for irrigation is also critical. Further devitalization and tissue damage may occur if toxic solutions are used to irrigate wounds. Studies by Brånemark et al[11] have shown that normal saline solution is the least toxic and best tolerated of the commonly used solutions. Solutions such as hydrogen peroxide, chlorhexidine, povidone-iodine, and hexachlorophene cause direct tissue damage. These agents kill fibroblasts and will further devitalize tissue and predispose the wound to infection. "Don't put in a wound what you wouldn't put in your eye" is a good approach to irrigating solutions for injuries.[12]

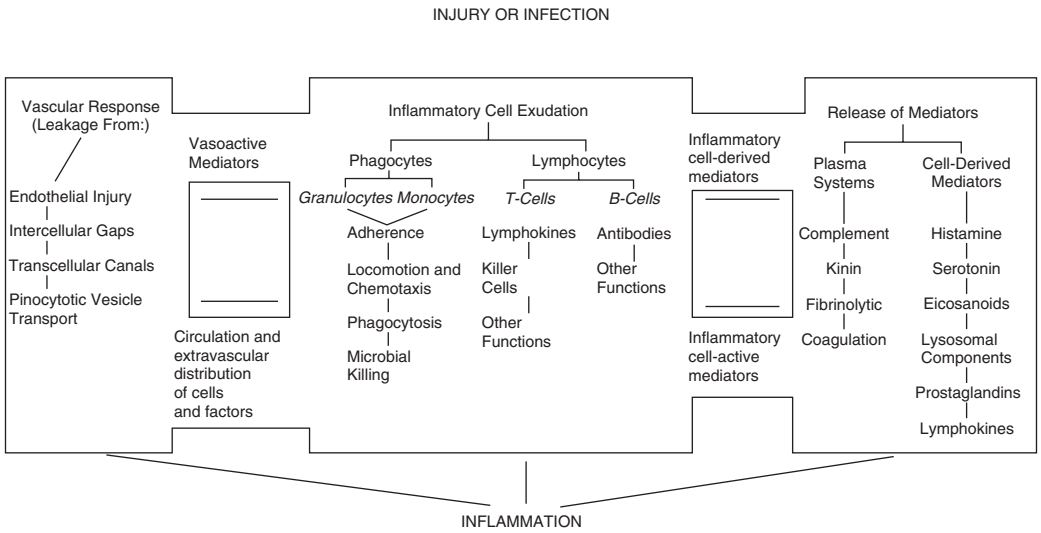


FIGURE 32-2 Interactions among the vascular response, inflammatory cell exudate, and release of mediators following an injury. (From Maderazo EG: Infections and the host. In Topazian RG, Goldberg MH, editors: *Oral and maxillofacial infections*. Philadelphia, 1987, WB Saunders.)

SYSTEMIC FACTORS

Through appropriate wound management, local factors can be controlled and modified by the surgeon to reduce the bacterial load in a wound site. However, it is impossible to sterilize a traumatic wound, and some bacteria will be present in the tissue. The immunocompetence of the host is the factor that usually determines whether a clinical infection will occur.

Once an organism has invaded the host through a break in the mechanical barrier, the next line of defense is the blood-transported phagocytic cells (i.e., neutrophils and monocytes). The disruption of the physical barrier provided by the skin and mucosa allows the direct entrance of surface organisms into the deeper tissue. Phagocytosis is necessary to remove the organisms from the tissue.

Phagocytosis requires a coordinated set of actions for the microorganisms to be removed. Initially, the circulating polymorphonuclear neutrophils (PMNs) must be brought to the site of the bacterial invasion through the bloodstream. When released from the bone marrow, the granulocytes normally have a life span of 4 to 8 hours in the bloodstream and another 4 to 5 days in the tissue. However, in infected tissue, this span is considerably reduced, because as the granulocytes phagocytose and kill the bacteria, they are also destroyed.

Monocytes circulate for a comparable length of time and also function by phagocytosing invading organisms. When they leave the circulation, however, they become established as tissue macrophages and may persist for years in a latent state until recruited by lymphokines secreted by T lymphocytes to phagocytose an organism.

A specific sequence of events is necessary to bring neutrophils out of circulation to an extravascular site where organisms are invading. Initially, neutrophils adhere to the capillary endothelial cells through a process known as margination. This process gives the neutrophils a foothold on entering the extravascular tissue. Margination can be disrupted (e.g., by injection of epinephrine or by a traumatic injury), which causes a transitory leukocytosis as the neutrophils are displaced from the endothelial cells back into the circulation.

Once the neutrophils have adhered to the endothelial cells, they can be mobilized to the source of infection by following the chemical gradient of chemotactic factors. Some of the known chemotactic factors include various bacterial toxins, the degenerative products of inflamed tissue, and certain reaction products of the complement system and blood clotting systems from the site of injury. The interactions among inflammatory cell exudates, the vascular response to injury, and the release of immunologic mediators are shown in Figure 32-2.

Once the neutrophils are mobilized to the site of the infection, additional reactions are necessary for bacterial killing. First, phagocytosis of the organisms must occur. This phagocytosis is enhanced by the local presence of opsonins, which coat the organism, allowing it to be more readily phagocytosed. Opsonins include the C3b and C5b fragments of the complement system. The phagocytosis of opsonized particles is facilitated in the presence of tuftsin. Tuftsin-releasing enzyme is produced in the spleen and therefore is deficient in splenectomy patients, which increases their risk of infection.[13]

Phagocytosis is also augmented by the presence of the antibody specific to the offending organism. When the antibody fragment attaches to the cell wall of the bacteria, it will activate the complement cascade. As noted, this will improve the host response by the elaboration of chemotactic factors to attract more PMNs and macrophages to the site. Monocytes and neutrophils also have receptors for the Fc fragment of the antibody, which significantly facilitates engulfment of the organism.

As bacterial lysis occurs, released degradation products attract more leukocytes and monocytes to the site of infection. Thus, the host response is augmented and will continue to increase until the infection is cleared.



FIGURE 32-3 **A,** This patient sustained multiple traumatic injuries, including a laceration of his lower lip, which was primarily closed. **B,** Breakdown of the wound occurred on the fifth day after injury. The blood glucose level at that time was 380 mg/dL owing to the systemic response to severe trauma.

Once phagocytosed, the bacteria are killed through two mechanisms in the neutrophil, the oxygen-dependent and oxygen-independent systems. In the oxygen-dependent system, interactions with reduced nicotinamide-adenine dinucleotide phosphate (NADPH) and oxygen create highly reactive and toxic free radicals. This process is also known as the respiratory burst.

The oxygen-independent system involves a lowering of the pH inside a phagocytic vacuole. In the neutrophil, additional bactericidal and bacteriostatic agents are produced, including lysosomes, which digest the bacterial cell wall, lactoferrin, which binds iron, and cationic proteins, which interfere with the metabolism of the organism.

The patient's state of health before the trauma and microbial invasion plays a significant role in determining the host defense responses. Certain disease states are known to compromise the four functions of neutrophils discussed earlier—adherence, locomotion and chemotaxis, phagocytosis, and microbial killing. For example, a patient with poorly controlled diabetes has defects in all the four functions of neutrophils, despite normal antibody formation and complement activity. The defects in PMN activity can be reversed if adequate amounts of insulin are present and a hyperosmolar state is avoided.

Defects in chemotaxis will be present in patients with inherent deficiencies in complement and in disease states that consume complement, such as systemic lupus erythematosus. In addition, an inhibitor to chemotaxis (chemotactic factor inhibitor [CFI]) is found to be present in larger quantities than normal in patients with cirrhosis of the liver, sarcoidosis, or chronic renal disease, thereby increasing their risk of infection after injury.

Patients with certain systemic diseases or deficiencies in the production of the preceding factors are, as expected, more susceptible to infection. As noted, patients who have had a splenectomy are lacking in tuftsin, rendering them especially susceptible to infection by encapsulated organisms, such as pneumococci, which resist phagocytosis by PMNs.

Disorders of phagocytosis and bacterial killing will also increase the chance for infection in these immunocompromised patients. Defects of opsonization can be caused iatrogenically in patients receiving exogenous corticosteroids. The steroid binds to the receptor site on the neutrophil for the antibody fragment of the immunoglobulin, preventing antibody-assisted phagocytosis.

ANERGY

After major trauma, a substantial reduction in immunocompetence may occur. Anergy can often be demonstrated, which shows that a significant loss of the delayed response to infection occurs. Severe trauma also causes overactivation of the T cell suppressor population.[14] In addition to these findings, it is known that the stress of trauma increases the output of endogenous epinephrine and corticosteroids. Epinephrine blocks the secretion of insulin, stimulates the release of glucagon and, along with the steroid, increases gluconeogenesis. This combination of events will lead to an abnormal rise in plasma glucose levels and to a notable increase in the susceptibility to infection. The blood glucose level of the patient seen in Figure 32-3 was notably elevated following injury and the persistent hyperglycemia may have led to immunocompromise, wound breakdown, and subsequent infection.

MANAGEMENT OF WOUNDS

After the immediate needs for life support are addressed following an injury, the patient's wounds should be treated. Lacerations should be covered with sterile moist sponges after hemostasis is achieved. Although the emergency room is not always the appropriate place for definitive wound care because aseptic conditions may be difficult to maintain, the treatment initiated here may prevent infection and may preserve the maximum amount of tissue.

On the basis of the history of the injury and direct examination, the surgeon should first evaluate the mechanism of injury to distinguish between blunt and sharp injuries. Significantly more force is necessary to cause soft tissue injury from blunt trauma than from shear

forces, such as glass shards or a knife.[15] The additional energy absorbed from blunt trauma causes a broader area of tissue contusion, ischemia, and necrosis. Thus, the stellate forehead laceration caused by striking a windshield is far more susceptible to infection than a laceration from a sharp object. The surgeon should consider more aggressive use of débridement and antibiotic prophylaxis in injuries resulting from blunt trauma.

In general, the risk of an infection depends on the following three factors:

- The amount and type of microbial contamination of the wound
- The condition of the wound at the end of the treatment (e.g., the presence of residual necrotic tissue, foreign bodies, and bacterial numbers)
- Host susceptibility[16]

Initial wound management is then directed toward reducing the number of organisms present in the wound. This reduction is carried out by vigorous cleansing, careful débridement of grossly nonvital tissue, and copious irrigation under pressure with normal saline only. Primary wound management must be performed with anesthetic for thoroughness. Local anesthetics, if used, should be administered by a field block to prevent deep wound inoculation of bacteria, which may occur if the anesthetic is injected directly into the wound. Solutions without epinephrine should be used to prevent local tissue ischemia.

A decision should be reached early about whether primary or delayed closure will be performed. In general, only wounds that are treated early and can be adequately decontaminated should be closed primarily. Because of their rich vascular supply, facial wounds may be closed primarily after a greater delay than would be acceptable in other areas of the body. The risk of infection in facial wounds is reduced because the preinjury quantity of bacteria in the facial region is usually much less than in other areas, such as the foot, in which the numbers and types of bacteria result in a much higher infection rate. Therefore, many authors believe that up to 24 hours following injury is an acceptable period in which to attempt primary closure of facial injuries.[17]

Wounds of the face are usually closed primarily. Puncture wounds are preferably left open to heal by secondary intention to reduce the potential for infection caused by the trapping of bacteria within the wound. Secondary healing of puncture wounds may also lead to an aesthetically satisfactory scar, especially on a concave surface, such as the medial canthus and nasolabial fold.[18] If adequate débridement, irrigation, and principles of closure are followed, this primary closure of facial wounds that are deeper or more extensive provides the most aesthetically satisfying result. More detailed coverage of this topic can be found in Chapter 25. In severely contaminated wounds or those in which a significant delay in treatment has occurred, a delayed primary closure technique should be used. In this technique, the wound is thoroughly débrided, irrigated, and packed open with frequent dressing changes. A wet to dry dressing is applied, which involves moistening sterile gauze in contact with the wound bed and overlaying this with layers of dry gauze. This dressing has a wick effect and draws out serous and any other exudate from the wound. Changing the dressing at least twice daily accomplishes two goals:

1. It permits observation of the wound bed to determine whether an infection is developing.
2. The removal of the pack results in débridement of dead cells and exudate that have adhered to the gauze surface.

The wound is repacked at least twice daily and observed for 3 to 5 days. If no signs of infection are present, the wound margins are sharply incised and primarily closed. Wounds treated by delayed primary closure will heal as fast as those closed primarily, because the reparative processes have already been initiated. It has been shown that as long as a clean wound is closed within 4 days following an incision, the wound strength is equivalent to 7 days, regardless of whether primary closure or delayed primary closure was used.[19]

Before the closure of questionably contaminated wounds, a technique can be used to provide a rapid estimate of the number of bacteria present in the wound.[20] This method may then guide the surgeon in determining whether primary or delayed closure will be used. To perform this test, the wound surface is cleansed with isopropyl alcohol to remove surface organisms and a biopsy specimen is taken from the wound. The specimen is homogenized and diluted 1:10 with thioglycolate. With a micropipette, 0.02 mL of the suspension is placed on a glass slide and is confined to an area 15 mm in diameter. The slide is oven-dried for 15 minutes at 75° C (167° F) and then Gram-stained.

Under high power (97×), the entire slide is examined for the presence of organisms. If any are noted, the wound is considered to contain more than 10^5 microbes/g of tissue. As noted, wounds with fewer than this critical number are unlikely to become infected and may be closed primarily. This technique was validated by comparing it with the more time-consuming method of serial dilutions and plating of colonies. The rapid slide technique results correlated with those of the serial method and are available within 1 hour, instead of the 24 to 48 hours necessary for the serial dilution technique.

The method of wound closure will also affect the chance of infection. As discussed, each additional suture allows an infection to occur with a lesser number of bacteria. However, to prevent the formation of a residual hematoma, sutures must be placed in sufficient numbers to close all the dead space. Studies have shown that approximately one third of all wound infections are due to residual hematoma.[21]

Hemostasis should be meticulously achieved but not at the expense of creating areas of nonvital tissue in the wound. Careful ligation of vessels and appropriate use of electrocautery should be used . If a hematoma can be predicted because of the exposure of large areas of medullary bone or the raising of a large flap, drainage of the wound should be established.[22] A closed system, suction-type drain (e.g., Jackson-Pratt) exiting from a separate stab incision is least likely to serve as a conduit for bacterial ingress into the wound. Drains should be removed as soon as possible, usually within 48 hours or earlier if drainage has ceased.

Topical hemostatic agents are occasionally necessary to arrest bleeding from the cut edges of cancellous bone or injured organs and when the precise source of a continuous ooze cannot be localized. Many formulations are available, including gelatin foam (Gelfoam, Upjohn, Kalamazoo, Mich), microfibrillary collagen (Avitene, MedChem, Humacao, Puerto Rico), and oxidized regenerated cellulose (Surgicel, Johnson & Johnson, Arlington, Tex). The use of these agents must be tempered by the knowledge that most have been shown to act like foreign bodies, predisposing the patient to infection when a normally subinfective inoculum of bacteria is present. Oxidized regenerated cellulose is the only hemostatic agent shown to be bactericidal and thus is the preferred agent.[23]

For superficial skin closure, reinforced tape (Steri-Strips) has been shown to be superior to a cutaneous suture in terms of preventing infection.[24] If skin sutures are placed, they should be removed in 3 to 5 days to preclude tissue reaction, the formation of stitch abscesses, and permanent scarring.

The surgeon must avoid crushing and damaging the tissue. Devitalized tissue will result from grasping the wound margins with tissue forceps. Instead, atraumatic skin hooks should be used and placed from within the wound to elevate the margins for suturing.

PREPARATION OF THE PATIENT FOR SURGERY

Factors influencing infection in the trauma patient who is scheduled for surgery are as follows:

- Length of the preoperative period of hospitalization
- Use of razors to shave the operative site
- Nature of preparation of the operative site
- Maintaining normothermia
- Oxygen therapy
- Associated resuscitative procedures (allogeneic blood transfusions)

Keeping the preoperative stay short is a factor known to reduce the likelihood of infection by diminishing the period during which colonization with resistant hospital-acquired bacteria may occur. In the traumatized patient, this is accomplished by early operative intervention rather than admitting the patient for a few days before surgery. For example, if there will be a delay in scheduling the operation for an open reduction, the patient could be considered for discharge and then readmitted on the day of surgery. Traumatically injured patients are unlikely to have an infection within 48 to 72 hours of hospitalization, but the rate increases for longer stays.[25] Having surgery within 24 hours of admission was shown to reduce the chance of infection when compared with longer time intervals.[26]

The presence of hair in or around the operative site must be considered by the surgeon. It is well documented that preoperative shaving will notably increase the rate of infection owing to damage to the epidermal barrier and introduction of skin flora into the planned operative site. The preferred method of hair removal is by clipping or depilatory cream. If shaving is necessary, it should be performed only at the start of the case.

The physical preparation of the surgical patient is also important in controlling the possibility of infection. Although antibacterial agents are most often used in surgical preparation, studies have not shown a decrease in the rate of infection when compared with the rate of infection seen when a simple soap and water scrub is used. This finding is consistent with the fact that it is the mechanical aspect of the surgical scrub that reduces the local factors of infection (e.g., number of bacteria, presence of dirt), and that this mechanical factor is of more value than the antibacterial agent. In an open wound, iodophors and chlorhexidine solutions are contraindicated because they may cause tissue devitalization. A nonionic surfactant (e.g., decylpolyglucose [Sea-Clens, Sween, North Mankato, Minn]) is recommended for cleansing open wounds. This agent will not devitalize tissue and has been shown to be nontoxic, even when injected intravenously. Using the surfactant on a sterile sponge, the wound can be thoroughly scrubbed to remove debris and reduce the amount of bacterial flora. Edlich et al have recommended using this agent exclusively on traumatic wounds.[15]

Oxygen delivery is critical to the tissue to reduce the potential for infection. The role of oxygen in improving wound healing is being critically reviewed in the literature. Studies have shown that the prediction of infection of a surgical site can be correlated to the local oxygen tension of the wound.[27] The administration of supplemental oxygen is therefore postulated to reduce the risk of infection. Studies by Grief[28] have demonstrated a reduction in wound infections by the administration of 80% oxygen for the period of surgery and via face mask 2 hours following the completion of the procedure. This easy method of oxygen delivery would indicate a substantial benefit for the patient and consideration for earlier administration to the trauma patient should be considered, particularly if surgery is being deferred.

Because the goal is to deliver this increased oxygen to the tissue, it is the increase in the subcutaneous oxygen tension (Pso_2) that is critical to improve bacterial killing by white blood cells. To deliver more oxygen locally, the local tissue perfusion must be optimized. Factors affecting local tissue perfusion include maintenance of normothermia. Because many trauma patients may have prolonged exposure to the elements at the scene of the injury, the surgeon should expect a notable decrease in core temperature on arrival in the emergency department. Peripheral vasoconstriction will reduce local tissue blood flow, reducing the oxygen supply to the injured site. The trauma patient's core temperature needs to be monitored and consideration for warming blanket placement is usually indicated. Exposure of the patient during the primary, secondary, and tertiary surveys for injuries further reduces the core temperature. Mild hypothermia triples the risk of infection created by the reduction in oxygen supply.[29] Finally, even mild hypothermia will increase intraoperative blood loss and the possible necessity for blood transfusions (see later), which are also associated with an increased infection risk.[30]

Further reducing blood flow to injured tissue is the presence of hypovolemia. Tissue hypoxia is directly related to hypovolemia, so fluid deficits should be

corrected. Although not studied in trauma patients, the use of supplemental crystalloids (an increase of 1.1 liters on the first day versus the control group) increased the Pso_2 in surgical patients.[31] Optimizing tissue perfusion and oxygen delivery involves the administration of greater amounts of crystalloid than what normally would be indicated by observing blood pressure and urine output alone. Fluid supplementation of an initial bolus of 10 mL/kg followed by 16 to 18 mL/kg/hr (continuing for 1 hour postoperatively) notably increases Pso_2 in surgical patients.[32]

One seeming paradoxical issue in the improvement of tissue oxygen supply for reducing infection is that the transfusion of allogeneic blood actually increases the risk of infection. The increase in infection is proportional to the number of units transfused and is postulated to be related to immunosuppression.[33] This, in conjunction with hemorrhage, creates an additive immunodepressive effect.[34] Of note is that this immunosuppression can be shown to be present for several years with reduced lymphocyte function, natural killer cell cytotoxicity scores, and helper and/or suppressor cell ratios.[35]

PROPHYLACTIC ANTIBIOTICS

The administration of prophylactic antibiotics is often indicated in the injured patient. Using the most common classifications of wounds—clean, clean-contaminated, contaminated, and dirty or infected—traumatic wounds fall into the last two categories. Because of this, the risk of infection is much higher in these two types of wounds than in clean operative incisions and antibiotics prove valuable when administered prophylactically. Traumatic wounds are already inoculated by bacteria, so many authors consider the administration of antibiotics adjunctive in this situation as opposed to a true prophylactic or therapeutic indication.

To obtain the maximal protective benefit and reduce the rate of infection, prophylactic antibiotics must be administered within 3 hours after the inoculation of bacteria.[36] When the administration of the antibiotic is delayed beyond 3 hours, the bacteria are able to invade and multiply in the wound. The products of hemostasis (e.g., fibrin) further isolate the bacteria from the host defenses, allowing enough bacterial growth to cause an invasive infection.

Thus, the decision whether to administer prophylactic antibiotics to the trauma patient must be made as soon as the patient comes to the emergency department. The American College of Surgeons recommends the administration of antibiotics to the traumatized patient in the situations listed in Box 32-1.

The seriousness of the consequences of an infection must be considered when determining if an antibiotic should be administered. If the consequence is trivial, such as the occurrence of a dry socket, no antibiotics are necessary. However, infection of a joint or bone from an open fracture may lead to permanent disability and, in these situations, antibiotics are indicated. They are also indicated with jaw fractures because teeth and bone may be lost if infection occurs. Maxillofacial wounds communicating with the oral cavity are associated with a higher risk of contamination because saliva contains 10^8 to 10^9 bacteria/mL.[37] These wounds also warrant antibiotic administration.

BOX 32-1 Indications for Administration of Prophylactic Antibiotics to Trauma Patients

- When the wound enters a joint space or when it is associated with an open fracture
- When there is heavy contamination
- When adequate débridement is not appropriate
- When débridement is delayed
- In burns
- In injuries prone to clostridial infections

(From Altheimer WA et al: *Manual on control of infections in the surgical patient*, Philadelphia, 1984, JB Lippincott.)

The surgeon must determine the appropriateness of thoroughly débriding and decontaminating the wound. If the injury involves essential structures—such as nerves, glands, or major amounts of facial tissue—the surgeon may be reluctant to débride the area extensively. If nonviable tissue, foreign bodies, and dead space are left behind, the risk of infection is vastly increased despite the use of antibiotics. A delay in débridement and decontamination also permits the wound to fill with serous exudate and cellular debris and an invasive infection may be initiated.

Other factors that affect the decision regarding whether to use antibiotics may be present. Correlations exist between the length of surgery and risk of infection. As a general guideline, if the surgery is expected to take more than 4 hours, prophylactic antibiotics should be administered. As noted earlier, certain preexisting medical conditions that potentiate the risk of infection may be present. Local wound healing and host defenses are often compromised in morbidly obese or malnourished patients and antibiotics are often given in these situations.[38]

In trauma patients, prophylactic antibiotics should be administered by the IV route. Obtaining high serum levels of the antibiotic is mandatory and additional delays because of absorption from the oral route must be avoided. The IV route is also the most predictable method of achieving and maintaining blood levels.

The amount of antibiotic administered must be altered from standard dosing based on the amount of fluid resuscitation the patient has undergone. Units of blood, crystalloid, and other fluids can greatly increase the volume of distribution (V_d) available for the antibiotic to be diluted, thus potentially causing a subtherapeutic serum level.[39] The amount of cephalosporin necessary to prevent infection in an experimental animal model with vascular shock was found to be increased seven times.[40]

The frequency of dosing is related to the antibiotic's half-life ($t_{1/2}$). Repeat doses should be given at an interval of twice the $t_{1/2}$ of the drug.[41] A drug such as penicillin has a $t_{1/2}$ of 0.5 hour and would be repeated at 60- to 90-minute intervals for prophylaxis.

Once the operative procedure is completed, with the wounds débrided and closed, there is little indication for

the continuation of antibiotic therapy. However, when there is the potential for continued salivary leakage into the wound, antibiotics are given for a period of 3 days to allow a seal to form between the wound and oral cavity. An example of such a situation would be an open reduction of a mandibular fracture with a tooth in the line of the fracture. Because of the seriousness of the potential infection (osteomyelitis), antibiotics are continued until there is soft tissue coverage of any exposed bone. There is a tendency to administer antibiotics for an excessive length of time in the severely injured patient. One study[42] found that IV antibiotics were administered for an average of 15 days to noninfected patients. Besides the cost factor, the surgeon must consider possible toxicity and other side effects from prolonged administration.

The choice of antibiotic will be based on the potential type of infection that can occur from the injury. Facial soft tissue wounds are subject to staphylococcal infection, but prospective studies of wounds have not shown a benefit from prophylactic antibiotic administration.[36] Instead, proper wound care principles (débridement, irrigation, hemostasis, and careful closure) will prevent most infections in this region.

The compromised host is an exception to this guideline. Patients known to have a higher incidence of infections, such as those with poorly controlled diabetes or asplenic patients, may require antibiotic coverage. Facial lacerations should be treated with vigorous wound care and a decision should be made regarding the necessity for prophylactic antibiotics. The use of a cephalosporin, such as cefazolin, should be considered for facial lacerations. Penicillin should be used only if there is concern for intraoral bacterial contamination. If an antibiotic is used, it must be administered immediately on presentation because delays reduce its potential effectiveness.

Another group of patients who should have antibiotic coverage are those with prosthetic heart valves and any of the other risk factors for the development of bacterial endocarditis (valvular disease). Cases of prosthetic valve endocarditis (PVE) have been reported following facial wound infections.[43] There are additional complications in the management of these wounds because most of these patients are receiving anticoagulant therapy.

Recommendations for wound management in patients with prosthetic valves include immediate administration of IV antibiotics on entering the emergency department. The regimen used for facial lacerations is cefazolin plus gentamicin.[43] This combination is synergistic against the resistant forms of staphylococci, which are responsible for most cases of PVE. If the patient is allergic to the penicillins, vancomycin is used. Wound management should be carried out in the operating room instead of the emergency department, even if general anesthetic is not indicated. This practice provides a more aseptic environment and reduces the risk of hospital-acquired infection.

The length of time necessary for administration of the antibiotic is determined by the nature of the wound. Because the wound occurred in the community, it is initially considered contaminated but can be rendered clean by appropriate wound care. In this case, antibiotics would be continued for a maximum of 2 days. If adequate débridement is not possible, a 7- to 10-day course would be indicated.

BOX 32-2 Guidelines for Administration of Antibiotics to Trauma Patients

- Administer immediately on arrival at the emergency room
- Administer the antibiotics IV
- Dose at twice the $t_{1/2}$ of the antibiotic
- Discontinue the antibiotic at the end of surgery unless persistent contamination is anticipated (e.g., salivary leakage)
- Choose an antibiotic on the basis of the potential infectious organisms (e.g., penicillin for intraoral trauma versus a cephalosporin for contaminated external wounds)

Once the need for antibiotics has been established, administration should follow the guidelines in Box 32-2.

EARLY DETECTION OF INFECTION

After a traumatic injury, careful and continuous monitoring of the patient is necessary to determine whether infection may be developing. The cardinal signs of rubor, dolor, calor, and tumor may be present. However, early detection of an infection will permit treatment to be more effective than if delayed until a full-blown infection is present.

Local wound changes that indicate a developing infection include redness, increased heat, and localized edema. The differentiation between cellulitis, which is an early sign of bacterial invasion, and inflammation, which is part of the usual process of wound healing, may be difficult.

If the condition is due to the traumatic insult, it will be noted in the first 24 hours following an injury. If bacteria are responsible for the local tissue damage, the inflammatory response will persist and progress beyond the first 24 hours. Local vasodilation of capillaries will cause the area to become erythematous and warm. Chemical mediators of inflammation (e.g., histamine, bradykinin, serotonin, prostaglandins, products of the immune system) will collect in the area. Swelling intensifies, nd the wound may become indurated as the extravascular fluid clots owing to the presence of fibrinogen and other plasma proteins, which in turn block lymphatic drainage of the injured area. This reaction is the host's attempt to contain or wall off the invading bacteria.

The systemic response to infection and injury will include immediate leukocytosis caused by the release of granulocyte-releasing factor from the breakdown of the inflamed tissue. Leukocytes are recruited from the bone marrow and spleen and marginated intravascular leukocytes are mobilized in response to injury. The stress of the traumatic injury is also known to cause a transient leukocytosis through increased secretion of endogenous corticosteroids and epinephrine.

Inflammation will also cause the release of a colony-stimulating factor that increases the rate of production of leukocytes from the bone marrow. This activity will cause an increase in immature forms (band cells) to be noted in a peripheral blood smear and is more diagnostic

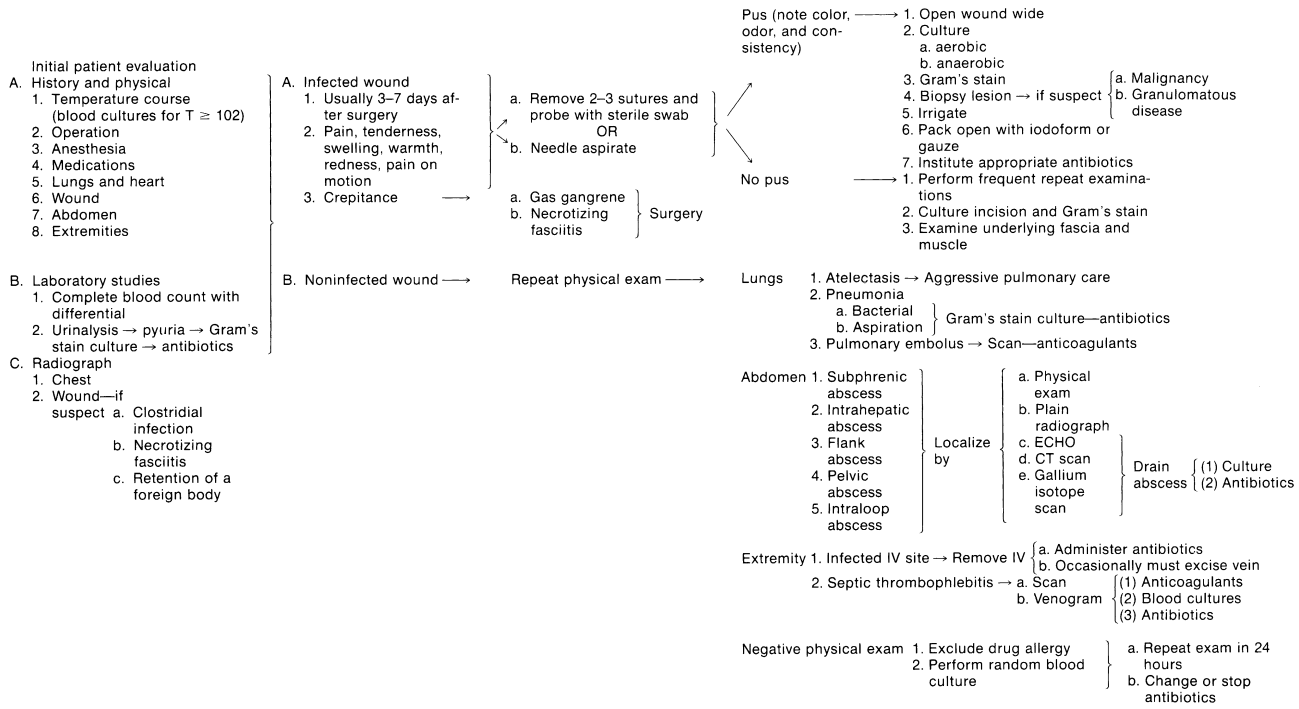


FIGURE 32-4 Steps in evaluating the cause of infection in a trauma patient. (From Majeski JA, Alexander JW: Complications of wound infections. In Greenfield LJ, editor: *Complications in surgery and trauma*, Philadelphia, 1984, JB Lippincott.)

of an infectious process than of a simple increase in the number of neutrophils.

The formation of pus at the suspected site of infection is the result of a continuing host response to the invading organisms. As tissue macrophages and neutrophils continue to phagocytose bacteria, they die and lyse. The local blockage of tissue lymphatics causes a cavity to form in the region; this cavity contains a combination of dead white blood cells and necrotic tissue, which is clinically noted as pus. The formation of pus is an irrefutable local sign that infection has occurred. In the severely anergic patient (anergy may occur even in previously healthy individuals following major trauma), pus may form without the preceding signs of inflammation.[44]

Monitoring the patient's temperature may also confirm the presence of an infection. Body core temperature is regulated by the hypothalamus and normally fluctuates around a range of 1° F (–17° C), with the peak temperature occurring at approximately 6 PM daily. The diurnal increase in endogenous corticosteroids in the morning usually blunts a fever, so measurements should be obtained in the late afternoon or early evening when the corticosteroid levels are lowest. Rectal temperature recordings should be made because they reflect body core temperature more accurately.

Fever, a body temperature higher than normal, is usually caused by exogenous substances (primarily bacteria and endotoxin) and released endogenous proteins known collectively as pyrogens. These pyrogens act on the thermostatic control in the hypothalamus to reset the homeostatic temperature at a higher level, resulting in fever. Endogenous pyrogen is produced from white blood cells, with the major source being neutrophils, monocytes, and eosinophils. Neutrophils are induced to release pyrogen as a result of phagocytosis. This induction is confirmed by the presence of a lag time between phagocytosis and a rise in temperature.[45] Thus, the neutrophils do not contain pyrogen but are stimulated to form it after contact with exogenous pyrogens.

The differential diagnosis of fever in the trauma patient is often complicated by multisystem injury. An algorithm for guiding the workup of an infection in the trauma patient is presented in Figure 32-4. Clinical inspection of the wound is the most important diagnostic tool in determining if the fever is due to wound infection. Local signs, such as increasing inflammation, induration, local pain, and edema, often precede frank drainage and pus formation.

Any drainage from a wound site should always be carefully collected for Gram staining and cultures. If local signs warrant, sutures should be removed and the wound opened to permit further evaluation and allow drainage. To obtain fluid for cultures, the ideal collection technique is to prepare the skin surface with an antiseptic, allow it to dry, and aspirate the fluid into a sterile syringe. Any air in the syringe is expressed and the syringe is capped and immediately transported to the laboratory for aerobic and anaerobic cultures. The laboratory should be alerted that anaerobic cultures are being submitted. Gram staining is also performed at this time to provide immediate evidence of bacterial invasion and some preliminary indication of the type of bacteria present, because the cultures will take at least 24 hours to yield positive results.

Other causes for a rise in the patient's temperature must also be considered. The cause of the fever may be differentiated by the time at which it occurs. For example, after an operation, a fever that develops in the initial

24-hour period is often a result of atelectasis, which is not an established infection, but the collapse of the small airways that entrap bacteria in the lungs. The alveolar macrophages and recruitment of neutrophils initiate the inflammatory response; pyrogens are produced and fever ensues. If left untreated, pneumonia can develop as the bacteria proliferate and invade the collapsed lung segment. Patients with traumatic injuries have a sixfold higher rate of acquiring pneumonia in the intensive care unit than nontrauma surgical patients.[46] The risk is increased if there is chest trauma and frequent sputum cultures are indicated. Early pneumonia is often due to *Haemophilus influenzae*. Individuals with chronic alcoholism also have a twofold increased likelihood of having pneumonia.[47]

Urinary tract infection should be considered in any patient who has had a Foley catheter placed at some time. Urosepsis in these patients is usually from a hospital-acquired organism, necessitating culture and sensitivity testing before treatment.

The development of so-called third-day fever should alert the surgeon to the possibility of an infection caused by an IV catheter. Catheters are responsible for 40% of fevers that develop on the second or third day of hospital admission.[1] As a rule, IV sites should be changed every 48 hours. In addition, many hospitals routinely change all lines inserted in the field or in the emergency room on admission to the floor. These acutely placed lines are associated with a higher rate of infection.

Patients with closed head injuries are also at risk for maxillary sinusitis, which may lead to persistent bacteremia[48] and unexplained fever. These patients have often received corticosteroids along with prolonged nasogastric or nasotracheal intubation. Facial films obtained at the bedside can show opacification; early treatment with lavage will resolve the infection rapidly.

Finally, the surgeon must be aware of noninfectious causes of fever. The most common is a drug fever, which represents a hypersensitivity reaction. Eosinophils, which are involved in allergic reactions, are stimulated to produce endogenous pyrogens and are often found in increased numbers during a drug fever. Eosinophilia will be noted in the differential white blood cell count in cases of drug fever. Cessation of the offending drug (usually an antibiotic) is indicated; the temperature will return to normal in 48 to 72 hours.[49]

Patients with maxillofacial trauma often have sustained blunt head injury, which may cause contusion of the hypothalamus. Typically, the loss of temperature regulation in these patients will be noted by periods of hyperthermia and hypothermia.[45]

TREATMENT OF WOUND INFECTION

Once a wound infection is diagnosed, local treatment is indicated and a decision regarding systemic therapy must be made. Cellulitis is the most common infection in surgical practice. True cellulitis is an invasive nonsuppurative infection. The signs of redness, warmth, and pain are due to the inflammatory response elicited by the invading bacteria. Walling off the inflammation is not seen because of the fibrinolytic agents elaborated by many of the invading organisms, usually beta-hemolytic streptococci.

Cellulitis is managed with antibiotics and warm soaks. Penicillin is the antibiotic of choice for non–hospital-acquired cellulitis.[50] Early therapy for cellulitis increases the chance that the host response can remove the invading organisms. Incision and drainage are indicated only to relieve pressure and only if ischemia is developing.

Once a local abscess or collection of pus develops, surgical management of the wound is indicated. The abscess cavity may be surrounded by a cellulitic area, but incision and drainage of the wound are part of the initial management. Surgical drainage provides many important functions in the management of the infection. By establishing drainage, the number of bacteria present in the tissue is notably decreased. In addition, the local collection of bacterial products, such as endotoxins, is reduced. The fluids that collect in a wound become less active in supporting host phagocytosis and neutrophil killing. Opsonization is also reduced and drainage of a wound will improve these vital neutrophil functions.

Finally, drainage of an infection also provides specimens for Gram staining and for culture and sensitivity testing. Appropriate antibiotic therapy may be instituted while waiting for the culture results. However, antibiotic administration is not a substitute for surgical management of an infected wound, which should not be delayed.

INFECTIONS CAUSED BY ORAL AND MAXILLOFACIAL TRAUMA

SOFT TISSUE LACERATIONS

The large quantities of endogenous organisms contaminating an intraoral tissue injury would apparently predispose many patients to infection. The bacterial count in saliva is high (10^8 to 10^9 bacteria/mL), with anaerobes outnumbering aerobes by about 5:1. In reality, the rate of infection from an intraoral laceration is low. The well-vascularized tissue may confer an advantage on the host's ability to prevent an infection in the presence of a large number of bacteria. Complete débridement of devitalized intraoral tissue can be accomplished with less concern for the aesthetic result than would be necessary for extraoral lacerations.

The routine use of antibiotics for the uncomplicated intraoral wound is unnecessary. Instead, thorough débridement, removal of any foreign bodies, irrigation, and careful closure are indicated. If an antibiotic is indicated, penicillin is the drug of choice because of its effectiveness against oral anaerobes.

Soft tissue lacerations of the face and scalp have also been shown to be relatively resistant to the development of a wound infection. In studies of infection following soft tissue lacerations, the rate of infection of facial lacerations was only 1.3% compared with 12.5% of lacerations of the feet.[51] This difference in infection rate is most likely caused by the lower number of endogenous bacteria found on the face when compared with the feet. It has been shown that all tissues have the same resistance to infection (tongue, fat, muscle, skin), but an infection

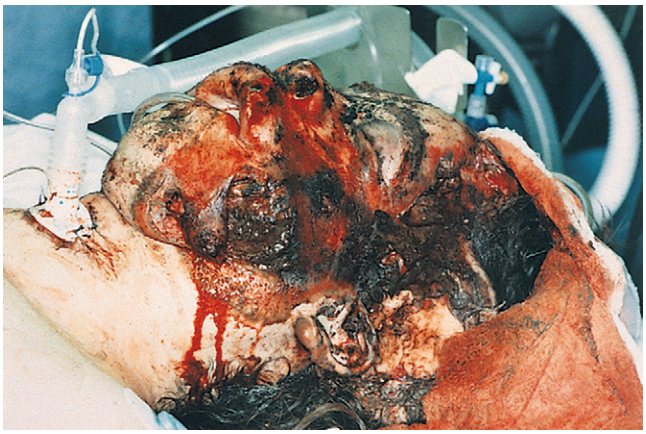


FIGURE 32-5 A patient with significant road abrasions and avulsed tissue incurred after being dragged by a motor vehicle. These road burn injuries can become infected by *Bacillus* spp., which are usually rare pathogens.

uniformly developed in those tissues once a level of more than 10^6 bacteria/g of tissue was reached.[36]

Again the decision regarding whether to use prophylactic antibiotics for lacerations of the face is not clear-cut. No study has shown a decrease in the rate of infection if an antibiotic is given. Instead, the clinician must be guided by some of the factors known to increase the risk of wound infection, as discussed earlier.

Certain mechanisms of injury will direct the clinician toward a specific treatment. For example, road burn injuries (Fig. 32-5) are frequently contaminated with *Bacillus* spp. other than *Bacillus anthracis*. *Bacillus* spp. are saprophytes existing in soil and dust, which previously were thought to be nonpathogenic.[52] They are typically resistant to β-lactam antibiotics. Thus, in heavily contaminated wounds or in those that cannot be completely débrided, antibiotics are indicated. An interesting factor may be the length of the wound. Some studies have noted an increase in infection rate based on the number of sutures necessary to close the wound. Stellate lacerations with abrasion of the wound edges also have an increased rate of infection compared with simple linear lacerations.

It must be emphasized that if antibiotics are to be given, they should be administered by a parenteral route immediately on entry to the emergency room. There is no benefit to closing a laceration and handing the patient a prescription to be filled at some later time if there is a concern about potential wound sepsis. No prophylactic effect is demonstrated by antibiotics administered 3 hours or more after injury.[53]

ANIMAL BITES

Lacerations from animal bites are considered heavily contaminated wounds. Immediate treatment of the wound is indicated, with measures directed at reducing the number of organisms present. High-pressure saline irrigation and thorough débridement are the mainstays of treatment.

Dog bites of the face and scalp are much less likely to become infected than those at other sites. Again, studies have failed to demonstrate a significant benefit to the administration of antibiotics for facial wounds caused by animal bites.[54] Instead, of all the variables controlled for, the lack of débridement was found to cause the highest rate of infection.

The microbiologic features of infections caused by animal bites will differ from those usually noted in traumatic injuries, owing to the different flora of the animal's mouth. A gram-negative rod, *Pasteurella multocida,* is responsible for 30% of infections from dog bites. Its pattern of infection is such that rapid development of local erythema, pain, and swelling occur within 24 hours of injury, enabling the diagnosis to be made on the basis of clinical course. If the infection occurs after 24 hours, it is usually caused by *S. aureus* or *Streptococcus viridans,* and occasionally by *Bacteroides* spp. and fusobacteria, all of which are common intraoral organisms in a healthy animal.[55]

Asplenic patients and other immunologically compromised individuals are also susceptible to infection by a group of aerobic, gram-negative bacilli found in animals. These organisms are identified by the Centers for Disease Control and Prevention (CDC) by alphanumeric names—IIj, EF-4, and DF-2. Infection with these organisms is usually manifested by cellulitis, bacteremia, purulent meningitis, and endocarditis. Thus, because of the serious sequelae of this infection, penicillin prophylaxis is indicated after a dog bite in all asplenic and alcoholic patients (including bites from one's own pet).[56] Penicillin is also the drug of choice for the prevention and treatment of *P. multocida* infections; tetracycline and erythromycin may be used in patients with a known history of an allergic reaction to penicillin.

An additional consideration in the treatment of animal bites is the determination of the necessity for prophylaxis against rabies. The rabies virus causes an acute encephalomyelitis in infected individuals, with almost 100% mortality. Typically, rabies is transmitted through a bite by an infected dog, but cats, horses, cows, skunks, bats, raccoons, and foxes are also associated with its transmission. It is unusual to find rabid rodents, lagomorphs (rabbits), birds, or reptiles.

The infection is transmitted from the saliva of an infected animal to the nervous system of the bitten person by spread from the peripheral nerves to the spinal ganglia and into the central nervous system. This migration of the virus occurs rapidly, with viral particles found in the brain within 72 hours.

Bites from animals that may be infected with rabies must be treated aggressively. Postexposure prophylaxis is given in cases of unprovoked attacks by domestic animals (unless the animal is known to be immunized) and in cases of wild animal bites. Initial wound care is important, consisting of thorough irrigation with quaternary ammonium compounds with 70% alcohol, which have been shown to be rabicidal.[57] Rabies immune globulin and active immunization are also given, along with prophylaxis against tetanus (see later).

HUMAN BITES

Many authors believe that human bites are more serious than animal bites and should be treated differently. However, contemporary management that includes



FIGURE 32-6 **A,** Human bite wound causing avulsion of a portion of the lower lip. The wound was packed open for 4 days and observed, with daily dressing changes. At the time of closure, there were no signs of infection. **B,** Primary closure of the wound. **C,** Its appearance 3 weeks following closure. Excellent cosmetic results were obtained with delayed primary closure. (Courtesy Dr. David Forman.)

débridement, prophylactic antibiotics, and copious irrigation has reduced the infection rate of human bites to the face to about 2.5%.[55] As with animal bites, the anatomic location of the injury plays an important role, with facial injuries having a much lower incidence of infection than extremity wounds.

The aesthetic results of treating a human bite are improved if primary closure can be performed. A delay in treatment or a failure of adequate surgical débridement of the wound, however, may lead to wound breakdown and a compromised aesthetic result. Thus, in the attempt to attain primary closure, extensive surgery may be necessary and should probably be undertaken in the operating room.

The high bacterial count in saliva results in an extensive inoculum in the wounded person. Often, the surgeon may elect to treat these wounds by delayed primary closure. In these cases, the wound is packed with moist gauze, which is changed twice daily. The dressing changes remove the fibrinous exudate that collects in the wound and could support bacterial growth. In addition, the twice-daily dressing changes provide a chance for frequent observation and monitoring for the development of infection. After a 4-day waiting period, primary closure can be carried out, with little risk of infection (Fig. 32-6). This technique of closure is particularly indicated if there is a delay in treatment or in the arrival of the patient in the emergency room.

Antibiotics, if indicated, must be administered immediately on arrival of the patient. Again, no clinical trial clearly shows an advantage to antibiotic administration for bites to the face. In fact, in low-risk human bite wounds, which penetrate only the epidermis and are treated within 24 hours, no decrease in infection was seen with the administration of antibiotics.[58] The oral cavity preponderantly contains anaerobes sensitive to penicillin and this is the drug of choice. Antitetanus therapy is unnecessary, because *Clostridium tetani* has never been shown to be present in the mouth[59] (for an in-depth discussion of bite injuries, see Chapter 27).

FACIAL BONE FRACTURES

Because the morbidity of osteomyelitis is so notable, the appropriate management of facial bone fractures is important. Early management is necessary to prevent infection resulting from the frequency of oral contamination of the fracture site. It is a rare mandibular fracture that is not considered contaminated at the time of presentation. Exceptions would be closed fractures of the subcondylar region. These, along with closed fractures of

TABLE 32-1 Antibiotic Recommendations for Maxillofacial Trauma Patients

Injury	Antibiotic	Pencillin-Allergic	Duration
FACIAL SOFT TISSUE			
<12 hr from injury; cleaned, no foreign bodies remain	None	NA	NA
>12 hr, contaminated; foreign bodies remain; delayed closure	Cefazolin, 1 g q8h (add metronidazole, 500 mg IV q6h, to above if grossly contaminated)	Clindamycin, 900 mg IV q8h (add metronidazole, 500 mg IV q6h, to above if grossly contaminated)	48 hr postclosure, change to PO
>12 hr, contaminated, with intraoral communication	Aq Pen G, 2.0 mU IV q4h (add metronidazole to above if grossly contaminated)	Clindamycin, 900 mg IV q8h (add metronidazole to above if grossly contaminated)	48 hr postclosure, change to PO
MANDIBULAR FRACTURES			
Condyle, closed	None	NA	NA
Open fracture (intraoral wound)	Aq Pen G, 2.0 mU IV q4h	Clindamycin, 900 mg IV q8h	IV until fracture trx, then 48 hr postop
Open fracture + gross contamination	Ampicillin-sulbactam, 1.5 gr IV q6h	Clindamycin, 900 mg IV q8h plus	72 hr post-treatment
± delayed trx		Metronidazole, 500 mg IV q6h	
ZYGOMATIC, MAXILLARY FRACTURE	None	NA	NA
FRONTAL SINUS FRACTURE	Ampicillin-sulbactam, 1.5 g IV q6H	Clindamycin, 900 mg IV q8h	72 hr postoperatively
PANFACIAL FRACTURE (CONTAMINATED)	Ampicillin-sulbactam, 1.5 g IV q6h *or* Piperacillin-tazobactam, 3.375 g IV q6h	Clindamycin, 900 mg IV q8h *plus* Levofloxacin, 750 mg IV or PO qd	72 hr postoperatively

NA, Not applicable.
Adapted from U.S. Army Institute of Surgical Research: Joint theater trauma system clinical practice guideline: Guidelines to prevent infection in combat-related injuries, 2012 (http://www.usaisr.amedd. army.mil/assets/cpgs/Infection_Control_2_Apr_12.pdf).

the zygoma and other facial bones, would not be indicated for antibiotic treatment[60].

If definitive treatment of mandibular fractures is to be delayed, temporary intermaxillary fixation is indicated to prevent mechanical pumping of saliva and bacteria into the fracture site. This can be readily achieved with intermaxillary fixation screws or the placement of bridle-type wire for fractures within dentate segments. Movement of the fracture also causes rebleeding at the fracture site, which increases the local hematoma and causes a more anaerobic environment. The temporary intermaxillary fixation will also make the patient more comfortable.

ANTIBIOTIC THERAPY

All jaw fractures involving tooth sockets must be considered compound fractures requiring antibiotic treatment. More recent studies have looked at the risk of infection of facial fractures and noted that mandibular fractures are the most common types associated with infection. Rarely are infections noted with other facial fractures. Because this decision can be made early, with an examination of the patient, parenteral administration should not be delayed, even if definitive treatment of the fracture is deferred. As noted, delays in the administration of an antibiotic may allow bacteria to become established and isolated from the systemic circulation and to multiply into the critical number necessary to cause an infection of the fracture site.

The surgeon's choice of antibiotic will be guided by many factors, including the following:

- Identification of the causative agent or the usual organism that may cause an infection if prophylaxis is indicated
- Use of the least toxic antibiotic
- The patient's drug history—to avoid known drugs to which the patient has previously reacted adversely
- Use of a bactericidal as opposed to a bacteriostatic drug, because the bactericidal drug relies less on the host's resistance, kills the bacteria directly, and works faster
- Cost of the antibiotic regimen[41]

A guideline for consideration of antibiotic administration is presented in Table 32-1.

MANAGEMENT OF TEETH ASSOCIATED WITH MANDIBULAR FRACTURE

A significant controversy centers on the management of teeth in the line of fracture and their relationship to infection. Studies of complications of mandibular fractures have shown a higher incidence of infections when

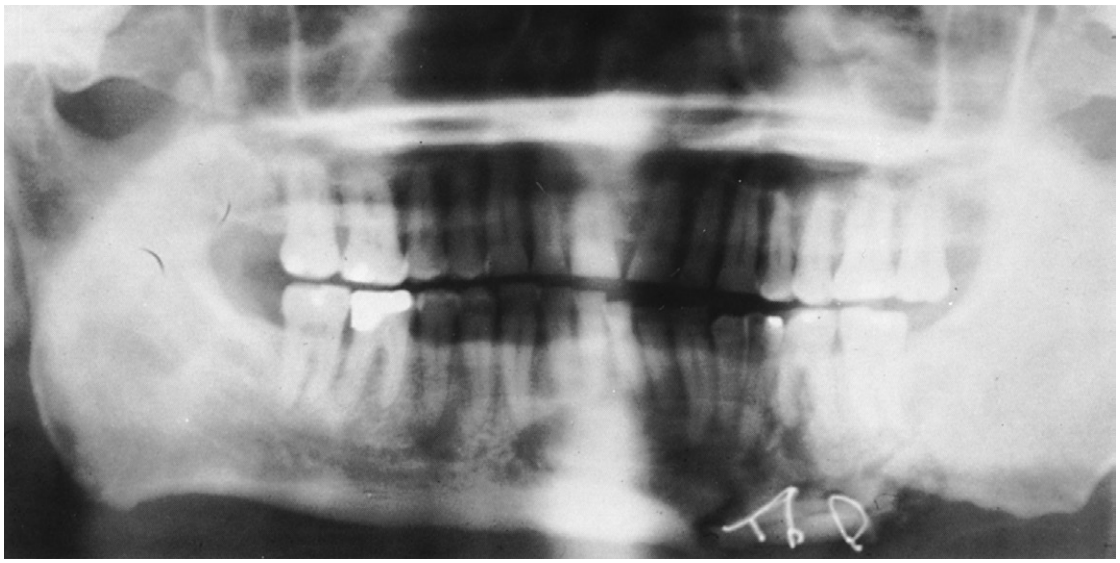


FIGURE 32-7 Osteomyelitis developed after the open reduction of this mandibular fracture. The placement of multiple wires in an area of comminution requires stripping of the periosteum from the small bone fragments and a subsequent devascularization.

teeth are involved in the line of fracture. This finding has led some clinicians to recommend extracting all teeth in the line of fracture. In 1965, Bradley suggested removal of all teeth associated with a fracture.[61] More recently, a retrospective review of 327 mandibular fractures appeared to recommend leaving in place healthy nonmobile teeth in the line of fracture.[62] Contemporary fracture management supports removing teeth in the line of fracture only if the following conditions exist:

- The tooth is loose.
- The tooth is grossly carious or periodontally involved.
- More than 50% of the root is exposed in the fracture line.
- Adequate reduction is mechanically blocked by its retention.

Retention of healthy firm teeth may help in the reduction of a fracture and preserve the dentition.

Zallen and Curry have shown that with the administration of antibiotics, the incidence of infections is significantly reduced if teeth are involved with the fracture.[63] Although their study was not controlled, the incidence of infection was 50% without antibiotics and 6% if they were given. When mandibular fractures are compounded into the oral cavity (i.e., through the dentoalveolar structures), therapeutic antibiotics should be administered immediately. These fractures are contaminated or even infected at the time of presentation. The antibiotic of choice is penicillin. If the fracture is compounded onto the skin, a penicillinase-resistant penicillin should be added or a semisynthetic penicillin with a β-lactamase inhibitor should be used. Clindamycin is used in patients allergic to the penicillins. Alternatively, a cephalosporin, such as cefazolin, can be used, which has been shown to reach high levels in bone. Antibiotic therapy is continued for 3 days in these patients.

Continued mobility of a fracture causes rebleeding and decreased levels of oxygen tension at the fracture site. These local changes can increase the risk of infection. Bacterial contamination of the fracture site should be reduced on fixation of the fracture and some method of temporary fixation should be applied as soon as possible. Open reduction allows débridement of any nonvital tissue and evacuation of hematoma and permits close alignment of the fracture, thus reducing the dead space. However, open reduction may also result in further devascularization of tissue, especially when small bone fragments in a comminuted fracture have been stripped of the periosteal attachment. Considering these factors, the surgeon should exercise good judgment in performing an open reduction when notable communition of the fracture has occurred (Fig. 32-7).

BOX 32-3 Risk Factors for Osteomyelitis

- Ineffective fixation, reduction, and immobilization
- Delay in treatment (old fracture)
- Delay in administration of antibiotic therapy or inadequate antibiotic therapy
- More than 50% of a tooth root exposed in a fracture
- Premature removal of fixation devices
- Devitalized segments of bone in the fracture site because of excessive stripping of periosteum, overzealous use of transosseous wiring, or overheating of the bone by burs
- Decreased host resistance or increased susceptibility to infection (e.g., patient is alcoholic, diabetic)

INFECTIONS ASSOCIATED WITH FRACTURES

Owing to the compound nature of most fractures of the mandible, the risk of osteomyelitis is notable. Factors associated with an increased incidence of infection developing after mandibular fractures are noted in Box 32-3.

Infection of the mandible following a fracture is known as post-traumatic osteomyelitis. After injury and

subsequent reduction of the fractures, certain signs and symptoms indicate that an osteomyelitis is developing. The early signs of an acute suppurative osteomyelitis include the following:

- Deep intense pain
- High intermittent fever
- Paresthesia or anesthesia of the mental nerve (arising after the trauma and reduction of the fracture)
- A clearly defined cause[64]

At this phase, the infection is spreading through the intramedullary portion of the bone, with little cortical destruction; therefore, radiographs will not show any significant findings. Parenteral antibiotics should be administered at this time.

As the osteomyelitis becomes established, a firm cellulitis will develop over the involved portion of the mandible and lead to intraoral or cutaneous sites of drainage, or both. The cellulitis is often firm or brawny hard on palpation. Systemic signs are variable but may include a mild leukocytosis with a shift to more immature (band) forms of PMNs, a rise in temperature, and occasionally an increase in the erythrocyte sedimentation rate (ESR).

Although radiographic signs of osteomyelitis are not evident until late in the process or until 50% demineralization has occurred, they may precede the development of frank drainage. Classic radiographic findings of osteomyelitis include a moth-eaten appearance of the bone and the development of sequestra, which are islands of devitalized bone. The sequestrum is surrounded by an involucrum (a sheath of new bone), separated from it by a radiolucent zone.

The extent of the osteomyelitis will be greater than that noted on routine radiographic examination. To plan the appropriate surgery and ensure adequate treatment, scintigraphy is often a useful adjunctive study. Typically, technetium 99m–labeled phosphate compounds are administered IV and become concentrated in areas of increased bone activity. Because this test cannot differentiate between areas of increased formation and areas of resorption of bone, more information regarding the osteomyelitis can be obtained using a subtraction study. The test is carried out by performing a second scan with gallium, which is known to collect in white blood cells. An area that shows both technetium and gallium uptake probably represents acute or suppurative osteomyelitis. If the site shows an uptake of technetium but not of gallium, it probably represents an area of bone repair. Scintigraphy is also useful for monitoring the course of the disease and efficacy of treatment and may also indicate when treatment can be safely stopped.

Once established, osteomyelitis associated with fracture is treated using a combined surgical and medical approach. Nonvital tissue, foreign bodies, and associated teeth must be removed. At the surgical procedure, specimens of bone should be obtained using meticulous technique and submitted quickly for culture and sensitivity testing, without secondary contamination. This practice must be strictly observed to preserve fastidious anaerobes that may be the causative organism of the osteomyelitis. Débriding infected areas and establishing drainage are important. Continuous irrigation and drainage systems can be placed through a closed wound following appropriate débridement if the infection is extensive or refractory to conventional drainage and irrigation. Antibiotic-containing solutions may be of benefit for irrigation but have not been comprehensively studied in regard to their efficacy. Recent studies of systems that deliver a high concentration of antibiotic locally to a site of osteomyelitis have shown promise. These systems use materials that are impregnated with an antibiotic, usually gentamicin, which is then implanted in the infected wound. The benefits of such a system are that high and sustained concentrations of antibiotics are delivered locally, with low systemic levels, thereby reducing the toxic side effects of some agents.

In addition, a means of fixation must be used to prevent further movement of the fracture segments. This often requires the use of external pins to span the gap created by the removal of the involved bone. Reconstruction of the bony defect usually is not attempted until all signs of infection are gone, generally after at least 2 months. The absence of infection is determined on the basis of a lack of local signs of infection, such as drainage or cellulitis. A bone scan using the technetium and gallium subtraction technique can be carried out (see earlier) to corroborate the clinical impression that the infective process has cleared. The secondary reconstruction of continuity defects resulting from an osteomyelitis is discussed in Chapter 36.

This discussion of osteomyelitis centers on its treatment in the mandible as opposed to the maxilla and other bones of the facial skeleton. The endochondral bone of the mandible is structurally similar to the long bones of the body, which are more susceptible to osteomyelitis. The intramembranous bone of the maxilla has less medullary tissue and thinner cortical plates, which allow the infection to pass through quickly and into the surrounding tissue; hence, it does not have the opportunity to become established as readily as it does in the mandible. The blood supply to the maxilla is more extensive and therefore less susceptible to disturbance from infection.

The use of hyperbaric oxygen for osteomyelitis of the mandible has been reported.[65] Its clear benefit over surgical and medical treatment has not been demonstrated, but it may be of use in cases that are refractory to more conventional means of therapy.

Often, a 6-week course of antibiotics is referred to in the treatment of osteomyelitis. This is based on previous studies that reviewed the management of childhood hematogenous osteomyelitis in contrast to osteomyelitis secondary to a contiguous focus of infection or chronic osteomyelitis. These latter two types depend on combined surgical and antibiotic management, and resolution is expected more quickly. Surgery is necessary because antibiotics will not penetrate necrotic bone.

Certain antibiotics attain a much greater serum concentration in bone than others. In experimental osteomyelitis infections caused by *S. aureus,* clindamycin was found to have a 98% serum concentration in the infected bone. Cefazolin concentrations reached only 6%, whereas cephalothin showed a 3.5% concentration.[66]

MIDFACIAL FRACTURES

Facial bone fractures involving sites other than the mandible are usually involved with one or more of the sinuses. The microbiologic characteristics of an infected sinus differ from those of an infected oral cavity, specifically because of the presence of *Streptococcus pneumoniae* and *H. influenzae*. However, the healthy sinus has been shown to be a relatively sterile environment,[67] and the choice of prophylactic antibiotics should be based on the most likely source of the potentially infective bacteria.

Le Fort II and III fractures may communicate with the cranial cavity, as evidenced by a cerebrospinal fluid (CSF) leak from the nose or external ear canals (CSF otorhinorrhea). Antibiotic prophylaxis to prevent the possibility of a meningeal infection is controversial because studies have not shown a reduction in the incidence of meningitis in patients who have received prophylaxis. In fact, after 5 days of systemic antibiotics, the nasopharynx usually becomes colonized with more resistant organisms,[68] generally acquired in the hospital—making treatment of meningitis more complicated. Therefore, early reduction of the fractures is indicated, which will normally stop the CSF leak. This reduction is indicated as soon as the patient is neurologically stable.

NOSOCOMIAL INFECTIONS

The hospitalized, traumatically injured patient is at risk for having an infection during the period of admission. Nosocomial infections affect a notable number of patients each year, causing delays in discharge and notable morbidity and increased cost of care.

Traumatized patients are at increased risk for nosocomial infection because they are compromised hosts. The mechanical barrier of the skin and mucous membranes is violated by injury or interventions, such as surgery and insertion of IV lines. Host defenses are also reduced owing to some of the systemic effects of the trauma. Of additional concern is the fact that hospital-acquired infections often involve organisms with unusual virulence or resistance to antibiotic therapy.

The diagnosis of a nosocomial infection is similar to that of any type of infection. The source of the fever in the hospitalized patient may require a full systemic workup, including evaluation of the lungs, wounds, IV sites, urine, and blood. Cases of septicemia have been reported to occur because of contaminated IV fluids.[69] Thus, the source of infection may require consideration of many factors.

Owing to the variable resistance patterns of the responsible organisms, antibiotic therapy is withheld until specific culture and sensitivity test results are available, if possible. Other therapeutic interventions are important, such as pulmonary physical therapy for suspected atelectasis or pneumonia. Review of the patient's hospital course, including any IV sites or catheterizations, may further assist in the investigation. In addition, the bedridden traumatized patient is susceptible to thromboembolism and fat embolism, which may cause fever.

TETANUS

Tetanus is caused by the production of an exotoxin from *C. tetani*, an obligate anaerobic, gram-positive rod. The disease has a fatality rate of about 45% in the United States,[70] but active immunization is successful at preventing the disease.

Because viable spores are present in soil, in house dust, and on clothing, even minor injuries may cause the disease in unimmunized individuals. The organism is not invasive but gains entry through puncture wounds and lacerations; the most frequent sites are the hands, feet, and legs.

When evaluating a wound that may be likely to cause tetanus, an immunization history should be obtained. If a full set of three vaccines has been given and a booster received within the previous 10 years, no further therapy is necessary. When passive immunization is to be given, a single dose of human tetanus immune globulin (TIG) is administered. Subsequently, the patient should complete an active immunization series.

Certain wounds are classified as more tetanus-prone than others. Linear wounds, less than 6 hours old with a sharp mechanism of injury (e.g., by a knife or glass), are considered nontetanus- prone and TIG is never indicated.[71] If a previously immunized patient has a tetanus-prone wound and has not been immunized within the past 5 years, a booster dose of tetanus and diphtheria toxoids is administered.

The diagnosis of tetanus is made by clinical signs, because organisms are recovered in only about 30% of cases. The incubation period is 2 to 56 days, with earlier onset associated with a poorer prognosis. The classic signs of trismus (lockjaw), rigidity of the facial musculature (risus sardonicus), and reflex spasms are pathognomonic for the disease. The patient is managed with antiserum (TIG), muscle relaxants, tracheostomy, and antibiotics (penicillin).

TREATING THE PATIENT WITH VIRAL INFECTION

Maxillofacial trauma includes a high incidence of interpersonal violence, much of which may be related to obtaining or selling illegal drugs. Because these individuals are often IV drug abusers themselves, the incidence of human immunodeficiency virus (HIV) infection is much higher than in the general population. The acute nature of trauma also does not typically permit a fully detailed review of risk factors for each patient, especially during resuscitative and other emergent treatment.

In a study of urban trauma patients, the incidence of HIV infection (HIV Ab+ or Ag+) is found to be 4.3%, with an incidence of surface antigen of the hepatitis B virus (HBsAg) of 3.1%.[72] Predictors of a preexisting viral infection included patient aged 20 to 49 years, IV drug abuse, prior HIV testing, shock, and death (all $p < .05$). IV drug abuse was the single most significant predictor. Patients who required resuscitation or eventually died of the trauma had a 12% to 21% infection rate with HIV, hepatitis B virus (HBV), or both.[72]

This important proportion of virally infected patients requires heightened awareness and training to prevent disease transmission. One factor associated with violating barriers to the oral and maxillofacial surgeon is the placement of arch bars. Single gloving of the surgeon is

BOX 32-4 Strategies for Preventing Transmission of Infection to the Operative Team from the Patient

- Use protective gloving
- Use double gloving for all surgeries
- Consider triple gloving or double gloving with an internal glove liner for insertion of arch bars
- Change gloves after completion of arch bar insertion and rescrub
- Change gloves after every 2 hr of surgical time
- Only one surgeon is to pass wires at a time
- Never use fingers as retractors
- Never do anything like bending needles or recapping syringes
- Use impermeable gowns
- Use face shield–splash guard or eyeglasses with side shields
- Obtain appropriate hepatitis B vaccination and boosters
- Consider use of composite resin bonded arch bars

associated with a 100% incidence of glove perforation when using wires.[73] This was reduced to 37% with the use of double gloves. Triple gloving eliminated the inner glove perforation entirely during arch bar placement but caused paresthesia of the surgeon's fingers and reduced dexterity.[74] Other factors include procedures lasting longer than 3 hours or more than 300 mL of total blood loss.[75] Therefore, many maxillofacial trauma repairs place the surgical team at risk. Strategies to prevent infection from the patient to the surgeon and staff are listed in Box 32-4.

Although HBV vaccine has been available for more than 10 years, many surgeons are not adequately immunized. Many have had vaccines more than 5 years previously, but approximately 25% received an inadequate or inappropriate vaccination.[76]

Patients with viral infection may also have an increased risk of infection because they are immunocompromised. In a series of orthopedic trauma patients, HIV-seropositive patients were significantly more likely to acquire a postoperative infection (16.7% versus 5.4% in the seronegative group). Compound limb fractures had an infection rate of 55.6% in the seropositive group as opposed to 11.3% in the seronegative group.[77]

In a study of maxillofacial trauma patients at an urban hospital, 19.8% of mandibular fracture patients were seropositive for HIV, as were 7.75% of patients with midfacial fractures. This is consistent with interpersonal violence contributing to mandibular fractures, whereas midfacial fractures more often occurred subsequent to an MVA. The seropositive mandibular fracture patients had a preoperative infection rate of 26% and there was a trend toward a higher postoperative infection rate as well. These infections responded to amoxicillin and clavulanate (Augmentin) or clindamycin.[78] HIV-positive patients showed a significantly higher rate of postoperative infections (30% versus 9.5% of HIV-negative patients) in mandibular fracture cases. Open reduction in this group of patients was associated with a 45% infection rate,[79] indicating that closed reduction should be used if possible.

SUMMARY

The acutely injured patient often attracts a great deal of initial attention in the emergency room because of the immediate need for resuscitation. Once the patient's condition is stabilized, the risk of infection should be evaluated and appropriate therapy started. The traumatized patient is severely compromised in the ability to resist even a normal bacterial insult. The progression of infection may result in loss of structures, limb, and even life if sepsis occurs. It is therefore incumbent on the treating physician to be aggressive in wound débridement, bacterial load reduction, and appropriate antibiotic and antitoxin administration, and in assuming the management of the injuries to maximize the functional and aesthetic result. Surgeons also have a responsibility to themselves, residents in training, and other staff to maintain a high standard in regard to prevention of disease transmission from a potentially virally infected patient to a member of the surgical team.

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CHAPTER

33

Principles of Fixation for Maxillofacial Trauma

Ashish A. Patel | Vasiliki Karlis

OUTLINE

History of Fracture Treatment and Development Of Modern Osteosynthesis
Biology of Bone And Bone Healing
Biophysics of the Facial Skeleton
Methods of Fixation
Rigid Versus Functionally Stable Fixation
Compression Plate Osteosynthesis
Noncompression Osteosynthesis
Mandibular Fixation
Locking Plates
Miniplates
Reconstruction Plates
Lag Screws
Midface and Upper Face Fixation
Bioabsorbable Plate Fixation
Complications of Internal Fixation
Surgical Site Infection
Dental Injury
Nerve Injury
Malocclusion

HISTORY OF FRACTURE TREATMENT AND DEVELOPMENT OF MODERN OSTEOSYNTHESIS

Bone fractures have been treated with various conservative techniques for centuries and it was not until the eighteenth century that internal fixation was first documented. Icart, a French surgeon in Castres, performed ligature fixation with brass wire on a young man with a humeral fracture. Since then, many surgeons developed several methods for internal fixation, but one of the greatest leaps in bone trauma surgery was in 1886, when Hansmann of Hamburg published a technique using retrievable metal bone plates with transcutaneous screws. Soon after, a Belgian surgeon, Albin Lambotte, improved these techniques and coined the term *internal fixation*. Lambotte developed and manufactured a variety of bone plates and screws and much of his armamentarium remained in use until the 1950s.[1]

In the twentieth century, Sherman improved on Lambotte's designs and created parallel, threaded, fine-pitched, self-tapping screws. This hardware was made of corrosion-resistant vanadium steel, which was a strength improvement over silver and ivory fixation materials. Although sturdy, these early alloys were inflammatory and were eventually replaced entirely by titanium in the 1970s. In the 1930s, Eggers rediscovered an older design for sliding slot plates, which eventually led to the development of a compression plate by Danis in 1947. Luhr helped advance the principles of compression and dynamic compression, but it wasn't until 1977 that he developed these techniques to the maxillofacial skeleton. Spiessl later popularized dynamic compression bone plating of the mandible using Arbeitsgemeinschaft für Osteosynthesefragen-Association for the Study of Internal Fixation (AO-ASIF) techniques.

From Luhr and Spiessl's work, eccentric dynamic compression plating was developed and adapted for craniomaxillofacial trauma use, but lost popularity due to its highly technique-sensitive nature and no proven benefits over other modern fixation methods.

BIOLOGY OF BONE AND BONE HEALING

Bone is a complex and ever-evolving connective tissue and serves multiple purposes. Besides being the main constituent of the human skeletal system, bone is highly metabolically active and essential for the regulation of serum electrolytes—namely, calcium and phosphate. Marrow cavities are filled with hematopoietic elements necessary to manufacture and maintain blood components and regulate the immune system. Bone is comprised of calcified bone matrix and three major cell types, osteocytes, osteoblasts, and osteoclasts.

Bone's organized structure is illustrated in cross section revealing the haversian system, or osteon. Each osteon contains concentric layers of compact bone surrounding a central haversian canal, which harbors the neurovascular bundle supplying the unit. Cells suspended in this highly calcified, highly vascular structure are perfused via small capillary-containing cylindrical cavities called canaliculi.[2]

Bone healing can be broadly categorized in two ways, primary and secondary. Primary, or direct bone healing, requires rigid fixation and immobility of fracture segments with a minimal gap between them (less than 100 μm). Osteoclasts migrate to the fracture site and widen

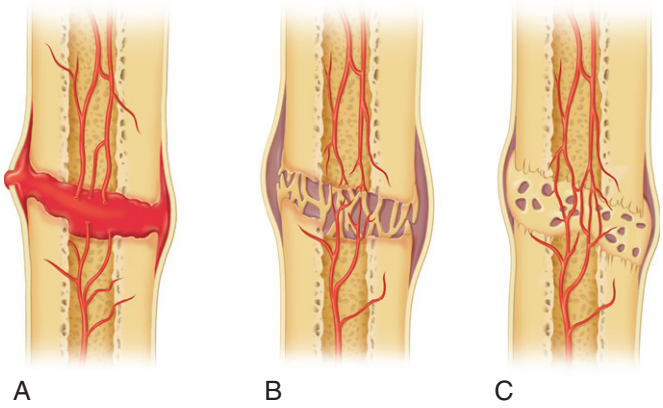


FIGURE 33-1 **A,** Fracture hematoma. **B,** Soft callus. **C,** Hard callus.

adjacent haversian systems, allowing osteoblasts to deposit bone matrix, or osteoid, eventually to calcify into organized mature lamellar bone.[3]

Secondary, or indirect bone healing, is more complex and occurs when a significant gap or interfragmentary motion is present. Secondary bone healing involves the formation of a fibrocartilaginous intermediary bone callous (Fig. 33-1) There are four distinct stages of indirect bone healing but the end product is the same as mature bone formed in primary healing. The initial insult propagates the inflammatory stage. A hematoma between and around the fracture develops and stabilizes, drawing inflammatory cells to the site. Necrotic and nonviable bone near the fracture is cellularly débrided and repair is initiated by angiogenesis and the activation of osteoprogenitor cells and fibroblasts. The second, or soft callus, stage is characterized by conversion of the hematoma to a fibrocartilaginous mass to bridge the fracture. Fibroblasts and mesenchymal elements are highly active in laying down new collagen to create the substrate into which the third phase, or hard callus stage, develops. During this period, osteoid is calcified and periosteal and endosteal bone ingrowth starts to replace the soft callus as a result of endochondral bone formation. Finally, in the remodeling stage, the woven bone of the hard callus matures and organizes to a trabecular structure to re-create the native preinjury structure.[4]

Although distinct, both types of bone healing may occur simultaneously in the same fracture. As three-dimensional structures, bones may have varying levels of contact and fracture reduction in the same general site, resulting in endochondral and lamellar elements in different areas at the same point in time (Fig. 33-2).

BIOPHYSICS OF THE FACIAL SKELETON

Although complex, the facial skeleton does not consist of many moving parts. The major axis of bony motion in the face is around the mandibular condyles, or temporomandibular joints (TMJs). The muscles of facial expression originate on various bones of the craniomaxillofacial skeleton, are invested in the superficial musculoaponeurotic system, and insert on each other and the facial skin. These have little effect on forces exerted on facial bones.

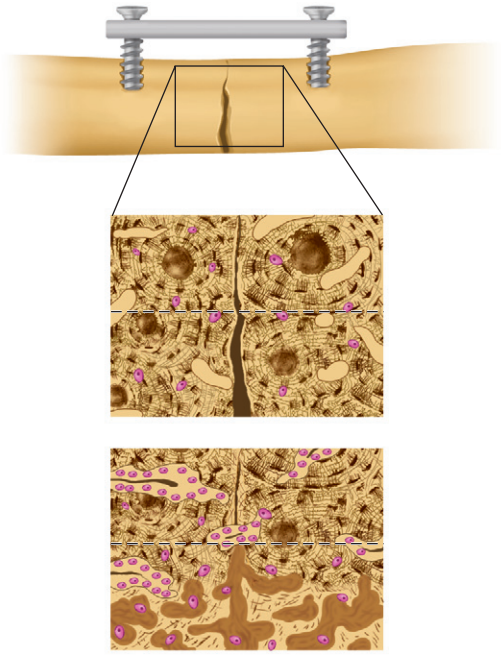


FIGURE 33-2 In many fractures, it may be difficult to achieve and assess complete bone contact through the entire width of the fracture. In this case, healing by primary intention may still occur, but as contact healing where there is good apposition and gap healing where there is a space. In gap healing, bone is deposited parallel to the fracture and eventually remodels and becomes parallel to the long axis of the native bone.

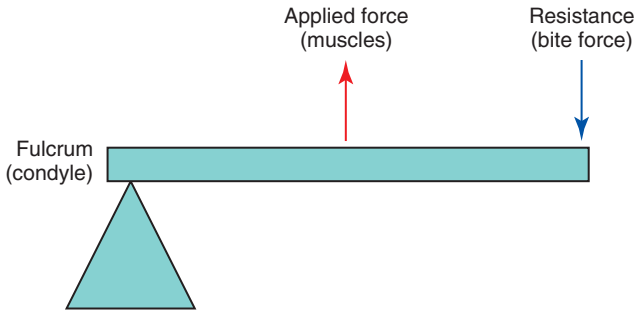


FIGURE 33-3 The mandible as a class III lever.

The muscles of mastication and suprahyoid muscles, however, produce significant forces on the jaws and surrounding osseous structures. Bite force is generated by contracture of the masseters, temporalis, and medial pterygoids; the sum of these vectors allows for occlusion of the teeth via movement of the mandible.

It is imperative to understand the physics and forces applied to facial bones to fixate fractures properly and use biomechanics to our advantage. Due to its dynamic nature, the mandible bears most of the forces applied by facial musculature to the skeleton. Beam mechanics dictates that the mandible is a class III lever, with the condyle being the fulcrum, the muscles of mastication acting as the applied force, and bite load acting as the resistance (Fig. 33-3). This rationale applies to one side of the mandible at a time, but as a horseshoe-shaped bone, the mandible is more than a simple class III lever. Vectors

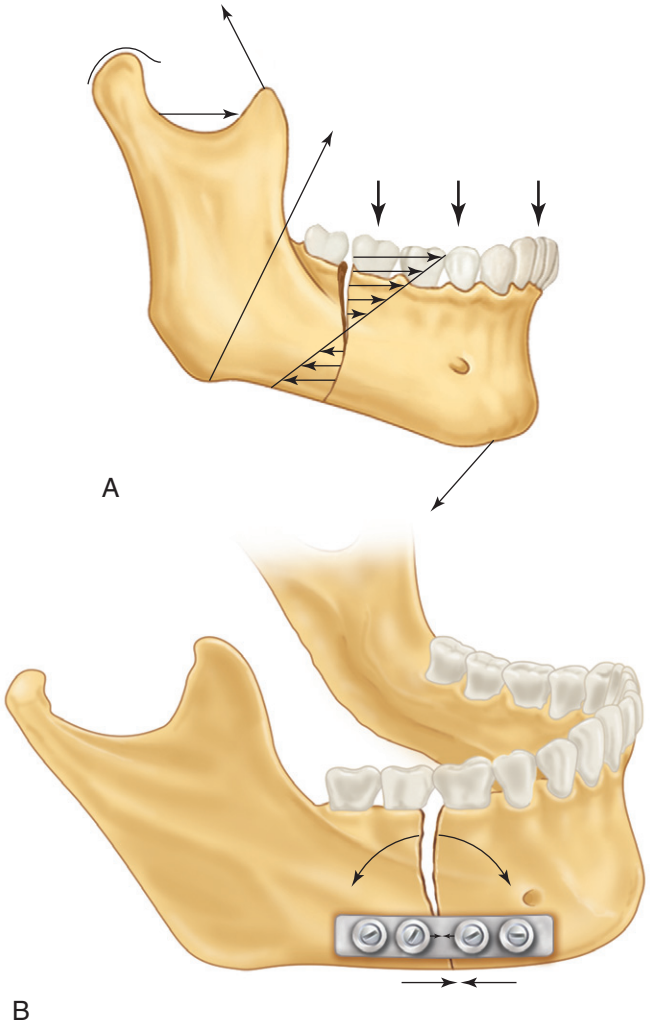


FIGURE 33-4 **A,** The effects of the masticatory apparatus *(thin arrows)* and occlusal load are demonstrated *(thick arrows).* Under function, the mandible exhibits the greatest tensile forces across the superior border and compression at the inferior border. The neutral zone lies at approximately the level of the inferior alveolar canal. **B,** When using compression plating at the inferior border, tensile forces at the superior border may be exaggerated. A tension band or arch bar is required to counteract these forces.

from bilateral muscle contracture on a unilateral fracture produce rotation and torques that cannot be easily described by simple beam mechanics.[5,6] Varying bone thickness, occlusal arrangements, and multiple vectors of muscle contraction also cannot be accounted for in this model. For the purposes of understanding rigid fixation across fractures, beam mechanics applies.

When loaded, the mandible exhibits maximum tension at the superior border and maximum compression at the inferior border (Fig. 33-4). This is a gradient and, between the zones of tension and compression, lies a neutral zone in which opposite forces total zero. In this model, it would be mechanically advantageous to apply rigid fixation hardware along the zone of tension, or superior border.[7] Biologically, this is complicated by the presence of teeth, thin cortical bone, and thin overlying soft tissue. The neutral zone is dynamic and depends on the moment of the bite force applied, but generally correlates to the plane in which the inferior alveolar neurovasculature lies, and therefore not acceptable to place hardware. The inferior mandibular border is a biologically sound location for hardware placement. Bicortical screw fixation here is extremely stable, there is adequate vascular soft tissue to prevent dehiscence or hardware palpability, and dental structures usually do not extend to this area. To compensate for lack of stability at the zone of tension, a tension band can be applied. This could be a monocortical low profile miniplate, lag screw, or arch bar, all of which resist tensile forces without violating biologic principles of surgery.

In vivo, the mandible is more complicated than the model just described. Because of the three- dimensional nature of the mandible, multiple muscular insertion sites, and bilateral nature of the muscles, zones of compression and tension are not always static around a fracture. For example, in the mandibular corpus or parasymphysis, forces may be inverted if bite force is applied posterior to the fracture line secondary to forces applied from the contralateral musculature.[8] This creates compression at the alveolus and tension at the inferior border. Torsional forces also act on segments due to the bilateral nature of the forces applied. This is easily demonstrated by rotation of segments around the neutral zone when only one point of fixation is implemented or widening of the mandible at the condyles due to alveolar process torquing.[9]

METHODS OF FIXATION

Traumatic injuries of the facial skeleton can be properly managed and treated in numerous ways. AO-ASIF guidelines of rigid fixation follow four basic principles to ensure adequate treatment of fractures: bony segment reduction, stable fixation and immobilization of fragments, maintaining blood supply, and early function.[10] As noted, primitive methods of internal fixation developed from the 1800s. Advancements in techniques and biomaterials and an understanding of biophysics have greatly changed how we currently apply fixation to facial fractures. Internal fixation with titanium hardware is still the most commonly used method of treating facial skeletal injuries and a plethora of systems are available to carry this out. Various sizes and shapes of plates and screws exist to meet the needs of the surgeon and to tailor treatment to each individual patient and fracture type.[11]

Adequate exposure of fracture segments is carried out while not compromising the adjacent blood supply. Maintaining vital periosteum aids in fracture healing, preventing postoperative wound breakdown and decreasing the rate of hardware infection. Fracture segments can be reduced using various methods, including bone reduction forceps, manual anatomic reduction, interdental fixation, and a combination of these. The fracture segments are stabilized by bending and adapting plates directly to the bony segments and fixated with screws. Primary closure of the wound may or may not require local flaps to maintain well-vascularized soft tissue coverage.

Cases in which surgical exposure of fracture sites may interrupt blood supply, such as severely comminuted fractures or contaminated wounds, pose a risk for hardware infection and may be an indication for skeletal pin external fixation.[12] This technique works especially well in comminuted and contaminated mandibular fractures requiring control of non–tooth-bearing proximal segments. Transcutaneous access to the mandible with stab incisions and blunt dissection is achieved. The mandible is drilled using a drill guide to protect the soft tissue and skeletal pins are inserted into the fractured segments. Ideally, two pins with a large distance between them should be placed into each large segment to prevent rotation. To ensure optimum biomechanics, each pin should be as large as possible and placed close to fracture lines (but not closer than 1 cm to prevent the risk of segment shearing). The external components of the skeletal pins are stabilized by being fastened to a rigid external bow or linking segments with the use of other, smaller subunit bars. Smaller shards of comminuted bone generally do not require fixation because they can be immobilized as they are sandwiched between larger externally fixated segments. Care is taken not to devitalize these small bone segments by avoiding aggressive débridement. Maintaining blood supply is essential to the healing of these fractures. In cases in which internal fixation cannot be applied without devitalizing bone segments, such as a grossly contaminated open mandibular fracture, external fixation may serve as a temporary measure to immobilize and stabilize the segments while the wound is washed out and sterilized over a period of days to weeks. This can be ultimately converted to internal plate fixation to avoid prolonged skeletal pin hardware application.[13]

Maxillomandibular fixation (MMF) or interdental fixation is widely used in the management of almost all injuries affecting the jaws. As described, this is an excellent method for achieving fracture reduction. Prior to the development of modern internal fixation, MMF was the mainstay of facial fracture treatment. By stabilizing the dentition in its known pretraumatic occlusion, bone segments will assume an anatomically acceptable configuration. Because MMF compresses fractures at the alveolus, the inferior border of the mandible may still demonstrate a gap. By combining this method with compression of the inferior border with bone reduction forceps and application of internal fixation methods, an ideal reduction can be achieved. Several hardware designs and techniques are available for MMF including arch bars, Ivy loops, Stout interdental wires, and MMF screws. The advantage of MMF with the application of arch bars is that they provide a tension band at the alveolar component of maxillomandibular fractures. This aids in resisting tensile forces of the fracture near the teeth. Also, arch bars have numerous fixation points (lugs), allowing more precise control of fracture segments and application of force vectors when placing the patient into MMF. In fractures not involving dentate portions of bone—that is,. mandibular angle, ramus, or condyle fractures—arch bar application may not be necessary to obtain MMF. If a stable and reproducible occlusion can be obtained, the use of MMF screws or Ivy loops can provide reduction and stabilization of the fracture in a much shorter, less technique-sensitive manner.[14] Hand articulation of the jaws has also been described and deemed successful by some authors. These methods are also more hygienic than the use of standard arch bars, prevent excessive biofilm accumulation, are less traumatic to the periodontium, and can be removed relatively quickly.[15] Although not as rigid and mechanically stable as arch bar MMF, with appropriate patient selection, screw and/or interdental wire MMF can provide excellent results for the nonoperative management of mandibular fractures.[16] A retrospective cohort of 75 patients with 83 mandibular angle fractures treated by Bell and Wilson[17] has described three different methods of intraoperative intermaxillary fixation prior to the application of a single, 2.0- mm miniplate via the Champy technique. There were no significant differences in outcomes, reoperation rates, and complications among the Erich arch bar, Stout interdental wire, and manual reduction groups.

MMF is still used as a primary modality of fracture treatment in patients for whom internal fixation may not be indicated. Minimally or nondisplaced biomechanically favorable fractures in patients with a sufficient complement of teeth to provide a stable premorbid occlusion, severely comminuted fractures, or intracapsular condylar fractures in which occlusion can be reestablished are some common scenarios for which 2 to 8 weeks of MMF without surgery may be indicated. MMF is considerably less invasive and more cost-effective and reduces complications associated with open surgery; however, it poses its own unique set of risks and complications. MMF invokes an aspiration hazard in patients such as those with severe gastroesophageal reflux, nausea, seizure disorder, alcoholism and dysphagia. Patient compliance is essential to prevent loosening or cutting of wires and mobilizing the forming callus. There is a great deal of evidence from orthopedic literature demonstrating ill effects on the skeletal system from prolonged immobilization and disuse of long bones. These effects are demonstrable not only on the immobilized bone itself, but on the associated muscles, vasculature, and joints. Bone immobilization results in hypomineralized osteoporotic cortices and trabeculae and an overall decreased oxygen tension and pH of the nutrient vessels. Muscles inserting on this bone also atrophy with disuse and usually require extensive physical therapy for the patient to return to full strength and mobility.[18,19] Decreased muscle demand inevitably results in hypovascularity and protein loss over time. Surrounding immobilized joints demonstrate pericapsular contractures and synovial hyperplasia, which decrease range of motion. Over time, the formation of intracapsular adhesions can result in pain, inflammation, and derangements in joint function. The dreaded complication of TMJ ankylosis may follow prolonged immobilization of displaced or comminuted condylar fractures.[20,21]

Condylar fractures are surrounded by controversy in respect to closed versus open treatment. Internal fixation tends to produce superior results in regards to anatomic reduction, but studies have shown variability in postoperative mandibular function, symmetry, and joint pain. Variability in fracture patterns of the condyle,

concomitant mandibular fractures, occlusal stability, patient compliance, postoperative physiotherapy, and operative technique have significant effects on study outcomes of closed and open fracture treatment and have led to opposing viewpoints. A meta-analysis of several studies has failed to show any overwhelming overall benefit of closed versus open treatment due to lack of consistency in reporting the variables described.[22,23]

RIGID VERSUS FUNCTIONALLY STABLE FIXATION

Internal fixation can be subclassified in several ways, but perhaps the most appropriate would be delineating between rigid and nonrigid. Rigid fixation can be defined as any type of directly applied bone fixation that prevents interfragmentary movement between fracture segments when that bone is under active load.[24] Examples of rigid fixation of a fracture include application of a reconstruction plate, two bone plates, two lag screws, or a compression plate and arch bar across a fracture. With the exception of the use of a load-bearing reconstruction plate, rigid fixation techniques rely on two point fixation—a stabilizing unit, such as a bone plate at the inferior border, and a tension band, such as a miniplate or arch bar superior to that.[25,26] On a histologic level, the benefit of rigid internal fixation with minimal gap between the bone segments allows for primary bone healing via haversian remodeling. The fracture gap is traversed by osteoclasts and then undergoes angiogenesis, followed by deposition of osteoid by adjacent osteoblasts. The bone remodels over time to create mature haversian bone. This is in contrast to fractures with a significant gap or interfragmentary motion, which heal by secondary intention and formation of an intermediary hematoma and bone callus.

Nonrigid fixation is just that—fixation that allows for movement between the bone fragments across a fracture line. Many older techniques, such as interosseous wiring or interdental bridal wiring, stabilize fractures to approximate segments but do not prevent interfragmentary movement. Depending on the magnitude of movement across the fracture, nonrigid fixation may result in nonunion or malunion. With the methods and materials available today for maxillofacial trauma surgery, many types of nonrigid fixation are no longer in use. Perhaps the most important nonrigid technique in mandibular trauma is the Champy method for the fixation of angle fractures. In 1978, Champy described the use of a single miniplate adapted to the superior border of mandibular angle fractures, with excellent results. This technique has been termed *functionally stable* because it allows for activation of the mandible during healing, even with interfragmentary motion.[27] Due to the rostral force vectors of the pterygomasseteric sling and caudal pull of the suprahyoid muscles, the superior border of the mandibular angle is under tension and the inferior border is under compression. Because the monocortical miniplate is applied to the superior border, the mechanical advantage favors stabilization of the fracture under active forces. Although functionally stable fixation of the mandibular angle reduces operative time, risk of dental injury, and cost, it is not ideal in all situations. Concomitant fractures of the mandible must be treated rigidly to prevent motion at multiple sites. The Champy method relies on the contralateral condyle being seated correctly in the glenoid fossa, without disruption of the temporomandibular relationship. If a contralateral fracture is present and not treated rigidly, bite forces across the angle can transmit to the distal segment, causing rotation around the opposite fracture line. This may result in widening of the mandible and subsequent malocclusion and facial width alteration. By treating the other fracture site rigidly, the angle can essentially be treated as an isolated injury.

COMPRESSION PLATE OSTEOSYNTHESIS

The use of compression plating systems in the maxillofacial skeleton has been used to treat mandibular fractures for many years. Although many surgeons prefer the ease of use of locking bone and reconstruction plates, compression and dynamic compression plating, if applied correctly, can be advantageous in immobilization and fixation of mandible fractures.

The goal of compression osteosynthesis, as described by AO, is establishing absolute stability across a fracture. This is defined as zero movement occurring between bones across the fracture, as well as complete immobility of the hardware against the bone. This creates an ideal environment for primary bone healing by generating friction between the bone segments in compression and minimizing the gap between them.[28] Linear compression between the segments counteracts torsional forces produced by the masticatory apparatus during function and prevents interfragmentary motion. Traction perpendicular to the fracture line is maintained by the plate itself, which shares the load under function and maintains compression of the segments.[29]

Today, most mandibular plating modules include dynamic compression plates for surgeons who wish to use compression osteosynthesis (Fig. 33-5). Although efficient in creating absolute stability in mandibular fractures, compressive plating techniques, even with the advent of the dynamic compression plate, are extremely technique-sensitive and prone to operator error. If the



FIGURE 33-5 Dynamic compression plate.

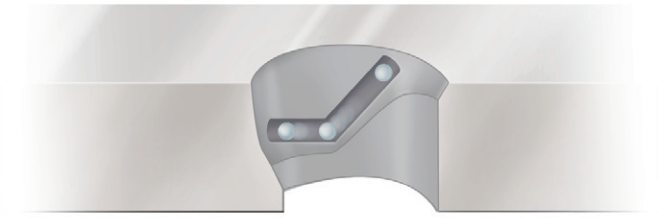


FIGURE 33-6 The holes on dynamic compression plates have an inclined plate, with the lowest edge closest to the bone surface. When screws are tightened in the active position, the head glides down the inclined plane, causing translation of the bone segment toward the fracture.

holes are not drilled in the proper location and screws are not placed in the correct sequence, the hardware can distract the fracture, creating gaps in bone, malocclusion, and excessive torsional forces across the plate.[30]

Dynamic compression plates are designed with eccentric holes with inclined planes. On either side of the midline of the plate, the plate holes are elongated, with the lateral side having the highest portion of the inclined plane and the medial with the lowest portion, or closest to the bone, of the inclined plane (Fig. 33-6). The plate should be adapted so that one eccentric hole is on each side of the fracture, closest to the fracture line. The outer planes of each hole are the active, or compression, sites. As screws are drilled and fastened into this high point of the inclined plane, they follow the plane down toward the bone as friction is created between the screw head and plane surface. When completely tightened, they lie on the innermost portion of the hole closest to the bone. Because this is completed on either side of the fracture, the bone segments are compressed toward each other while the plate remains static, minimizing the bone gap and achieving compression. The remainder of the holes distal to the fracture line are then drilled and secured with bone screws in a passive position so as to not compress or distract the bones and hardware further.

To instrument the dynamic compression plate properly and achieve successful compression, the plate must first be bent and accurately adapted to the bony segments. The fracture must be stabilized and reduced by MMF, a superior border miniplate, bone reduction forceps, or a combination of these techniques prior to bending the dynamic compression plate. Once adapted to the reduced fracture, the compression elements can be drilled. Drill guides provided by the manufacturer for compression plating are helpful in placing the screw hole correctly to achieve maximum compression. The drill guide has active and passive positions, with arrows to indicate the orientation (Fig. 33-7). The first hole adjacent to the fracture is drilled in a bicortical fashion with a drill guide with the active, or compression, arrow facing the fracture. This corresponds to the outer, or high, incline of the hole. A depth gauge is used to measure the desired screw length and the screw is inserted partially to stabilize the position of the plate. The most proximal hole on the opposite side of the fracture is drilled in the same fashion in the active position and the screw is inserted and tightened completely. As noted, the screw

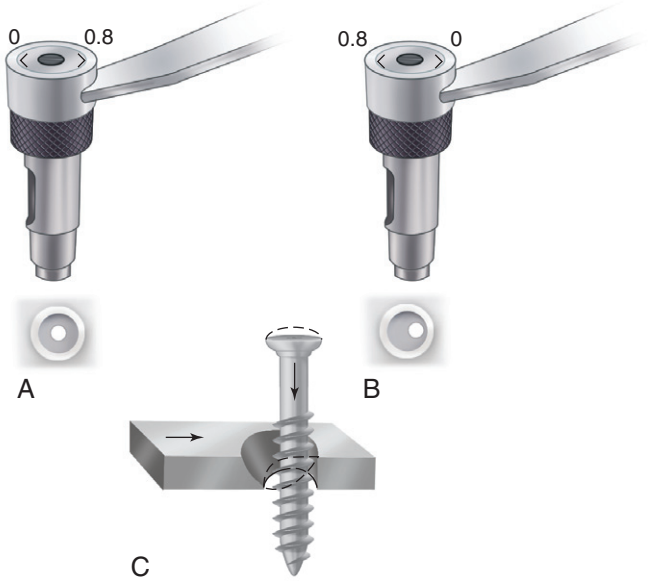


FIGURE 33-7 The drill guide for compression plating has two positions, active and passive. In the passive position (A), the drill hole and screw are placed in the center position of the plate hole, essentially acting as a noncompression system. In the active position (B), the screw is placed at the upper incline position of the plate and, as it is tightened (C), it moves down the inclined plane, resulting in compression of segments toward the fracture line.

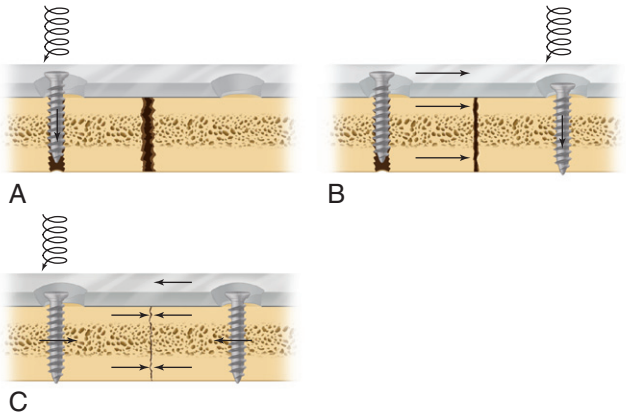


FIGURE 33-8 As each screw closest to the fracture line is tightened in the active position, the mandible translates across the plate toward the midline (A, B). When both active compression screws are tightened completely (C), compression across the fracture is complete.

will migrate down the plane approaching the fracture line and draw the bone segment toward its counterpart. The first screw is then tightened completely, producing the same effect on the opposite side and creating compression between the bony segments. The remainder of the holes are then drilled in the passive position and bicortical screws are inserted to stabilize the plate to the fractured mandible (Figs. 33-8 and 33-9). These serve to share the load further and reduce forces that would tend to counteract interfragmentary compression.

Dynamic compression plates actively draw fractured segments together. The resultant compression at this site, typically the inferior mandibular border, may result in

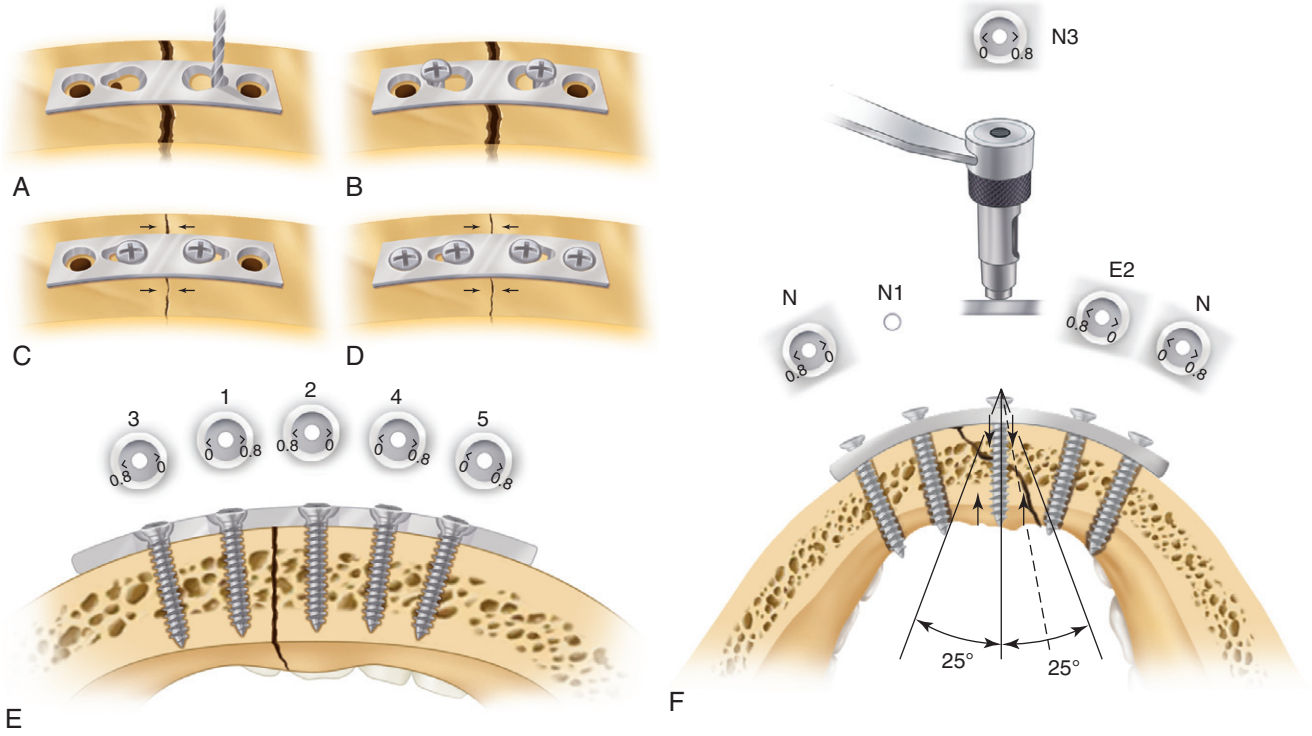


FIGURE 33-9 **A,** After adaptation of the plate to the mandible, the holes are drilled in the active or lateral positions of the holes adjacent to the fracture. **B,** Each screw is inserted and partially tightened. **C,** Each screw is then completely tightened, thereby accomplishing compression osteosynthesis. **D,** Screws are placed in the passive position in the remaining holes for increased stability. **E,** Order of screw placement for a dynamic compression plate (DCP). Note that positions 1 and 2 have screws engaged in the active position. **F,** In an oblique fracture of the symphysis, a DCP can be combined with a lag technique to ensure maximum reduction and compression of the fracture. The center screw (N3) traverses the fracture using a lag technique and using the passive or neutral position in the plate. The eccentric hole is used adjacent to the fracture (E2) to draw segments together in the horizontal plane; the remainder of screws are placed neutrally.

excessive tension at the superior border or alveolus. It is necessary to neutralize these forces to prevent gap formation in the zone of tension of the mandible. This is typically achieved by the use of a tension band. An arch bar, superior lag screw, or monocortical miniplate can be used as a tension band to reduce the distraction at the superior border. This applies to any load-sharing internal fixation system but holds especially true for compression plating.

Compression osteosynthesis is best applied in transverse fractures of the mandibular symphysis or body without comminution or bone loss. Obliquely oriented fractures can pose problems in this technique due to the nonsymmetrical nature of the fracture line. Plates are adapted and applied to the outer, or buccal, cortex of the mandible. Compression is applied parallel to the plate; equal distribution of forces occurs best in fractures that are completely perpendicular to the compression plate. Compression of obliquely oriented fractures may result in excellent compression near the plate, but distraction and unwanted force vectors elsewhere. An example of this is lingual cortical splaying of the mandible when fixation is applied. Although this can occur with the use of standard internal fixation osteosynthesis, the effect may be more pronounced in compression osteosynthesis. Compressive forces are maximized at the bone-plate interface, which can accentuate the amount of splay at the opposite cortex. To minimize this ill effect, one can slightly overbend the plate on the buccal aspect of the mandible so that when activated, compression is also achieved at the lingual cortex. In cases in which there is avulsion of bone at the fracture site, compression plating can distort the premorbid anatomy and contours of the mandible, leading to malocclusion and increased stress across the TMJs.

NONCOMPRESSION OSTEOSYNTHESIS

Noncompression osteosynthesis is widely used in managing traumatic injuries to the maxillofacial skeleton. This can be accomplished with a variety of methods including non-compression bone plates and reconstruction plates, both of which are available with locking mechanisms. These methods have broader applications and less degree of operator error when compared with compression osteosynthesis.

MANDIBULAR FIXATION

The mandible lends itself to a number of fixation techniques secondary to its geometry, length, bicortical structure, and complex applied muscle forces. Unlike most bones of the facial skeleton, the mandible is repeatedly

stressed and strained by the function of the masticatory apparatus. Fixation must be sufficient to withstand these forces during the healing period. Fracture plates are manufactured in various widths and universal fixation systems generally allow interchangeable screw diameters to be used in multiple plates, depending on the level of fixation desired. Other factors that should be taken into account when selecting the width of the fracture plate are quantity and quality of overlying soft tissue, patient compliance, and risk of reinjury. Thicker plates provide more stability than thinner counterparts, but may be palpable under soft tissue, may require more dissection, are more difficult to adapt, and have higher rates of dehiscence. The stress-shielding effect, especially when using thick reconstruction plates, has been evidenced in animal models of mandibular fixation, but has never been proven to occur in human craniomaxillofacial studies.[31]

After selecting the appropriate plate, the mandible fracture is exposed and reduced (see earlier) and the plate is adapted to the inferior border of the buccal cortex of the mandible. The plate may be overbent, if desired, to compensate for lingual segment splay, and is held in place with plate-holding forceps or the desired instrument. Two (ideally three) screws are required on each side of the fracture to ensure stability and prevent rotation of bone segments.

Although not as crucial as when applying dynamic compression plates, the sequence of drilling and screw placement is important. Screws are placed first on the sites most proximal to the fracture line and secured. When using bone screws, this prevents shifting of the plate on the mandible as screws are first secured to either side of the fracture because the plate may bend to adapt to the underlying bone as the screw is tightened. In a well-adapted plate, this is of less clinical significance but the plate may still warp. Holes are drilled bicortically with the use of a drill guide to prevent whipping of the drill bit and precise placement of osteotomies in the middle of each plate hole. Many drill guides can snap or screw into the plate hole at a 90-degree angle to allow for precise perpendicular drilling into bone. Hole lengths are measured with a depth gauge from the plate surface to the lingual cortex. If the exact screw size is not available—many systems increase in length by increments of 2 mm—it is best to round up to the next size to ensure capture of the lingual cortex. Previously, drill holes had to be threaded with a bone tap prior to accepting a screw. Modern mandible fixation screws eliminate this step with the use of self-tapping screws that create a thread pattern in the bone as they are screwed in. When placing screws, the same angle used to drill should be replicated to prevent distorting the osteotomy or breaking the screw. Because most plates are designed to accept two or three diameters of screw, a rescue or emergency screw can be used in the event that the initial one does not engage. If the osteotomy is widened by whipping of the burr or poor bone quality, a larger diameter screw may engage in the same site. When the screws most proximal to the fracture are secured, the remainder are drilled and placed with the same method in an alternating fashion in regard to the fracture (Fig. 33-10).

LOCKING PLATES

Modern plating systems are designed with fracture plates that accept standard bone and locking screws. This can be useful in securing plates that cannot be perfectly adapted to fractures or if bone quality is poor.[32] Locking screws are double-threaded; the head of the screw has an additional larger diameter thread that secures into the thread pattern of the plate hole. With this design, the screw actually locks into the plate irrespective of the bone. This prevents shifting of bone segments and deformation of the plate as the screws are tightened, which is particularly important in plates that are not well adapted to the underlying bone. Locking plate and screw systems prevent loosening and extrusion of the screw from the plate, even if it does not integrate to the mandible and resists mechanical yielding under stress.[33]

MINIPLATES

Miniplates have several applications in mandible fracture fixation; the most important is the Champy method of mandibular angle fracture fixation and its use as a tension band. These methods call for monocortical fixation of miniplates, but bicortical fixation of miniplates has applications in the mandible as well. These plates accept the same screws as standard mandibular fracture plates. Most miniplates are also of the locking variety if locking techniques are desired, but hold less significance in that application given the thin width and greater malleability as compared with traditional fracture plates.

The Champy method of mandibular angle fixation involves exposing and reducing a fracture, as described earlier, and using the biomechanical advantage to place a miniplate at the zone of tension—that is, the superior border. Depending on the exact location of the fracture, the plate is adapted to the external oblique ridge and ascending ramus with at least two screw holes on each side. To avoid damage to dental and neurovascular structures, monocortical drill holes are created with a protected burr of the desired diameter and the respective monocortical screws are secured. Adaptation of this plate commonly requires three-dimensional bending because the external oblique ridge has in-plane and out of plane curvatures (Fig. 33-11). This method has been proven to exhibit enough stability to withstand tensile forces at the superior border under function during the healing period.

As a tension band, the monocortical application of the miniplate resists distracting forces at the superior border of the mandible. After application of an inferior border fracture plate, a miniplate can be adapted to the mandible superior to the neutral zone and secured with monocortical bone screws (Fig. 33-12). Care must be taken to place this plate in the zone of tension while avoiding tooth roots. Even with monocortical fixation, damage to dental structures can occur because the relationship of teeth to the mandibular buccal cortex vary from patient to patient. In the edentulous mandible, tension bands should be placed at the superior border to maximize tensile force resistance. Miniplates are also more prone to screw loosening and infectious complications due to decreased stability and strength compared with thicker hardware.

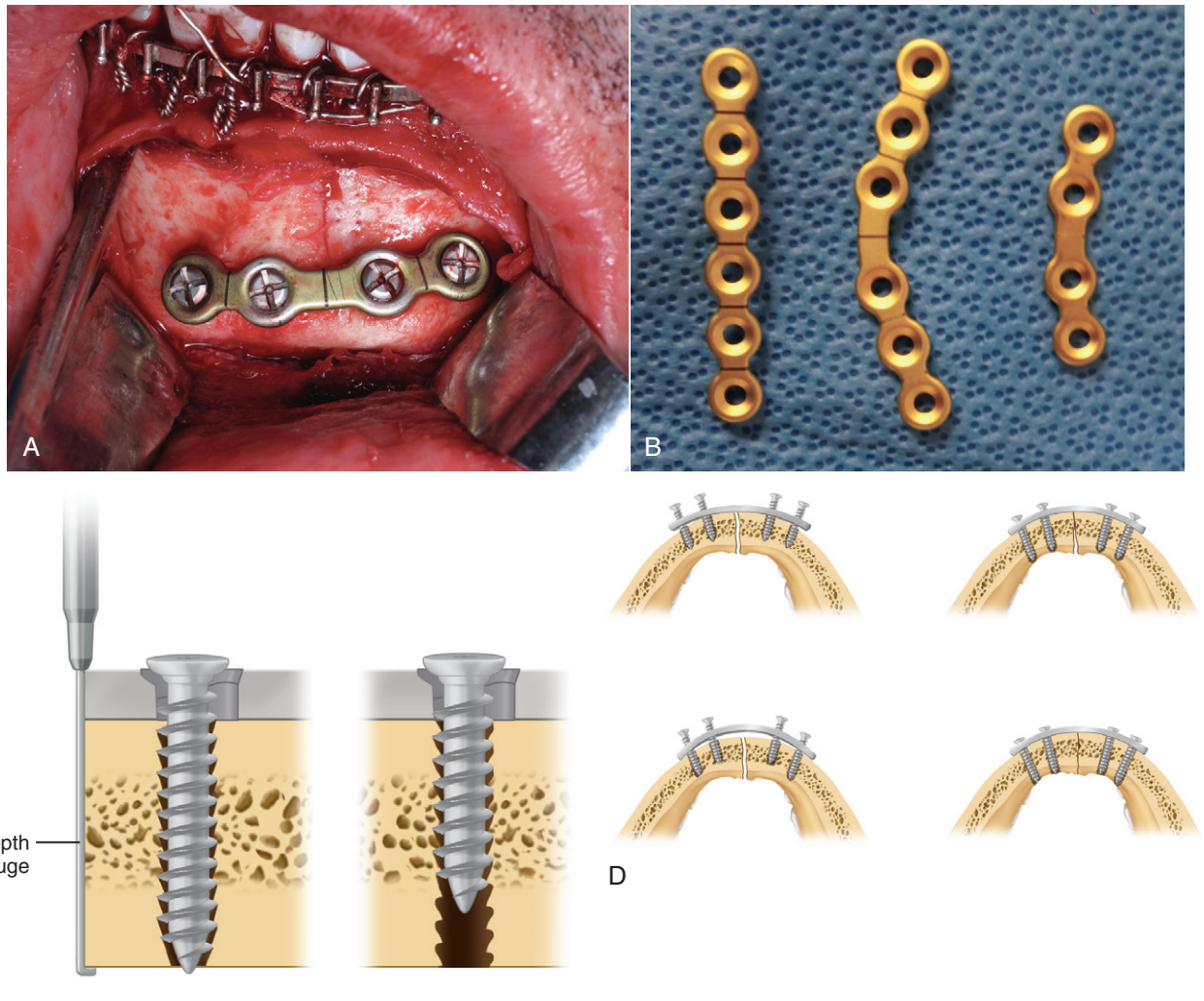


FIGURE 33-10 **A,** Four-hole fracture plate with bicortical locking screws used to fixate a mandibular symphysis fracture. The arch bar was left on as a tension band. **B,** Mandibular fracture locking plates of different designs. These particular plates accept 2.0- and 2.3-mm locking and nonlocking screws. **C,** A depth gauge should be used after drilling screw holes to ensure proper bicortical screw selection. The figure on the *right* shows a bicortical osteotomy with a poorly selected screw that only engages the outer cortex. **D,** Slight overbending of fracture plates can prevent lingual splaying of fracture segments as screws are tightened. Note the lingual gap when the plate is contoured to the bone surface without overbending.

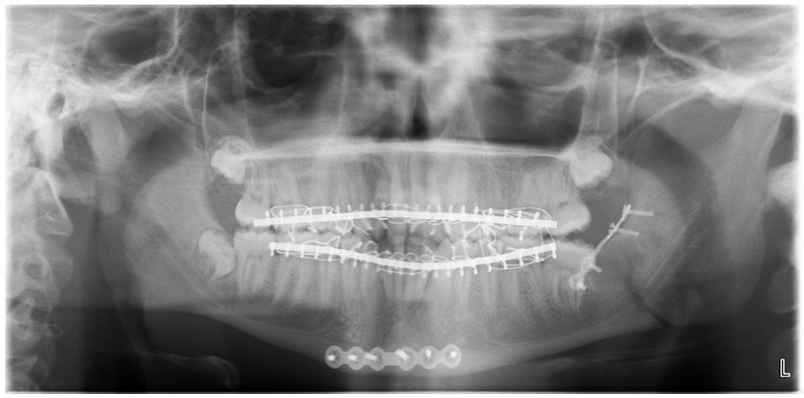


FIGURE 33-11 Postoperative orthopantomogram of a miniplate used in the Champy method for a mandibular angle fracture.

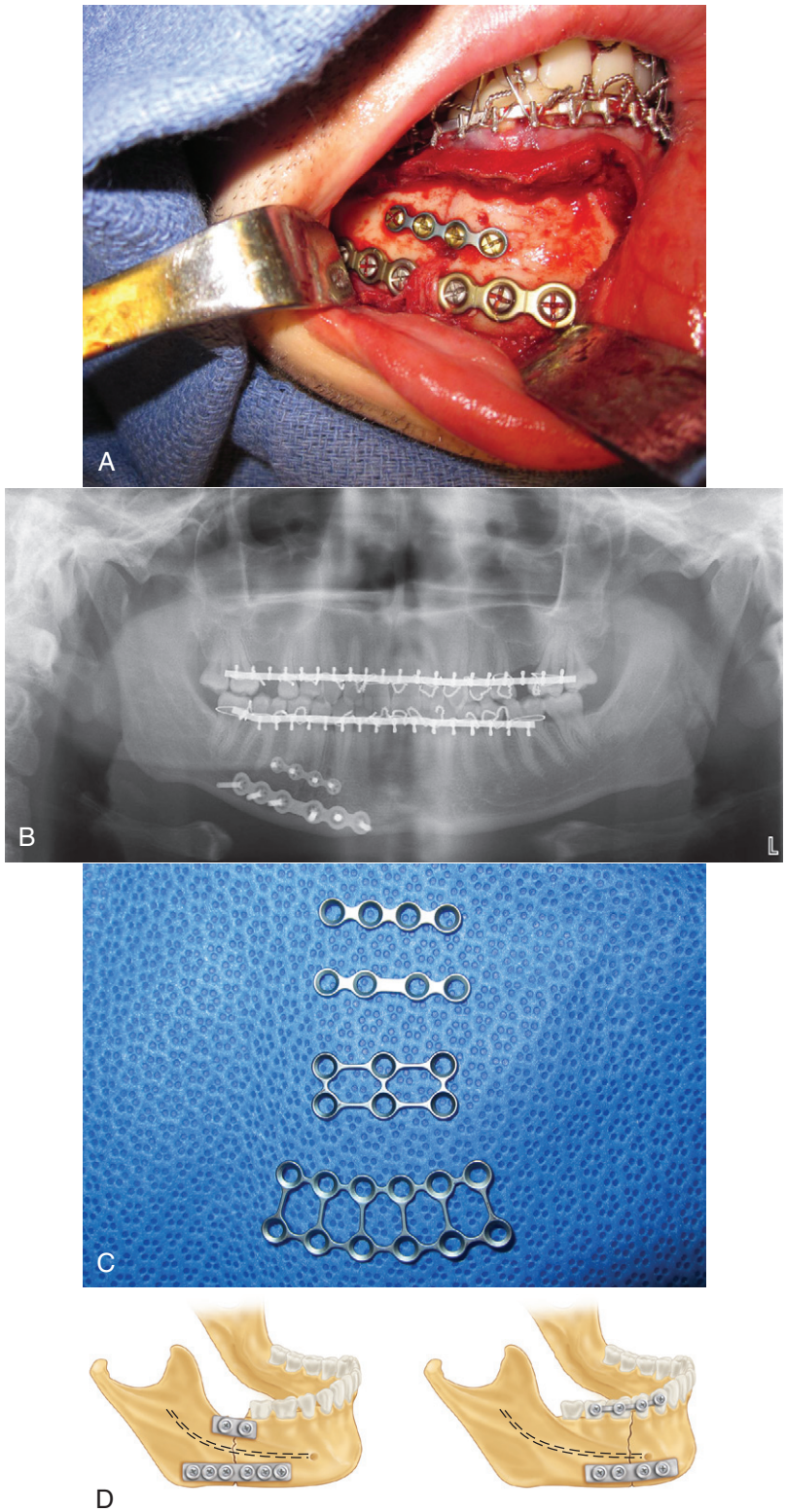


FIGURE 33-12 **A,** Four-hole miniplate with 2.0-mm monocortical screws used as a tension band to augment the stability of the fracture plate. **B,** Postoperative orthopantomogram showing position of the miniplate in relation to the dentition. Note that screws are angled away from tooth roots. **C,** Miniplates are available in several shapes and configurations. **D,** Use of a superior border fracture plate (Left) or bonded dental brackets and wire (Right) as tension bands.

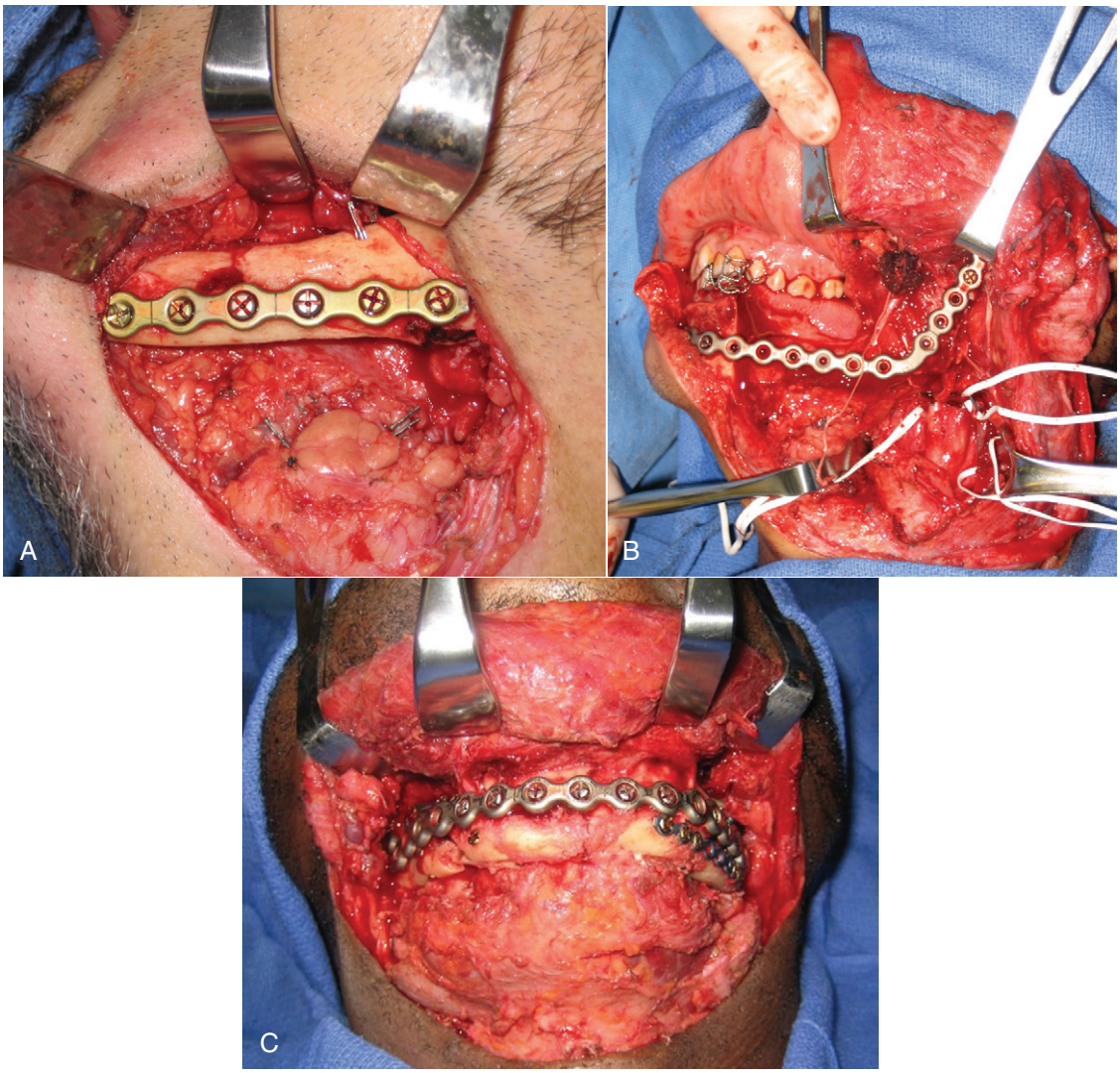


FIGURE 33-13 **A,** Low-profile reconstruction plate to fixate edentulous atrophic mandible fracture. **B,** Heavy reconstruction plate used to bridge a segmental defect. In this case, the plate is load-bearing. **C,** Heavy reconstruction plate in combination with miniplates and lag screws to fixate a severely comminuted mandible fracture secondary to a gunshot wound.

RECONSTRUCTION PLATES

Mandibular reconstruction plates are thicker and have a longer span than fracture plates, and are designed to be load bearing to span gaps and defects. In addition to reconstructing mandibular defects with or without bone grafts, reconstruction plates can be used to treat mandibular fractures that are comminuted, atrophic, or grossly unstable. When used to span a gap, four screws should be placed on each side of the defect to allow the plate to bear the most, if not all, the load of the mandible (Fig. 33-13).

Due to their size and thickness, reconstruction plates frequently pose problems in adapting to the mandible. Fortunately, the built-in locking mechanism can circumvent the need for perfect adaptation to bone. Unlike fracture plates or miniplates, which both allow for some degree of malleability when secured with bone screws, reconstruction plates exhibit a high degree of elastic deformation. If a reconstruction plate is not perfectly adapted to bone and is thoroughly tightened down with a nonlocking bone screw, bone segments can easily be distracted from their desired positions, resulting in malocclusion or segment splay. The locking mechanism proves to be essential in large spans with complex contours for which perfect plate adaptation is not possible. Rigid fixation with reconstruction plates is a reliable method, with relatively low complications in reconstructing mandibular defects with or without comminution.[34,35]

LAG SCREWS

Lag screw osteosynthesis is highly effective and efficient when used in the proper setting and eliminates the need for plate bending and the use of several screws. Lag screw osteosynthesis is a fracture compression technique that can be carried out by using true lag screws or a lag technique with long bone screws (Fig. 33-14). The classic application of this technique is in fixation of transverse mandibular symphysis and parasymphysis fractures[36] or

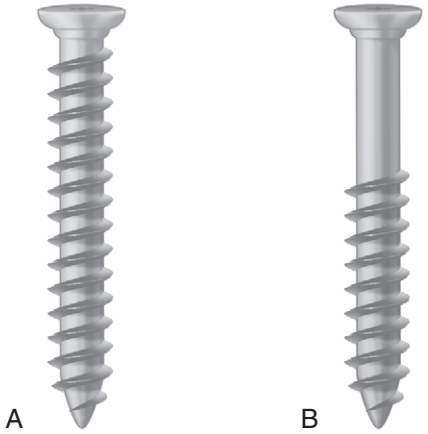


FIGURE 33-14 True lag screws have a smooth shank segment that does not engage the proximal cortex, allowing threading of the distal cortex and subsequently compressing it proximally.

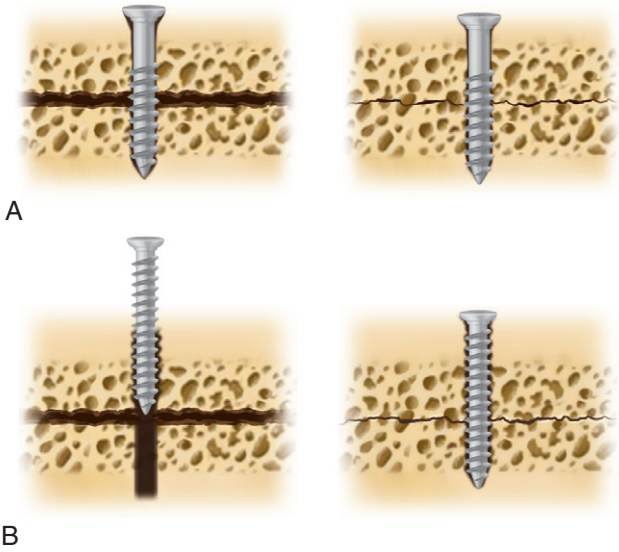


FIGURE 33-15 When using a true lag screw, the nonthreaded portion does not engage, resulting in compression of the segments across the screw. If using a standard full thread screw in a lag technique, the proximal segment must be overdrilled so that the screw can fit passively without the threads engaging the near segment. The distal segment is engaged by the screw threads.

obliquely oriented body and angle fractures. The premise of this technique is its ability to engage and pull, or lag, the distal cortex toward the proximal cortex across a fracture. This method provides a high degree of fracture compression, resulting in very stable fixation (Fig. 33-15). If done properly, this can be completed faster than with compression or noncompression plate osteosynthesis, with fewer postoperative complications.[37-39]

Unlike plate osteosynthesis, lag screw osteosynthesis directly traverses the fracture line, more evenly distributing compressive forces between segments and resulting in excellent stability and minimal to no lingual splay. After the fracture is exposed and reduced, a gliding hold is prepared from the near-cortex to the fracture close to the inferior border. This hole is of a larger diameter than the screw to be used to ensure that it does not actively engage this cortex. Next, a long drill guide is inserted into the glide hole and, using a drill of smaller diameter than the screw threads, the osteotomy is completed from the fracture line to the distal cortex. This is the traction portion of the osteotomy. The entire drill hole must not only traverse the fracture and both cortices, but should be perpendicular to the fracture. A depth gauge is used to measure the distances between the cortices and the appropriate screw length is selected. The near-cortex entry site may also be countersunk to allow for more broad distribution of the forces from the screw head because a large amount of compression will be generated. A long bone screw or true lag screw (a screw in which the proximal portion of the shank is unthreaded) is inserted passively through the gliding hole and purchased into the traction hole. When completely tightened, the engaged distal cortex will be drawn proximally and create compression at the fracture line. This process should be repeated with a second screw or second method of fixation to prevent rotation around a single axis (Figs. 33-16 and 33-17). When treating transverse or sagittal fractures of the symphysis, the screws should be placed through the outer cortex on either side. In oblique fractures, it may be necessary to engage the outer cortex proximally and inner cortex distally.

MIDFACE AND UPPER FACE FIXATION

Unlike the mandible, most of the facial skeleton is not subject to major muscular forces. The zygoma is the only other bone that displays significant effects from the masticatory musculature.[9] This must be taken into account when fixating zygomaticomaxillary complex fractures. Complex craniomaxillofacial trauma involving the frontal sinus, orbits, naso-orbito-ethmoid (NOE) complex, zygomaticomaxillary complex, and maxilla are best managed with the use of miniplate or microplate fixation[40] (Fig. 33-18). Because the necessity for plate strength and stability decreases in midfacial fractures, one must take into account the adverse effects associated with overzealous fixation. Thin soft tissue and overlying skin encasing the orbital and nasal complexes requires low-profile plates to prevent show-through, palpability, or dehiscence. In these areas, microplates provide adequate fracture stability and preserve patient comfort and aesthetics more than larger plates.[41] Compared with the mandible, midface and upper facial bones are thinner and more fragile. It is important to take advantage of the facial buttresses in fixating fractures to achieve screw and fracture stability[42] (Fig. 33-19). Even with the pull of the masseter attachment at the zygoma, zygomaticomaxillary complex fractures can be managed with miniplate or microplate fixation at multiple points, with stable results. Controversy exists regarding the ideal number of fixation points for zygoma fractures, but most studies have shown that miniplates or microplates provide sufficient stability. Outcomes measured by malar stability, orbital volume changes, facial asymmetry, or nonunion have shown variability in studies advocating one, two, or three points of fixation[43-46] (Fig. 33-20). The contraction of the masseter

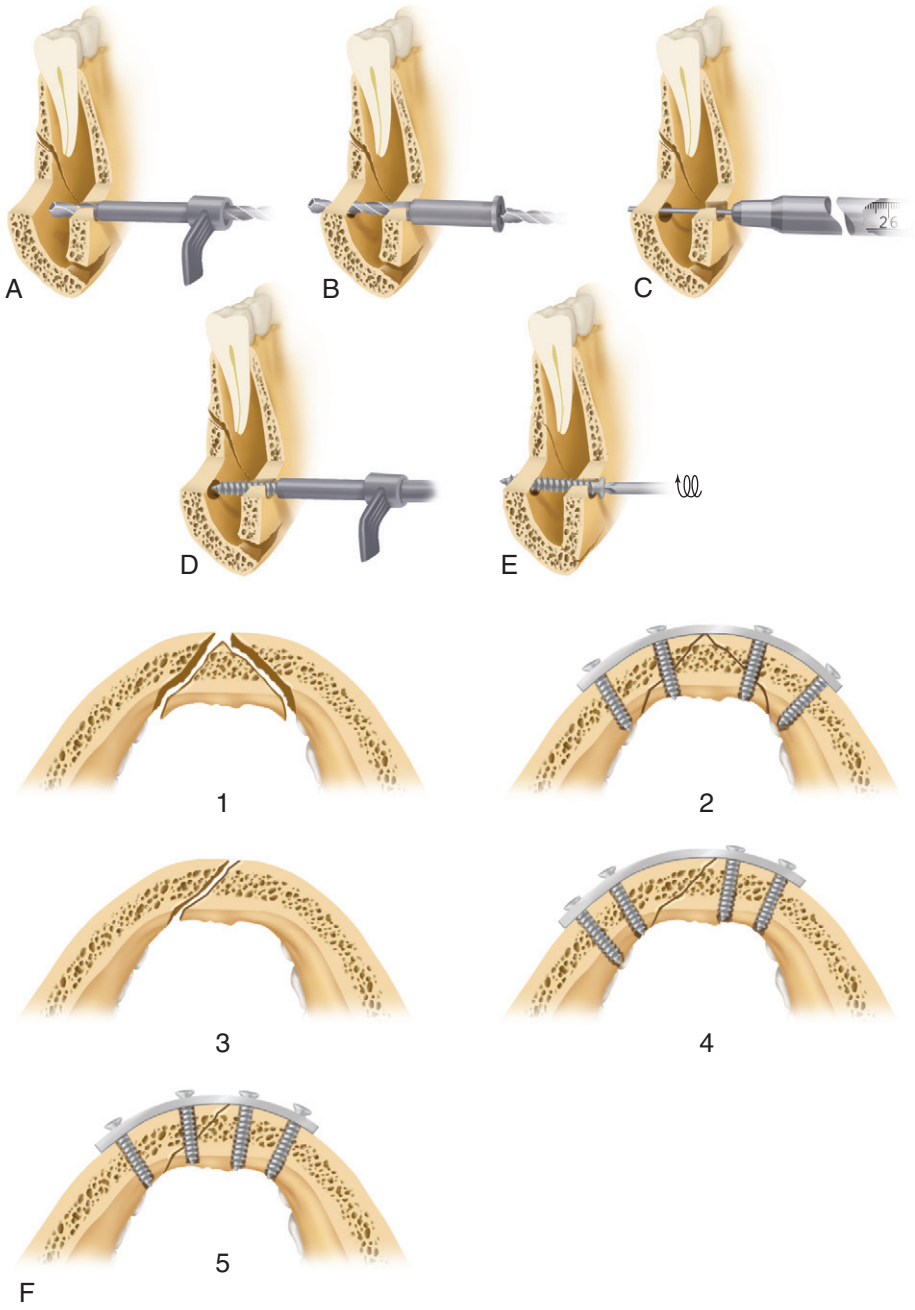


FIGURE 33-16 The steps in the lag screw technique. **A,** The outer segment is drilled to the fracture line with a large-diameter drill. The screw should fit passively in this segment, with only the screw head engaging the outer cortex. **B,** The inner or distal segment is drilled with a smaller diameter drill using a drill guide that fits the length of the first hole. The outer cortex is then countersunk with a special drill. **C,** A depth gauge is used to select the appropriate length screw. **D, E,** The screw is placed, resulting in compression. **F,** A combined approach with lag technique and plate osteosynthesis may be used in oblique or free-floating fractures. In fracture (1), the two proximal screws are engaged with a lag technique to draw the free lingual segment toward the outer cortex (2). An oblique fracture (3) may be treated with plate osteosynthesis (4) or combined with a lag technique (5). When using the combined technique, smaller length spans result between screws, reducing the amount of torsion across the plate.

muscle produces distracting forces at the zygomaticofrontal and zygomaticomaxillary sutures, both of which are important points of fixation, with adequate bone stock for screw stability. Increased points of fixation resist these forces but may or may not make a clinical difference.[47] Most studies have shown that it is not necessarily the method of fixation of zygoma fractures that makes them unstable, but the direction and degree of displacement.

BIOABSORBABLE PLATE FIXATION

With the advent of titanium internal fixation plates, rates of hardware infection and screw loosening have

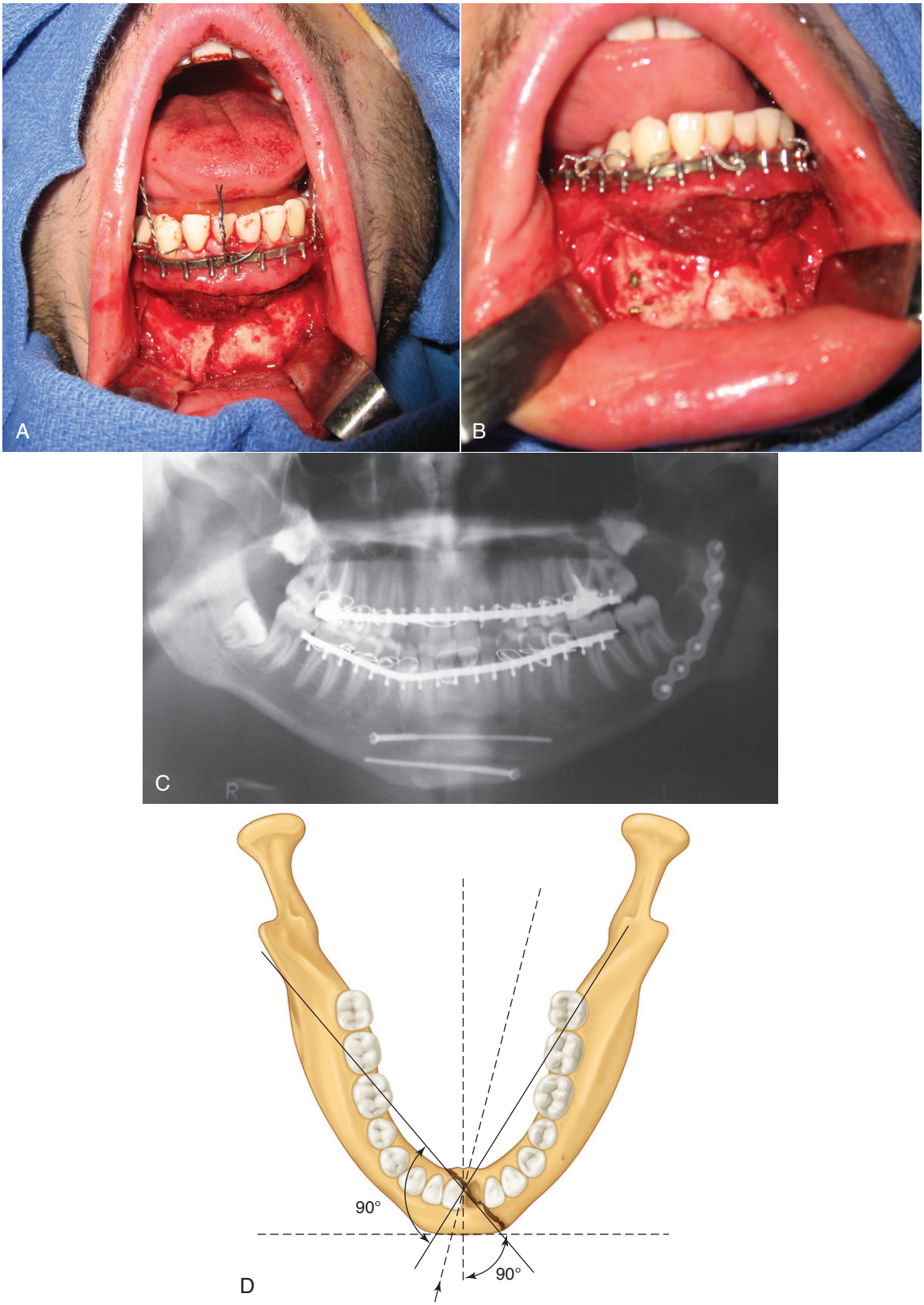


FIGURE 33-17 **A,** 22-year-old man with a transverse mandibular symphysis fracture. **B,** Application of two lag screws across the fracture. **C,** Postoperative orthopantomogram demonstrating correct hardware placement and compression of the fracture. **D,** To maximize the benefits of lag screw fixation, screws should be placed at an angle that bisects the lines perpendicular to the fracture and perpendicular to the bone surface where the screw will enter.

significantly declined when compared with less biologically compatible metals. Even so, late hardware failure requiring operative removal is still an active issue in maxillofacial trauma and subjects patients to additional risks associated with secondary surgery. The ideal implant should be completely biocompatible and resist infection, but as a nonbiologic alloplast, titanium is still subject to these effects. Unlike the surrounding bone, metal implants do not remodel or undergo angiogenesis. Frequently, surgical sites surrounding metal plates develop a hypovascular fibrous envelope, diminishing immune cells from migrating to the area and clearing bacterial contamination. If the plate becomes inoculated with microbial pathogens, an infection may ensue, creating inflammation, necrosis, and hardware loosening. In some European countries, it is commonplace to remove hardware routinely once stable bone healing is complete to prevent late infection, plate palpability, translocation during osseous growth, and/or impingement of dental prostheses. The advent of bioabsorbable fixation devices negates the need for hardware removal and can prevent many complications associated with long-term retention of permanent hardware.

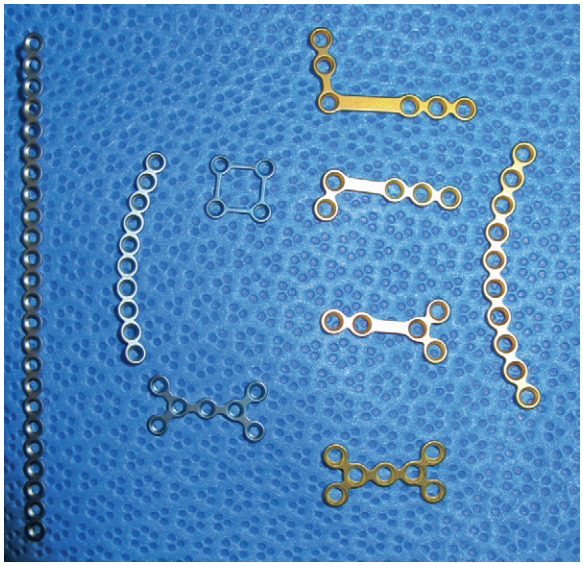


FIGURE 33-18 Microplates used for upper and midfacial fixation. The gold plates accept 1.7-mm screws and are best used for midface fractures; the blue plates accept 1.2-mm screws and are designed for upper face fixation. These plates are available in varying degrees of thickness and malleability.

Bioabsorbable implants were initially developed and used for pediatric craniofacial surgery in 1996, but have been described in the literature as early as 1971 for application in the facial skeleton. Traditional titanium plates are notorious for migrating into growing bone and can impinge on vital structures, depending on for which anatomic site they are used. Several reports of intracranial plate migration during bone growth drove the development of nonpermanent implants.[48,49] The advantage of a resorbable system for pediatric fractures lies in absorption of the plate in vivo before it can translocate to an unfavorable area. It has also been hypothesized that nonresorbing metal plates can restrict growth of the surrounding bone, leading to facial osseous hypoplasia and developmental defects. Bioabsorbable systems have been used and studied extensively in pediatric craniofacial surgery.

There are several varieties of bioabsorbable materials; the most modern are permutations of a polylactic acid and/or polyglycolic acid polymer. Differences in lactic-to-glycolic acid ratios, molecular weights, and polymerization mechanics may account for variability in resorption rates and handling characteristics. Polylactic acid resorbs

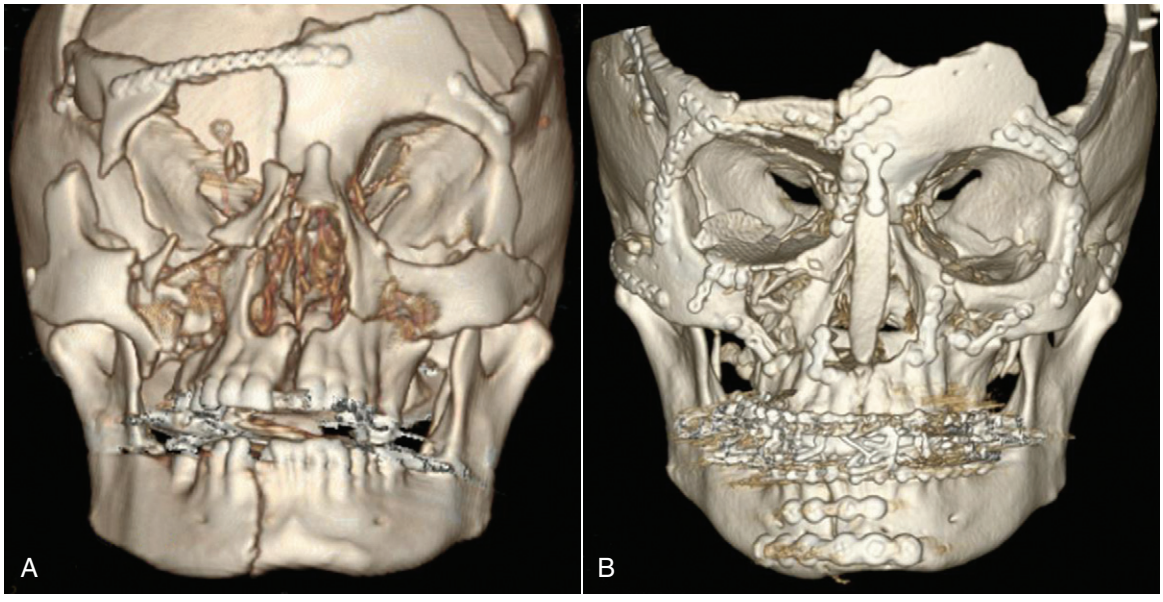


FIGURE 33-19 **A,** 40-year-old woman status post–six-story fall, sustaining head trauma and multiple facial fractures. This three-dimensional CT scan shows reconstruction after the initial decompressive craniectomy. **B,** Postoperative CT scan demonstrating microplate fixation of the midface and upper face, with concomitant bone grafting. Note the plate positions and segment reduction across all the major facial buttresses.

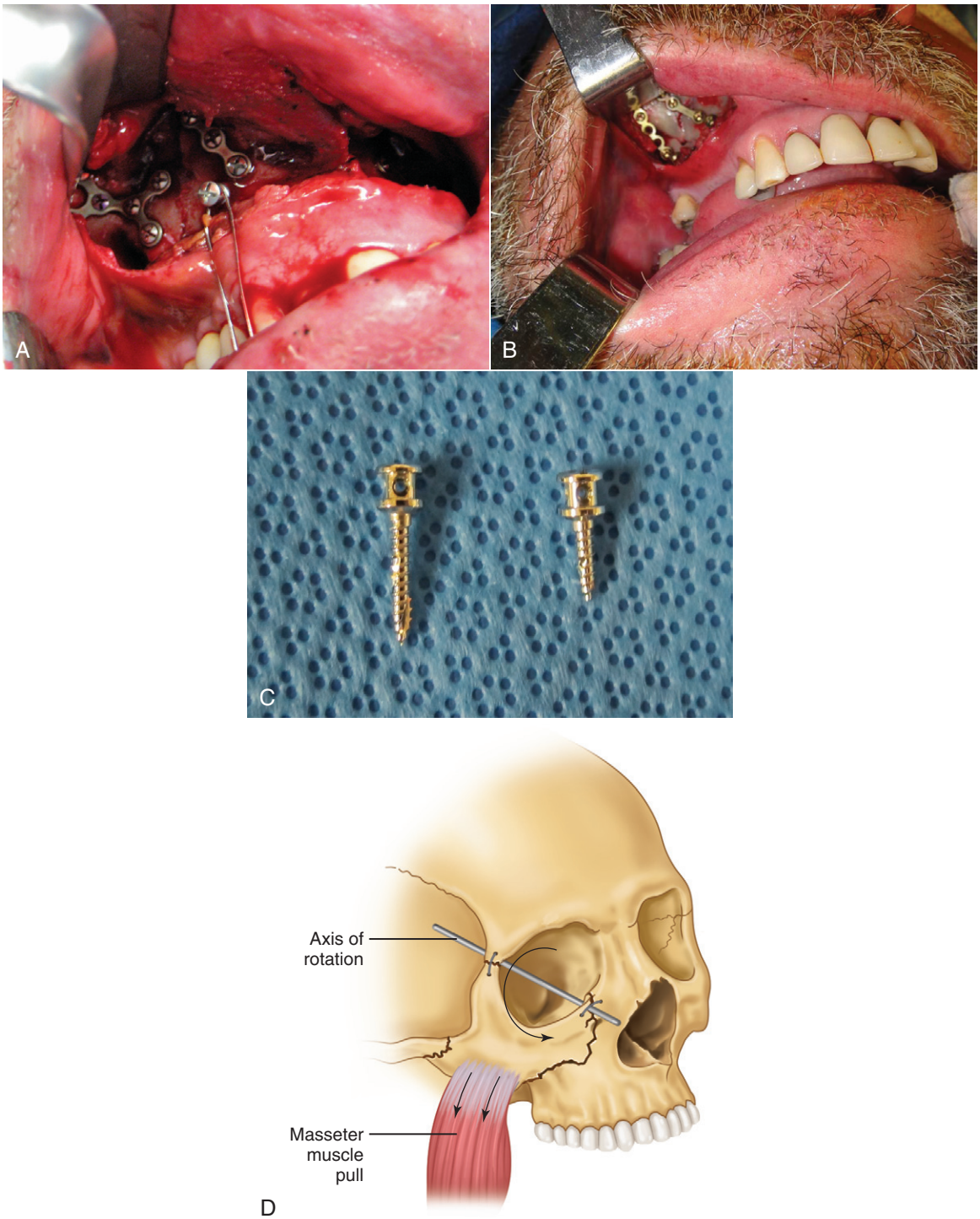


FIGURE 33-20 **A,** Microplate fixation of a combination of Le Fort I and zygomaticomaxillary complex (ZMC) fractures. Reduction and fixation across the zygomaticomaxillary and piriform buttresses provides maximum stability with the use of small hardware. Note that MMF was achieved with MMF screws. **B,** Microplate fixation of the zygomaticomaxillary buttress of a comminuted ZMC fracture with linear plates. **C,** MMF screws. Each screw has a hollow head design with slots in the same direction of the driver cross in which wires can be passed. **D,** Effects of the masseter muscle on ZMC fractures.

into lactate and takes longer than the hydrolysis of polyglycolic acid into carbon dioxide.[50] Reported resorption rates for these materials range from 12 to 36 months, as described by manufacturers, but many reports indicate that these plates can be palpated past the 3-year mark. The most commonly reported complications associated with this technique include not only plate palpability, but foreign body reactions, effusions, and infections.

A 5-year, multicenter prospective-retrospective study conducted by Eppley et al[51] have analyzed 1883 craniosynostosis patients younger than 2 years of age undergoing surgery with fixation by poly-L-lactic polyglycolic copolymer plates. Various devices composed of the same compound were used, including plates, meshes, threaded screws, and threadless push screws. Much of this depended on surgeon preference and evolution of hardware types from the manufacturer. They found device-related complications in 0.5% of patients; 0.3% of patients required reoperation.

Ahmad et al[52] have examined 146 cases of cranial vault reconstructions treated with LactoSorb plates in patients ranging from 2 months to 16 years of age. Outcomes similar to those of previous studies were analyzed, including plate palpability, wound infection, and wound healing. Six patients had palpable plates, two patients had palpable screws, and five patients developed surgical site infections over the course of a 1-year follow-up period.

As noted, bones of the upper and midfacial skeleton do not experience significant effects of muscle forces, allowing them to be fixated with minimal hardware. From a pure strength and stability standpoint, bioabsorbable hardware is inferior to titanium.[53] In cranial vault surgery, these differences have not been shown to be clinically relevant because hardware failure from stress and strain has not been reproducibly demonstrated. Most large-scale studies have shown that complication rates of bioabsorbable fixation are less than or equal to those with metal fixation. The mandible, however, poses entirely different problems. AO principles dictate that fracture stability is essential for proper bone healing, which is mostly dependent on the type of fixation.

Controversy exists regarding postoperative complications, infection rates, and hardware failure in the use of bioabsorbable implants for mandibular trauma; much of this may be attributed to operator technique, variations in hardware composition, and surgical patient population. Polylactic acid and polyglycolic acid plates, on average, provide half the strength of a traditional bicortically fixated bone plate across a fracture. In the mandible, this can produce negative outcomes. Although more large-scale clinical trials and long-term follow-up are needed to address the use of bioabsorbable plating in the lower face, many studies have shown rates of infection, bone union, and complications to be on par with titanium fixation. Proper case selection is essential to avoid adverse outcomes.[52,54]

COMPLICATIONS OF INTERNAL FIXATION

Modern methods of internal fixation provide many benefits over closed reduction of fractures but, as in all surgical procedures, complications exist. Many of these are exclusive to internal fixation and care must to be taken to minimize these risks.

SURGICAL SITE INFECTION

Surgical site infection is multifactorial in nature and has patient- and surgeon-dependent factors. Surgical site, access, type of hardware, technical errors, fracture mobility, and medical comorbidities are all elements that contribute to postoperative infection.[55] Fractures of the mandibular angle seem to carry the highest rate of infection regardless, of fixation technique. This may possibly stem from decreased bone to bone contact as compared with other mandibular sites. Teeth present in fracture lines without pathology or dental injury that do not impinge on fracture reduction and fixation may be left in place without increasing the risk of infection.[56] Various approaches to facial bone fractures have not been consistently shown to have significant differences in infection rates, but combined transoral-transfacial approaches to a single mandibular fracture site may increase the overall complication rate.[57]

There is no consensus on the use of antibiotics for fracture surgery. The use of perioperative versus extended antibiotics after fracture surgery has not shown any statistical differences in infection rates in some groups,[58] but others have reported that single-dose or single-day antibiotic therapy is superior in regard to infection rate reduction when compared with controls.[59] Many authors have questioned the need for postoperative antibiotics after internal fixation of the fracture, advocating that patient factors are more important in the development of surgical site infection.[60]

Application of excess hardware that does not change the clinical stability of the fracture may also be associated with increased complication and infection rates.[61] Severely contaminated open fractures tend to have higher rates of infection due to gross colonization of pathogenic bacteria. Poorly adapted plates or screws placed with weak purchase prevent adequate compression of fractures, increase interfragmentary mobility, and eventually may lead to hardware failure and site infection. Screw holes should be drilled linearly under copious irrigation to prevent widening and thermal necrosis.

Medical comorbidities associated with suppressed immune function include diabetes mellitus, acquired immunodeficiency syndrome, malnourishment, and chronic alcoholism, which lead to an increase in overall infection rates.[62] Hypovascularity of tissues secondary to multiple surgeries or radiotherapy prevent adequate immune proliferation at the surgical site and should be taken into account; medical optimization prior to surgery may be useful in reducing the risk of infection.

When infections do occur after fixation, the administration of antibiotics, with or without surgical débridement, irrigation, and hardware removal, may be necessary (Fig. 33-21).

DENTAL INJURY

In fractures of the maxilla or mandible, proper preoperative imaging can prevent iatrogenic tooth injury. Dental structures are usually damaged by misguided drilling or

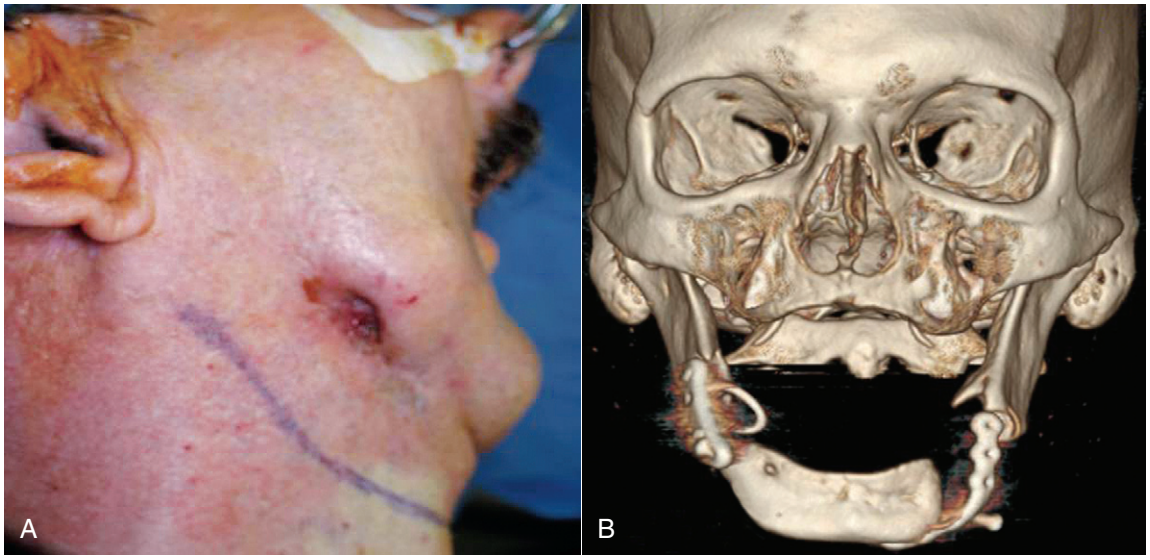


FIGURE 33-21 **A,** 55-year-old man with diabetes mellitus and chronic alcoholism presenting 2 years status post– open reduction and internal fixation (ORIF) of bilateral mandibular fractures and complaining of purulent drainage from bilateral neck. Note the orocutaneous fistula from infected plates. **B,** CT scan shows chronically infected hardware and extensive osteolysis, resulting in a free-floating anterior segment.

malpositioned screws. Bicortical fixation of the mandible should occur at the inferior border to avoid tooth roots and monocortical fixation of the maxilla and mandible should be placed just apical to the apex of teeth. In situations in which bone height is diminutive, it may be necessary to secure screws just between tooth roots by approximating their location. Dental radiography or computed tomography (CT) can be useful for measuring distances between teeth to prevent drilling into a tooth. If placing a screw is likely to damage a tooth, other methods of fixation can be considered to prevent this, including a single load-bearing inferior border plate or using an arch bar as a tension band. Placing arch bars may also damage periodontium and result in extrusion or avulsion of teeth. Care should be taken to place interdental steel wire apical to the heights of contour of the dentition. Passing wires between embrasures should be completed without macerating gingiva and may be facilitated by placing a gentle curve at the end of the wire. When ligating it to the arch bar, the vector of force should be applied apically and parallel to the long axis of the tooth to prevent iatrogenic luxation or avulsion.

NERVE INJURY

The inferior alveolar canal harbors the neurovascular bundle that supplies the mandibular dentition and soft tissues of the lip, chin, and associated gingiva. As noted, this is in the neutral zone of the mandible and should be avoided to prevent postoperative paresthesia, dysesthesia, or anesthesia. It is important to note that the canal runs approximately 2 mm inferior and 2 mm anterior to the mental foramen. Bicortical fixation should follow the inferior border and tension bands should be secured just above the neutral zone to avoid the inferior alveolar nerve. In transoral approaches to the mandibular body and parasymphysis, the mental nerve should be identified and protected throughout the operation. Full-thickness incisions to bone should be avoided in the premolar area until the main trunk of the mental nerve is identified from the subperiosteal pocket by gentle dissection. Once located, it can be protected with an instrument while the remainder of the mucosa is incised above it. Skeletonization of the mental nerve branches may be required to slide a plate below the foramen to adapt to the inferior border. Aggressive retraction in this area may also produce traction injury, resulting in neuropraxia or axonotmesis. Excessive retraction may also avulse the mental nerve from its foramen, resulting in permanent sensory deficits. Postoperative sensory deficits following internal fixation of the mandible have been reported at widely variable rates, likely due to differences in the surgeon's experience and technique.[63,64] Similar precautions should be taken when fixating maxillary, inferior, and superior orbital rim fractures to avoid damage to the infraorbital and supraorbital nerves.[65]

MALOCCLUSION

As described earlier, establishing the correct premorbid occlusion prior to the osteosynthesis of jaw fractures is essential in maintaining correct three-dimensional relationships of the teeth and bones. Appropriate measures should be taken to establish a stable occlusion prior to osteosynthesis. This may require the application of arch bars and MMF. Once established, accurate bending of plates and application of screws perpendicular to the plate can prevent splaying at the alveolus. Distraction of segments by poorly bent plates or overtightening bone screws in a poor sequence can result in a malocclusion. Even the slightest of discrepancies can be detected by the patient and result in patient dissatisfaction, TMJ disorders, parafunctional dental habits, and damage to teeth and periodontium secondary to misdirected occlusal forces. Malunion, or misalignment, of the jaws, and nonunion will result in a malocclusion. Surgeon

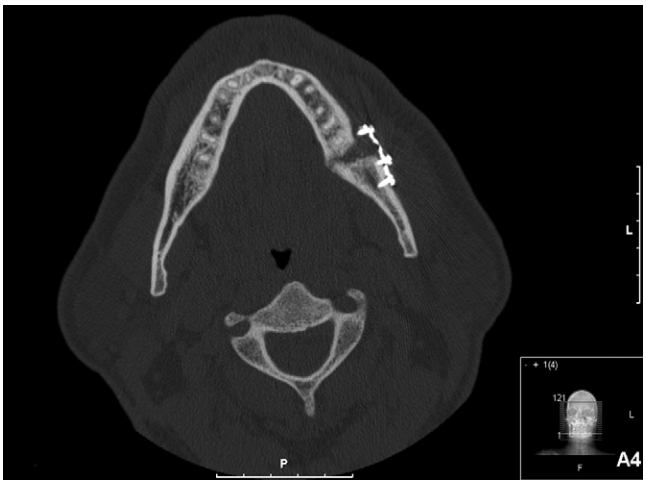


FIGURE 33-22 9-month post-ORIF CT scan showing nonunion of a mandibular body fracture, likely secondary to placement of a screw directly into the fracture line.

inexperience and postoperative infections are usually implicated in this result.[66]

Nonunion of bone segments across a fracture can be the result of poor fracture stability and interfragmentary movement. Large bone gaps may be infiltrated with fibrous soft tissue and also prevent osseous ingrowth. Compromised vascular supply secondary to aggressive periosteal stripping, soft tissue trauma, or irradiated tissue may prevent the necessary growth factors, nutrients, and oxygen from reaching the fracture site. This holds especially true for comminuted fractures. Postoperative infections should be managed in a timely manner to prevent devitalization of bone and subsequent nonunion (Fig. 33-22).

It is imperative to examine patients closely physically and radiographically for 4 to 8 weeks following fracture treatment. Gross fracture mobility may indicate a nonunion, which may require reoperation or closed reduction of the fracture. If not detected early and left untreated, nonunion may be associated with osteomyelitis, which may require extensive IV antibiotic treatment, surgical débridement, and delayed reconstruction.[67]

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CHAPTER

34

Minimally Invasive Oral and Maxillofacial Surgery Trauma

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OUTLINE

Endoscopic Treatment of Subcondylar Fractures
Extraoral Approach
Endoscopic Intraoral Approach
Endoscopic Treatment of the Frontal Sinus
Surgical Technique
Outcomes
Summary
Endoscopic Repair of the Zygomatic Complex Fracture
Surgical Technique
Discussion
Endoscopic Repair of the Orbital Floor Fracture
Surgical Technique
Summary

Minimally invasive endoscopy in oral and maxillofacial surgery has evolved significantly over the past several decades. Many patients have benefited from this technology, because they often experience less pain and swelling, shorter hospital stays, and fewer overall complications when compared with the standard maximally invasive procedures. With improved instrumentation and surgeon experience, many new, less invasive techniques and approaches have been developed for treating a broad scope of maxillofacial surgical procedures, such as orthognathic surgery, temporomandibular joint (TMJ) surgery, sialoendoscopy, facial cosmetic surgery, and trauma.

The management of maxillofacial trauma has benefited significantly from these advances. There are well-described minimally invasive approaches to the ramus condyle unit, thus allowing for repair of most subcondylar fractures. Treating orbital floor fractures, frontal sinus fractures, and zygomatic complex fractures with endoscopy have also been described. There are many advantages using these techniques over standard procedures involving open reduction. In this chapter, we describe these advantages as well as the indications, techniques, and outcomes of treating maxillofacial trauma patients with minimally invasive techniques.

ENDOSCOPIC TREATMENT OF SUBCONDYLAR FRACTURES

Condylar fractures are very common, making up almost one third of all mandibular fractures. There is extensive literature on the management of condylar fractures, which is a subject of significant controversy.[1-3] In general, there are three treatment modalities for the management of condylar fractures—observation, closed reduction with some period of maxillomandibular fixation (MMF) and physical therapy; and open reduction and internal fixation (ORIF). Most patients with condylar fractures have a satisfactory outcome measured by mandibular motion with nonoperative management.[4] The overall complication rate for treating subcondylar fractures by closed reduction or observation is only near 7%.[2] Not surprisingly, the vast majority of condylar fractures are treated in this manner. Anatomic reduction is rarely achieved with this approach, and post-treatment TMJ function is often dependent on adaptation of the altered condylar morphology with physical therapy, neuromuscular training, and guiding elastics.[5,6] Nevertheless, there are several instances in which ORIF is the preferred treatment, as described by Zide and Kent[7], and later by Haug and Assael[8] in their reports regarding indications for the open reduction of condylar fractures. Most agree that significant condylar displacement and ramus height instability are the two main indications to treat subcondylar fractures via an open approach.[9] In addition, many surgeons supporting open reduction cite decreased treatment and rehabilitation time, improved anatomic reduction,[10] consistent occlusal results,[11] and preservation of facial symmetry.[12] Furthermore, patients appreciate not having to have their jaws immobilized with MMF.

The topic of open versus closed reduction of subcondylar fractures remains controversial. However, when open reduction has been decided, options include using the submandibular, retromandibular, or preauricular incision. All these approaches allow appropriate access to the subcondylar region, thus enabling proper reduction and fixation. However, these incisions do not come without risks, such as facial scars, intraoperative or postoperative bleeding, salivary fistula, infection, and temporary or permanent facial nerve injury.[13-16]

With these open approaches, the incidence of facial nerve injury reported is as high as 30%.[17] In Ellis et al's

review of the literature, less than 1% of the 455 patients who underwent open treatment of condylar fractures had permanent facial nerve injury, whereas 12% had transient weakness.[13] Furthermore, in this series of 93 patients with subcondylar fractures treated open, 17.2% developed temporary facial nerve weakness, with no cases of permanent weakness in patients that followed up. In this same series, 7.5% of patients developed wide or hypertrophic scars postoperatively.

Although rare, the risks for treating subcondylar fractures with standard ORIF are not benign. The benefits of ORIF in treating subcondylar fractures often outweigh these risks. Some surgeons have sought to minimize the risks of treating subcondylar fractures further by developing minimally invasive approaches to the ramus condyle unit. With the aim of minimizing the risks in treating subcondylar fractures with ORIF, while still benefiting from the advantages seen in rigid fixation, some surgeons have turned their attention to minimally invasive approaches to the ramus condyle unit.[18-20]

Treating subcondylar fractures with endoscopy provides the surgeon with excellent visibility, magnifying and illuminating the operative field. A smaller incision and less dissection are needed, resulting in less postoperative swelling, less pain, shorter hospital stay, quicker postoperative recovery, lower risk to the nerve and smaller inconspicuous scars.[21-23] Nevertheless, the surgeon must invest a significant amount of time to learn and master these techniques and the cost of equipment is often high.

Both intraoral and extraoral endoscopic approaches have been described for the treatment of subcondylar fractures. Advocates for the intraoral approach cite benefits such as lack of visible scars and low risk of facial nerve injury.[24] However, its use is limited for some types of subcondylar fracture, particularly the medially displaced or dislocated fracture.[25] The extraoral endoscopic approach has made the treatment of medial overriding and dislocated fractures much easier, with minimal risk for facial nerve injury and a small, 1.5-cm submandibular scar.[18] Here we describe the extraoral and intraoral endoscopic approaches for treating subcondylar fractures.

EXTRAORAL APPROACH

The extraoral approach described here was developed at the Massachusetts General Hospital (MGH) to gain access to the ramus condyle unit (RCU) for a variety of procedures, including vertical ramus osteotomy with rigid fixation for mandibular setback, condylotomy for condylar sag, high condylectomy, condylectomy, costochondral graft reconstruction, coronoidectomy, and open reduction and rigid fixation of subcondylar fractures. The technique used in all minimally invasive ORIF cases at MGH is the extraoral approach described below.[18,26]

Surgical Technique

Surface landmarks are first drawn on the skin, including the outline of the mandible and the clinically and radiographically identified fracture site. The patient is placed into maxillomandibular fixation (MMF) in the proper occlusion. A 1.5-cm incision is made one finger-breadth below the angle of the mandible, and then dissection ensues to the masseter muscle bluntly. The muscle and pterygomasseteric sling are incised with needlepoint cautery. Careful subperiosteal dissection occurs along the inferior border and a subperiosteal blind sweep is performed along the lateral surface of the mandible using a suction-assisted endoscopic elevator (Snowden-Pencer, Tucker, Ga), thus creating an optical cavity. The position of the incision and mobility of the soft tissue in this region allow the 2.7-mm diameter, 30-degree Hopkins endoscope (Karl Storz, Carver City, Calif) to be placed in the wound and oriented parallel to the posterior border with direct access to the entire RCU, thus allowing the surgeon to visualize and work on the area of interest en face.

Using the elevator, subperiosteal dissection continues as the anatomic landmarks of the RCU are identified, including the posterior border, anterior border, sigmoid notch, coronoid process, and condylar neck. A curved, long-handled retractor is positioned to maintain the optical cavity (Synthes CMF, Pa). Irrigation is performed through the endoscopic irrigation port.

With endoscopic visualization, the proximal and distal fragments of the fracture are identified and mobilized. In the case in which the fracture is laterally overriding, a 24-gauge wire is passed through a 1.5-mm drill hole at the mandibular angle to distract the distal segment inferiorly, enabling the reduction of the fracture. The proximal segment is reduced with the condylar neck head positioned in the glenoid fossa. The fracture is reduced and the distracted distal segment is released, wedging the two segments together. The reduction is verified by directly visualizing the fracture lines at the posterior border and sigmoid notch. The fracture is fixated using a 2.0-mm, five-hole titanium miniplate. The plate may be positioned into place with a plate holder, and screwed to the proximal fragment first, enabling the surgeon then to remove the plate holder and control the proximal fragment while aligning the distal segment for fixation. Reduction at the posterior border is verified and the distal screws are placed, either through the incision or with the aid of a percutaneous trocar (Figs. 34-1 and 34-2).

When the proximal segment is medially displaced, an attempt is made endoscopically to visualize the proximal segment, and manipulate it into the lateral overriding position. Typically, this is accomplished endoscopically (Fig. 34-3). If unsuccessful, an endoscopic vertical ramus osteotomy can be performed to permit access to the condylar fragment.[22]

Outcomes

At MGH, in a retrospective analysis of 20 consecutive patients with subcondylar fractures ($n = 22$ sides) who underwent endoscopic ORIF, 1 patient had temporary marginal mandibular nerve weakness at 1 week follow-up, which resolved several weeks later. There were no permanent postoperative injuries to the marginal mandibular nerve and no unacceptable facial scars were encountered.[18,26] In all cases, there were no cases of postoperative malocclusion and all patients had normal TMJ range of motion without pain. Miloro has found similar results in a series of six consecutive subcondylar fractures treated with extraoral endoscopic ORIF.[27]

Of the 20 patients in the series treated at MGH, 14 were treated initially with endoscopic ORIF and 6 were treated after failed MMF. Of these fractures, 14 were displaced and eight sides were dislocated. Four patients with medial displacement required a vertical ramus osteotomy with subsequent ex vivo removal and fixation of the proximal segment and condyle. In all cases, the RCU heights were restored and there were no abnormalities in healing, facial or trigeminal nerve injury, jaw motion, occlusion, facial asymmetry, or pain. None of the patients required MMF postoperatively and all were discharged from the hospital in less than 23 hours after the procedure.[26]

ENDOSCOPIC INTRAORAL APPROACH

Jacobovicz and Lee first described the intraoral endoscopic approach to subcondylar fractures in 1998.[28] This approach showed promising results and those who supported it cited benefits such as lack of visible scars and low risk of facial nerve injury. The approach has its limitations, particularly for the management of medially displaced subcondylar fractures.[29] It has also been shown to be technically challenging, requiring a steep learning curve, even for those experienced in endoscopic oral and maxillofacial surgery.[30] Since its initial description, there have been several modifications described with the aim of easing the technical challenges encountered.[26,31,32]

Surgical Technique

An intraoral incision is made in the posterior mandibular buccal sulcus, with subsequent subperiosteal dissection along the lateral ramus, angle, and posterior border of the mandible, thus creating an optical cavity for a 4-mm, 30-degree angle scope (Karl Storz, Tuttlingen, Germany). A percutaneous, 40-mm guarded trocar is placed transbuccally, perpendicularly at the level of the subcondylar fracture. The optical cavity is maintained by retraction with the guarded trocar, the endoscope is placed through the intraoral incision, and the proximal fragment of the

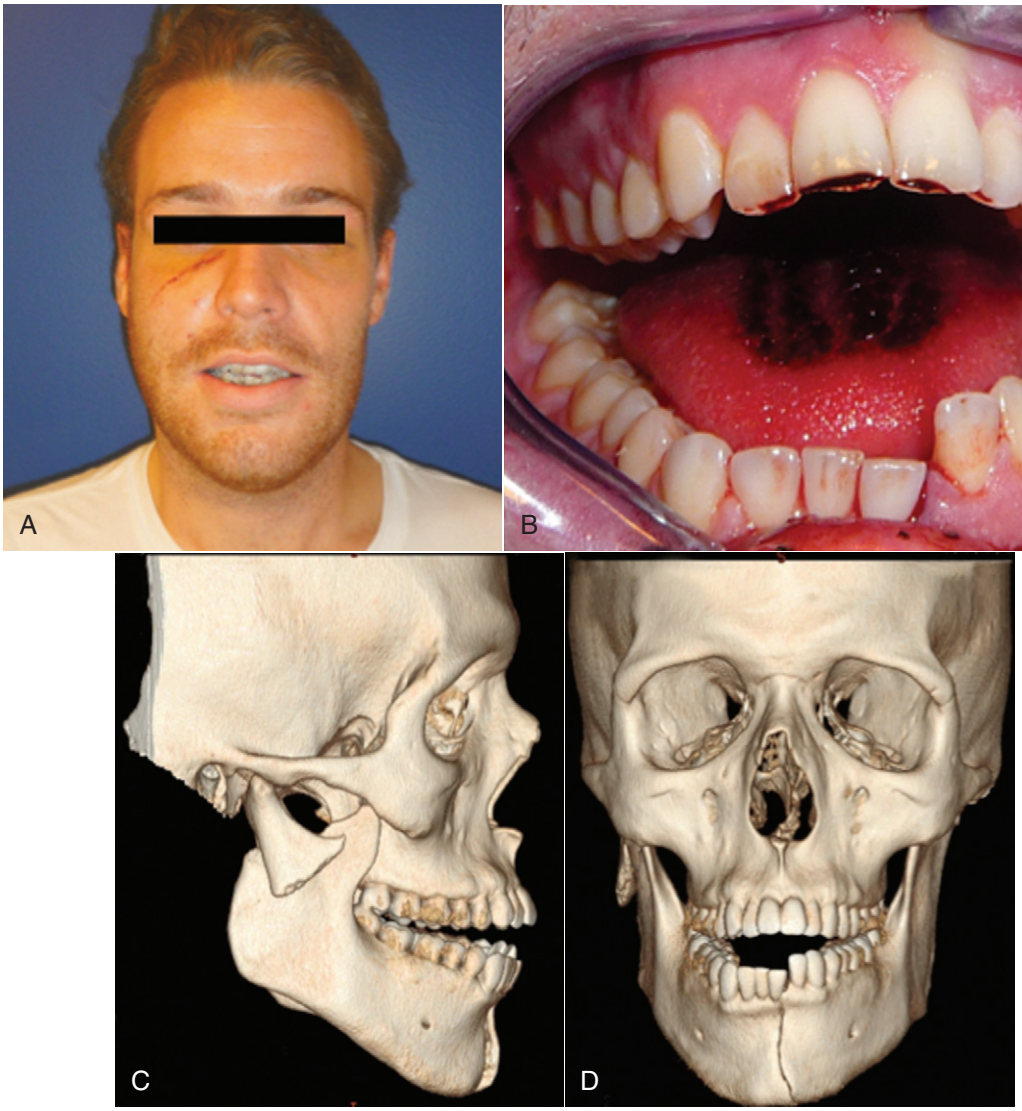


FIGURE 34-1 **A,** Right subcondylar fracture. **B,** Left parasymphysis fracture. **C, D,** Right subcondylar fracture with lateral override.

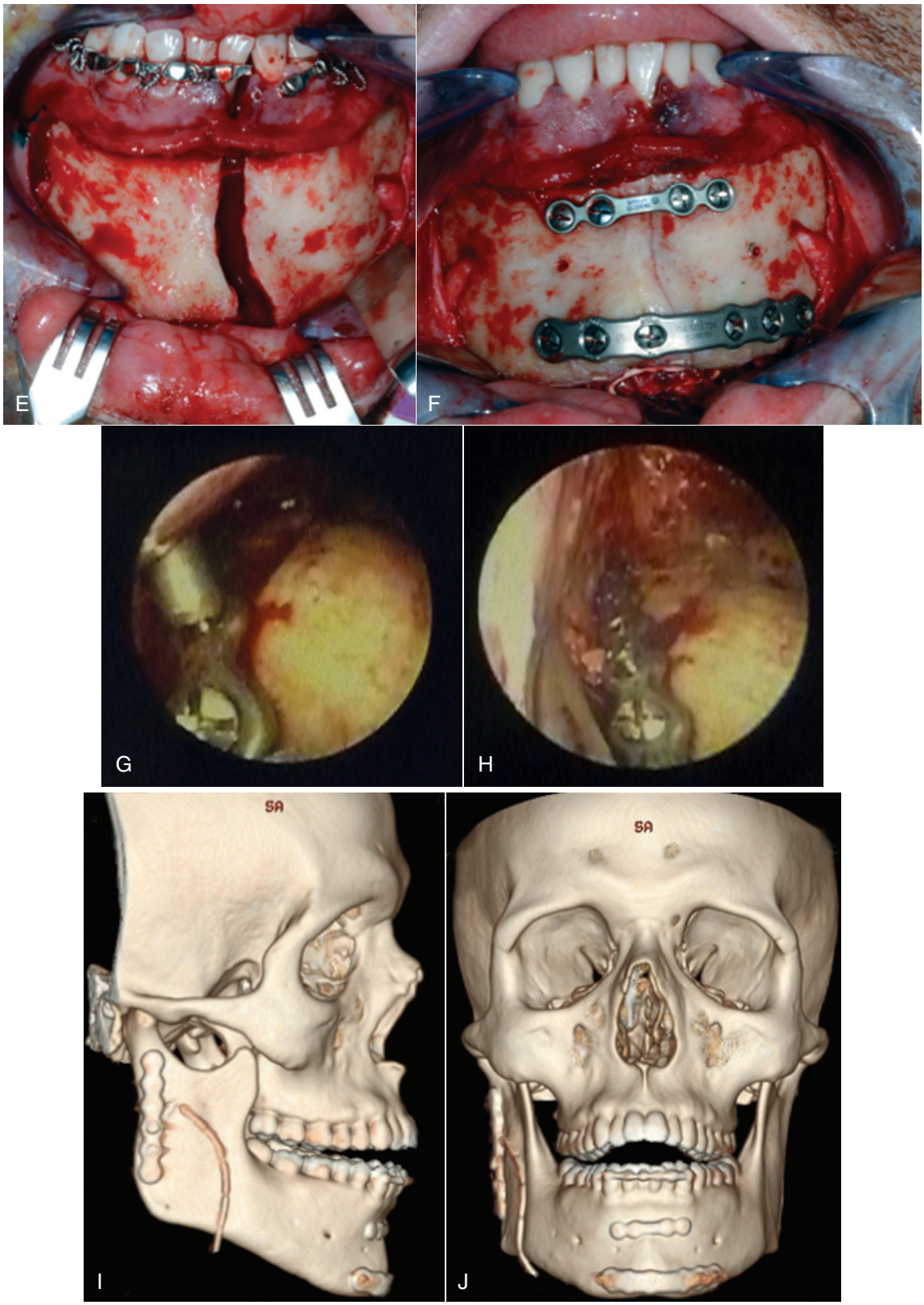


FIGURE 34-1, cont'd **E,** Displaced left parasymphysis fracture. **F,** Left parasymphysis fracture reduced + plated. **G, H,** Percutaneous screw placement with endoscopic guidance, with rigid fixation. **I, J,** CT scan, Immediately postoperatively.

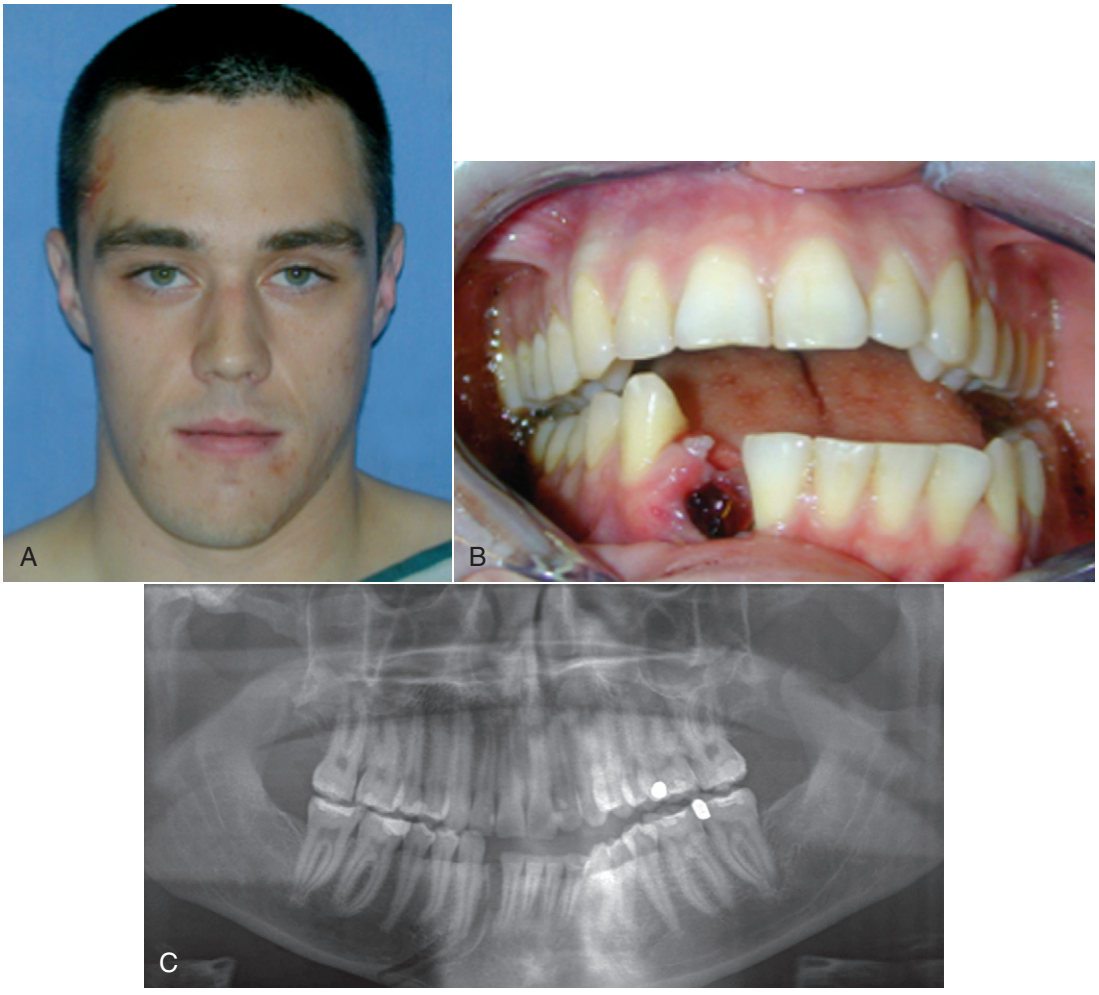


FIGURE 34-2 **A, B,** Right parasymphysis, left subcondylar fracture. **C,** Panorex, left subcondylar fracture, right parasymphyseal fracture.

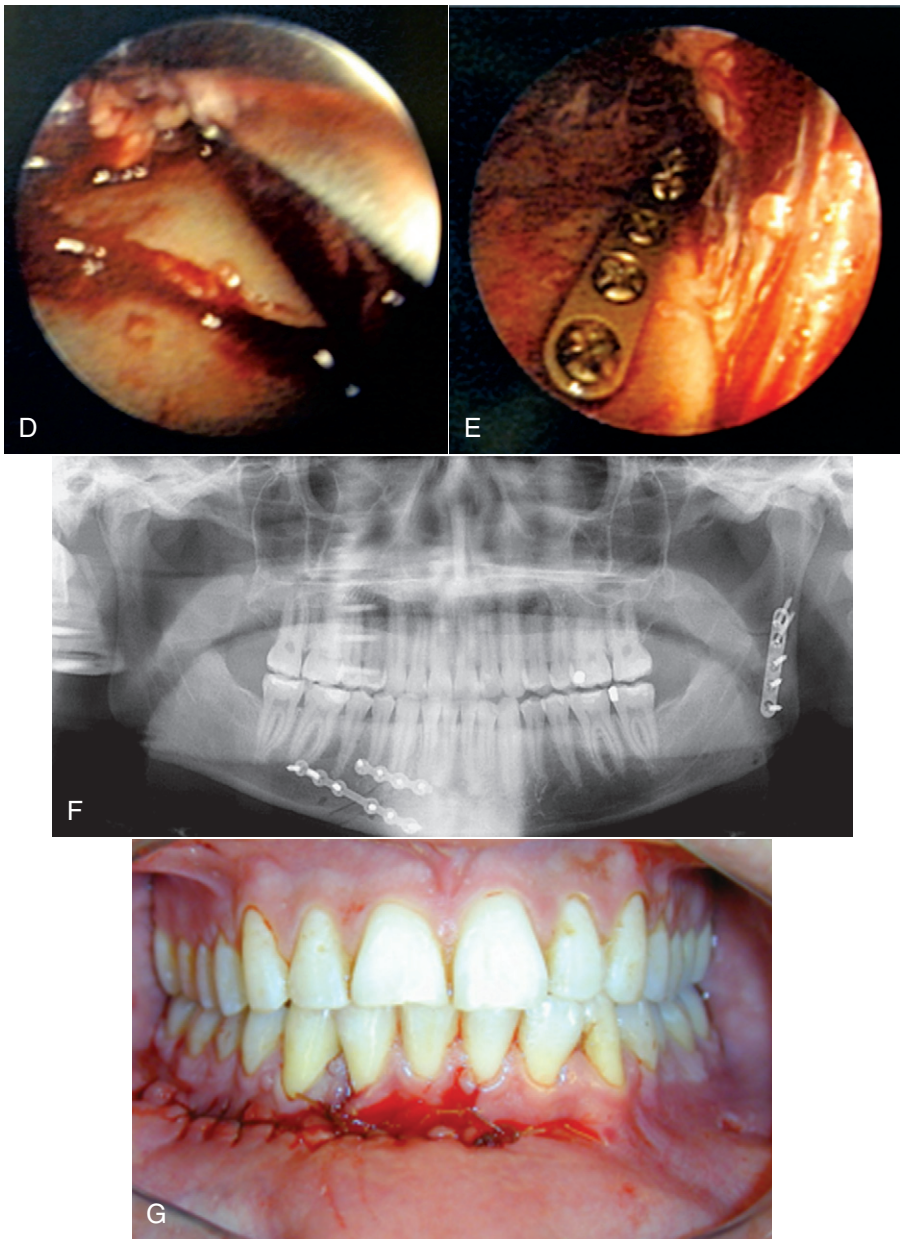


FIGURE 34-2, cont'd **D,** Left subcondylar fracture. **E,** Left subcondylar fracture with rigid fixation. **F, G,** Immediately postoperatively.

Continued



FIGURE 34-2, cont'd **H-M,** 6 weeks postoperatively.

subcondylar fracture is visualized and dissected under direct endoscopic vision (Fig. 34-4).

The patient is then placed into temporary elastic MMF, thus facilitating reduction of the fracture. The proximal segment can be reduced into position by applying a medially directed force against the lateral surface of the proximal segment with the trocar (Fig. 34-5). The anatomic reduction is verified with direct endoscopic visualization and positional screws are placed through the trocar for initial stabilization of the segments. A fixation plate is then used for definitive fixation. The patient is released from MMF postoperatively (Fig. 34-6).

Outcomes

With this technique, the authors confirmed good anatomic reduction, mandibular projection, lower face width, facial height, premorbid occlusion, and a maximal incisal opening of 39 mm without pain. Lee et al

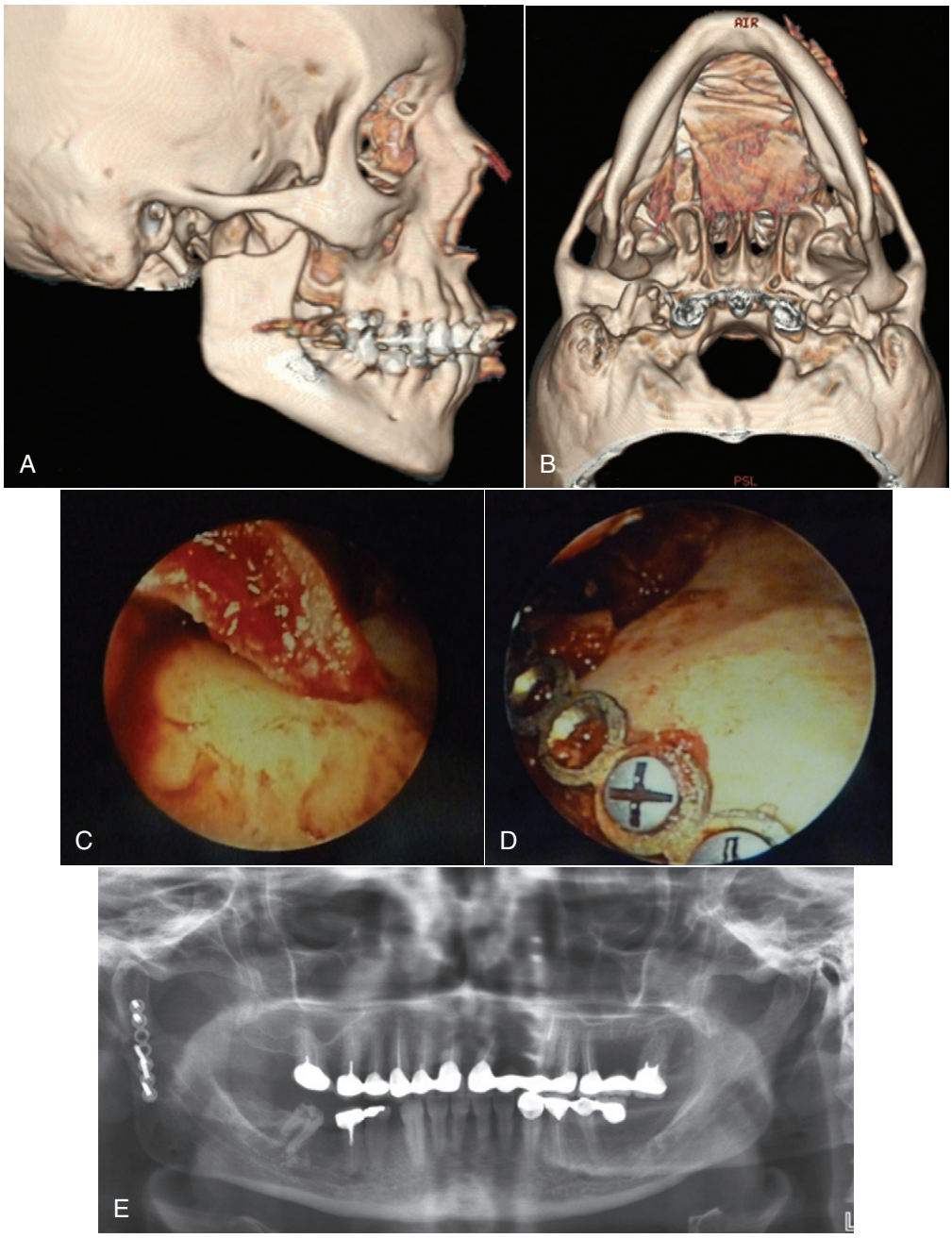


FIGURE 34-3 **A, B,** Right subcondylar fracture with medial override. **C, D,** Medial override converted to lateral override, with fixation after reduction. **E,** Right subcondylar fracture ORIF postoperatively.

followed this with a longitudinal study of 20 patients with 22 fractures that were repaired using this technique.[33] Functionally, all patients had restoration of their premorbid occlusion and an average maximal incisal opening of 43 ± 6 mm at 8 weeks postoperatively. All patients were pleased with the aesthetic results of the repair, including chin projection, jaw line, and symmetry; 21 of the 22 fractures showed radiographic evidence of fracture reduction. One patient with medial override showed an improved but imperfect reduction and there was no evidence of late condylar remodeling in any of the patients.

Operative times were also measured. Fractures with lateral override (20 of 22) were easily reduced and fixated endoscopically (131 ± 39 min). Medial override fractures (2 of 22) were challenging to repair and required a much longer operative time (269 ± 139 minutes)[33].

Since 1998, there have been several reports describing modifications of the intraoral endoscopic subcondylar fracture repair. Sandler used two separate trocars for improved reduction and fixation.[34] The first trocar was placed 20 mm anterior to the tragus along the canthal-tragal line and the second was placed 10 mm below the first site. In 2002, Schon et al used angulated drills and screwdrivers, eliminating the need for percutaneous trocar incisions.[35] Kellman described an additional 1-cm submandibular incision for easier viewing and reduction.[32] Chen et al used a bone clamp to distract the distal segment inferiorly for easier reduction and fixation.[36]

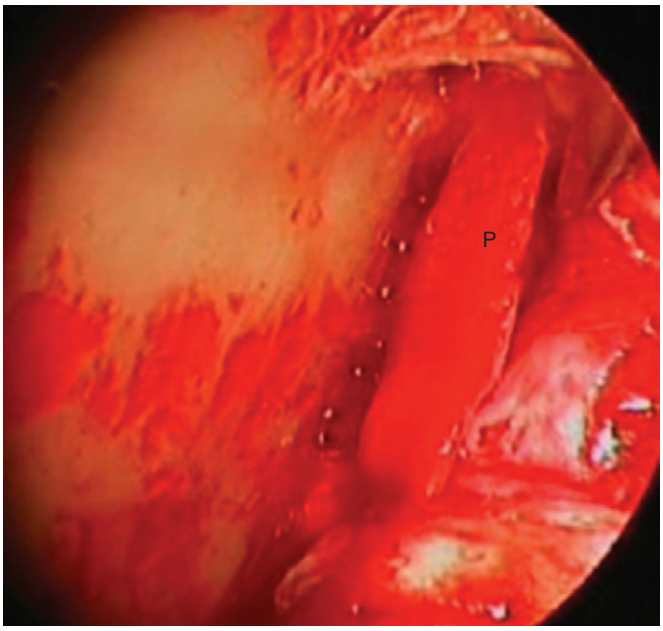


FIGURE 34-4 Via an intraoral incision, the endoscope is used to visualize the fracture and the proximal segment (P) is dissected.

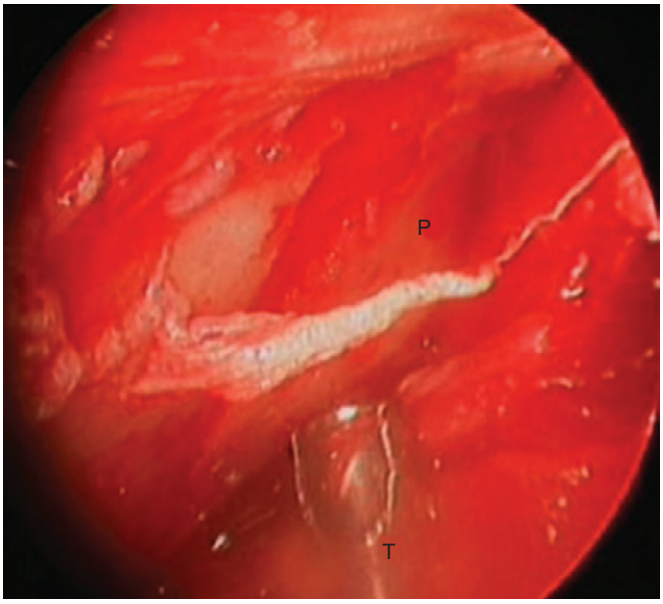


FIGURE 34-5 A percutaneous trocar (T) is used to help reduce the proximal segment (P).

In 2009, Schmelzeisen et al performed a randomized controlled trial with 34 patients who underwent traditional ORIF of subcondylar fractures and 40 underwent intraoral endoscopically assisted ORIF.[37] They evaluated the functional and cosmetic outcomes, operative time, and intraoperative and postoperative complications. Using the Helkimo dysfunction score, there was no statistically significant difference between the two treatment groups. The median operative time of the traditional ORIF group was 33 minutes faster than the endoscopic group. Two intraoperative complications for the endoscopic group included an anterior open bite that was fixed with a period of MMF and guiding elastics and

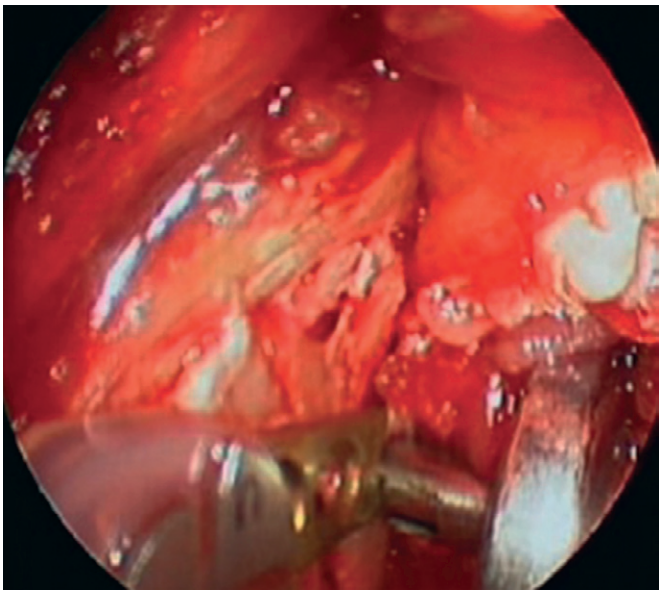


FIGURE 34-6 The plate is placed through the intraoral incision and the screws are placed through the percutaneous trocar.

unsatisfactory fixation of a miniplate secondary to osteoporotic bone. Facial nerve injury occurred in 10 ORIF patients and 5 endoscopic patients. A 50% recovery of facial nerve injury occurred in the ORIF group, whereas 80% recovered from the endoscopic group.

Discussion

The intraoral endoscopic technique for treating subcondylar fractures can be used for many simple subcondylar fractures, particularly with lateral override and minimal displacement. Advocates of the intraoral approach cite numerous benefits, including decreased facial nerve injury and lack of visible scars. It is clear from most of the literature that the technique can be extremely challenging, and even the most experienced surgeons have difficulty learning this technique[1,32,38] The intraoral approach results in viewing the fracture from a parallel view of the mandible rather than en face, making it challenging. It is also evident that when compared with the extraoral endoscopic approach, the intraoral endoscopic approach leads to more complications with medial override fractures. However, some authors still advocate use of the transoral endoscopic approach, even with medial override, and attribute the difficulty to surgeon training.[30]

With the extraoral approach, most patients easily accept a 1.5-cm submandibular scar and the risk of facial nerve injury (4.5%) is clearly lower than the traditional extraoral approach (30%), although not as low as the intraoral endoscopic approach (1.04%).*

Finally, some predict that the extraoral endoscopic procedure will eventually be performed in the outpatient setting with IV sedation, significantly decreasing operative time, cost, and manpower.

Both the intraoral and extraoral endoscopic techniques have their advantages and disadvantages, but

*References 19, 29, 30, 32, 34-36, and 38.

this should not distract from the ultimate goals of ORIF—return of premorbid occlusion, pain-free range of motion >35 mm, facial asymmetry, minimal scarring, and no postoperative facial nerve dysfunction.

ENDOSCOPIC TREATMENT OF THE FRONTAL SINUS

Frontal sinus fractures make up 5% to 15% of all facial fractures and are typically the result of high-velocity blunt force trauma to the maxillofacial area.[39] The frontal sinus is one of the thickest bones of the craniofacial region and can withstand almost 400 to 1000g of force before fracturing, twice the force tolerated by any other facial bone. Hence, head trauma, cervical spine injury, and other concurrent facial fractures are commonly seen in association with frontal sinus fractures, thus affirming the potential for injury severity. As such, the operative morbidity for repair of these fractures can be severe. Therefore, the importance of developing a less morbid, minimally invasive approach to repair these fractures is of interest.

The management of frontal sinus fractures has been well described and various algorithms have been developed to guide decision making for treatment.[40-42] Operative treatment depends on a number of factors, including the following: (1) nasofrontal duct involvement; (2) degree of anterior table depression; (3) degree of posterior table fracture; (4) neurologic status; and (5) cerebrospinal fluid (CSF) leak. Based on the degree and type of injury, treatment options may include observation, anterior wall reconstruction, osteogenesis, obliteration or exenteration of the sinus, and cranialization.

If operative management is necessary, a coronal incision is the typical approach used, although gull wing and open sky incisions have been described.[43] These are not without risks, because they often cause scarring, alopecia, facial nerve weakness, and paresthesia.[44,45] Minimally invasive endoscopic approaches to the frontal sinus have been developed with the aim of decreasing or eliminating these risks.[46] Some of these techniques were originally developed for use in brow and forehead lifts.[47] The severity and degree of involvement of the frontal sinus fracture dictate whether one can use the minimally invasive techniques that have been developed. If osteogenesis, obliteration or exenteration, or cranialization is required, the traditional approach is preferred. Only isolated anterior table fractures can be treated with endoscopy.[48] It has been reported that one third of all frontal sinus fractures are isolated to the anterior table,[49,50] although some have reported an incidence as high as 72.5%.[51] Operative interventions in isolated anterior table fractures often create a depression defect, which may be an aesthetic concern for some patients. This is a common indication for operative intervention in these fractures.

Treating frontal sinus fractures with endoscopy provides the surgeon with a magnified, direct visualization of the operative field. Smaller incisions with less dissection are required, which makes for an easier postoperative course, with less swelling and pain, shorter hospital stays, and smaller inconspicuous scars. The decision for an endoscopic approach depends on careful patient selection. The surgeon must invest a significant amount of time mastering the techniques, which pays off for the patient who benefits from its use.[26]

SURGICAL TECHNIQUE

The incisions for the endoscopic repair of frontal sinus fractures (isolated anterior table fractures) are similar to that of the endoscopic brow lift. Two 2-cm long longitudinal hairline incisions are made approximately 2 cm behind the hairline, one over the midline of the scalp and the other at the injury site (Fig. 34-7). In patients with a prominent forehead or receding hairline, the incisions should be closer to the hairline to allow easier visualization of the fracture around the curvature of the forehead. The incisions are made down to bone and a periosteal elevator is used to create subperiosteal pockets for each optical cavity. A blind subperiosteal dissection is performed through each incision for the first 3 to 4 cm. A 4-mm diameter, 30-degree Hopkins II endoscope (Karl Storz, Tuttlingen, Germany), with an external sheath for retraction of the soft tissue, is inserted in the midline incision (Fig. 34-8). Curved endoscopic dissectors are used to tunnel and connect each incision (Fig. 34-9). The tissue is elevated inferiorly to the supraorbital rim and the fracture is visualized directly with the endoscope. The bone fragments can then be elevated and repositioned into place. A stab incision can be made in the eyebrow region to help reduce the segments, if necessary.[26,46-48]

If the fracture is stable after reduction, fixation may not be necessary. Often, however, fixation is required for stabilization of the segments. After inserting miniplates through one of the port incisions, a percutaneous stab incision is used to pass screws, thus allowing the plates to stabilize the fractured segments (Figs. 34-10 to 34-12).

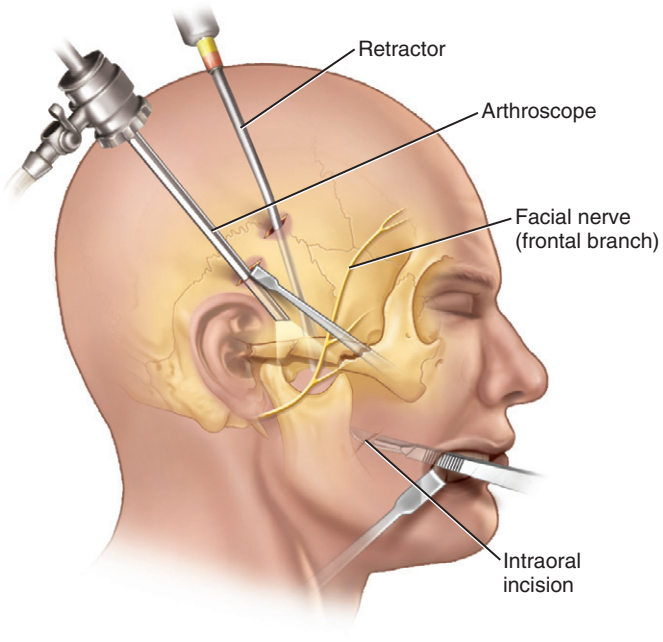


FIGURE 34-7 Incision lines (red) for an endoscopic approach for left side frontal sinus repair.

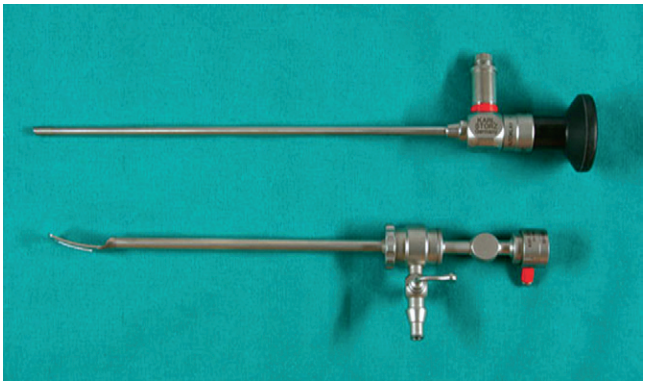


FIGURE 34-8 Endoscope *(above)* and sheathed retractor *(below).* (Courtesy Arnulf Baumann.)

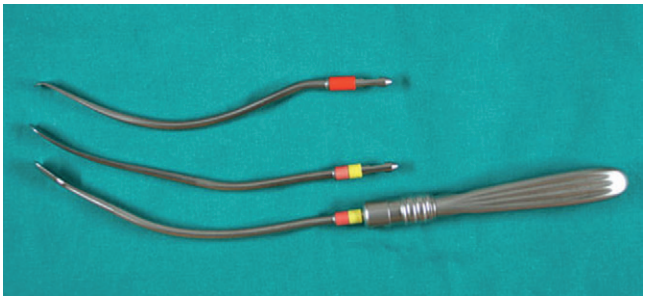


FIGURE 34-9 Curved dissectors. (Courtesy Arnulf Baumann.)

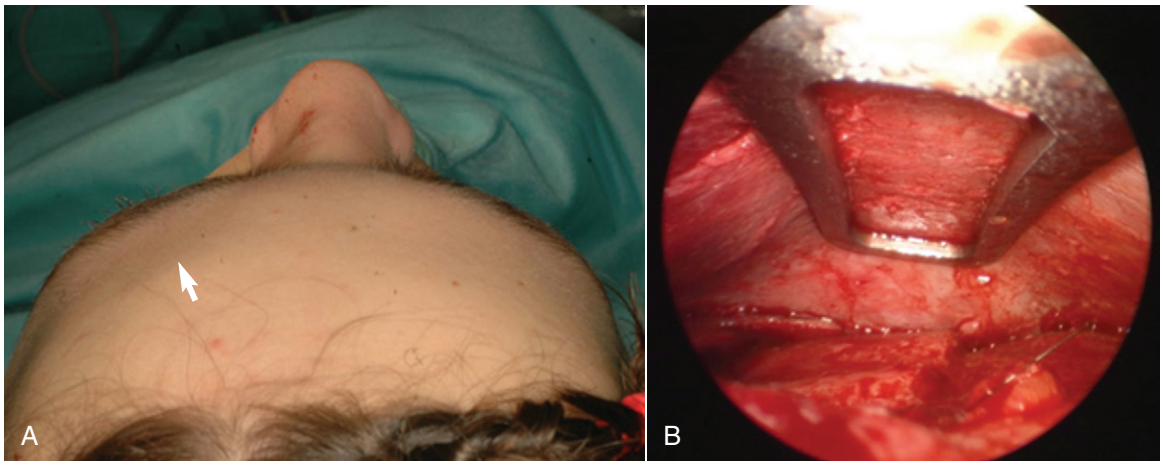


FIGURE 34-10 **A, B,** Depressed left frontal sinus fracture, with endoscopic view. (Courtesy Arnulf Baumann.)

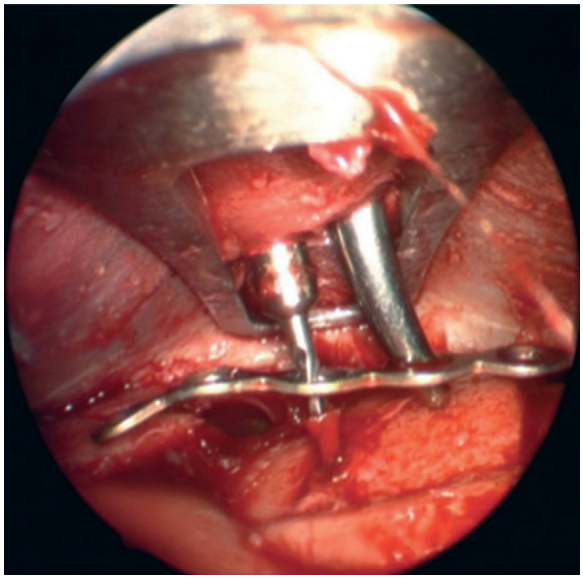


FIGURE 34-11 Miniplate held in place with a clamp. The drill is inserted through a percutaneous stab incision. (Courtesy Arnulf Baumann.)

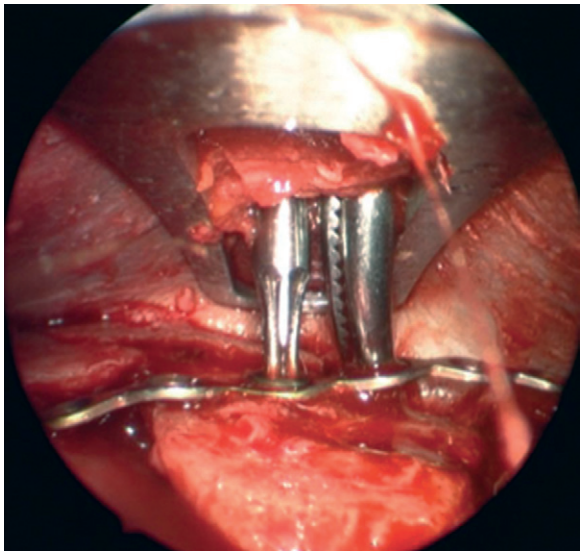


FIGURE 34-12 Percutaneous placement of screw to fixate the plate. (Courtesy Arnulf Baumann.)

In some cases, if the fractures are unstable after an attempt is made at endoscopic repair, or if great difficulty is encountered trying to plate the fractures, conversion to a standard open coronal approach may be necessary.

OUTCOMES

Since its initial description in 1996,[46] endoscopic frontal sinus fracture repair has evolved and there have been several modifications published. Hewitt et al[52] have described using a urethral sound to help reduce an isolated anterior table fracture. After making a small 2-cm Lynch incision, a 4-mm trephine burr was used to drill a hole to allow a 45-degree endoscope into the sinus. After visual examination with the endoscope, a urethral sound is placed through the trephine hole, maneuvered under the fracture, and pushed up to elevate the fracture while molding the fracture externally with digital palpation. The reduction can then be confirmed with endoscopic visualization.

Chen et al have described removing the reduced segments by an endoscopic grasper and used as free grafts.[53] The fragments are assembled on the table and fixated with miniplates, being sure to extend the miniplates beyond the segments so that they can be secured to bone in situ. The frontal sinus mucosa can be stripped off and the nasofrontal duct can be visualized with the endoscope. After the segments are assembled, it can be placed back through the wound and secured with percutaneous screws.

Even with these modifications, it is evident that isolated anterior table fractures with significant comminution are challenging to treat endoscopically and a coronal approach should be considered in these cases. Shumrick's series has shown successful endoscopic repair of anterior table fractures in 12 of 19 patients.[54] In the seven unsuccessful cases, the procedure required conversion to a coronal incision due to the degree of anterior table comminution.

SUMMARY

There is credibility for using minimally invasive techniques to treat frontal sinus fractures when indicated. These fractures must have minimal comminution and be isolated to the anterior table. If there is concern for nasofrontal duct obstruction or a CSF leak, the conventional coronal approach is preferred. Ideally, the fragments should be large and there should be a depression defect that is noticeable and of aesthetic concern to the patient. When indicated, the endoscopic approach for repairing frontal sinus fractures is a safe alternative to the conventional approach, with less postoperative pain and swelling and decreased risk for scarring, nerve injury, and alopecia.

ENDOSCOPIC REPAIR OF THE ZYGOMATIC COMPLEX FRACTURE

Zygomatic complex fractures are not uncommon and their treatment is dictated by the type and severity of the fracture. Nondisplaced fractures are typically treated with observation. Two major indications for the operative management of zygomatic complex fractures include facial asymmetry resulting from depression of the complex and/or trismus resulting from impingement of the coronoid process by the depressed arch. Depressed or rotated fractures can be treated with one or a combination of multiple approaches, including a maxillary vestibular incision, supratarsal fold incision, transconjunctival incision, and coronal incision. Historically, classification systems have been developed to guide the surgical approach based on fracture anatomy.[55,56] If the arch fracture is isolated, the conventional approach to treatment would be closed reduction with a Gillies[57] or Keen[58] approach. If the arch is comminuted and cannot be reduced with closed reduction, or if it is associated with a complicated ZMC fracture, a coronal incision has traditionally been the approach for performing ORIF of the arch.

The coronal incision has few limitations in terms of access, but it certainly is considered maximally invasive and can result in significant postoperative pain and swelling. The potential for scarring, alopecia, and paresthesia does exist.[45] Facial nerve injury is also possible, especially with zygomatic arch access, because a preauricular extension is required. Because of this, surgeons have been interested in minimally invasive techniques at repairing zygomatic complex and isolated zygoma fractures.[59]

SURGICAL TECHNIQUE

The endoscopic approach to the zygomatic arch typically involves a maxillary vestibular incision and two temporal incisions (Figs. 34-13 and 34-14). Preoperative mapping of the frontal branch of the facial nerve should be drawn, which is helpful particularly if percutaneous screw fixation is needed for arch fixation. The two scalp incisions are 2 cm in length and located behind the temporal hairline, superior and anterior to the helix of the ear. Dissection is carried out to the temporalis fascia and then continued bluntly to the temporal line of fusion, where the temporalis fascia splits into the superficial and deep layers. The superficial temporal fat pad is adherent to the superficial fascia but not to the deep fascia. Thus, the fat pad and superficial fascia can be reflected while

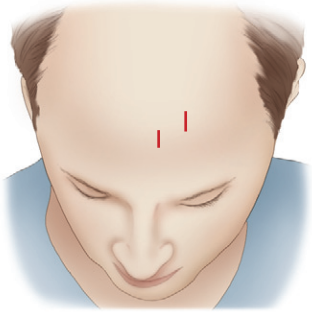


FIGURE 34-13 Schematic endoscopic approach to the right zygomatic arch with intraoral and hairline incision.



FIGURE 34-14 Endoscope inserted through temporal incision *(left),* with the curved dissector inserted from above. (Courtesy Arnulf Baumann.)

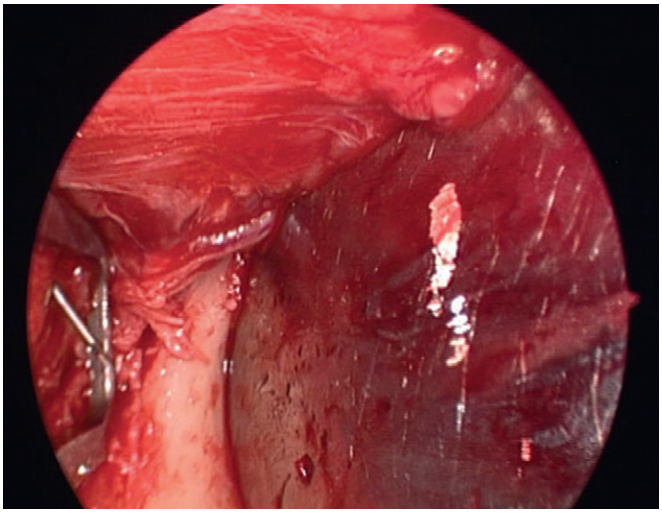


FIGURE 34-15 The zygomatic arch is exposed between the endoscopic retractor *(left)* and spatula *(right).* (Courtesy Arnulf Baumann.)

preserving the middle temporal artery, which is within the fat pad. This prevents bleeding during the endoscopic dissection. The superficial fascia and fat pad, and temporalis muscle if necessary, are reflected with the retractor. The periosteum of the zygomatic arch is then incised and the arch is exposed and dissected anteriorly (Fig. 34-15). An intraoral maxillary vestibular approach is also done with the endoscope, connecting the temporal incisions so that the arch is exposed with one tunneling dissection. Reduction and fixation is done with miniplates, using percutaneous screw placement with endoscopic visualization (Fig. 34-16).[59,62]

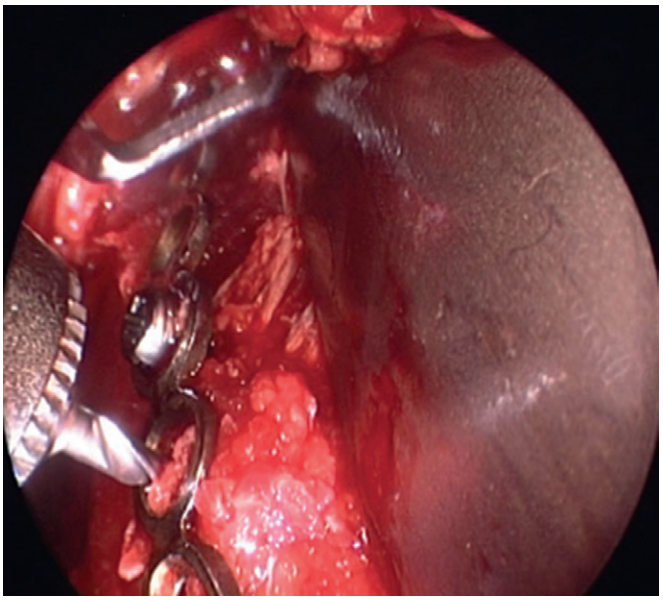


FIGURE 34-16 Reduction and fixation with percutaneous screw placement. (Courtesy Arnulf Baumann.)

DISCUSSION

Zygomatic complex fractures are typically repaired with multiple approaches. If the zygomatic arch requires open reduction, a coronal incision is typically necessary unless using minimally invasive endoscopic techniques. Lee et al[60] first showed an endoscopically assisted approach for repair of the zygomatic arch. In their series of 25 patients, a preauricular incision with a small temporal extension was used for endoscopic access. In 8 of 25 patients, there was temporary frontal branch weakness from retraction.

Chen et al[61] used a temporal approach for endoscopic repair of zygomatic arch and ZMC fractures. The dissection was superficial to the deep temporal fascia and of the 15 patients studied, only 2 developed temporary facial nerve weakness. The subfascial dissection, as described earlier, involves exposing the temporalis muscle directly, with the plane of dissection deep to the muscle. In Lee et al's study,[60] 25 patients had successful repair of their fracture without any facial nerve injury.

In conclusion, the endoscopic technique is a minimally invasive alternative to treating isolated arch fractures or comminuted ZMC fractures without the use of a coronal incision. The risk of facial nerve injury is low if the deep subfascial dissection is used.[62] With further advances and surgeon experience, new modifications to this approach will surely improve the results in repairing these fractures.

ENDOSCOPIC REPAIR OF THE ORBITAL FLOOR FRACTURE

Maxillofacial trauma can result in fractures of the orbital floor, roof, medial wall, or lateral wall. Orbital floor fractures are common sequelae of orbital trauma due to the floor's inherent weakness.[63] Conventional orbital floor

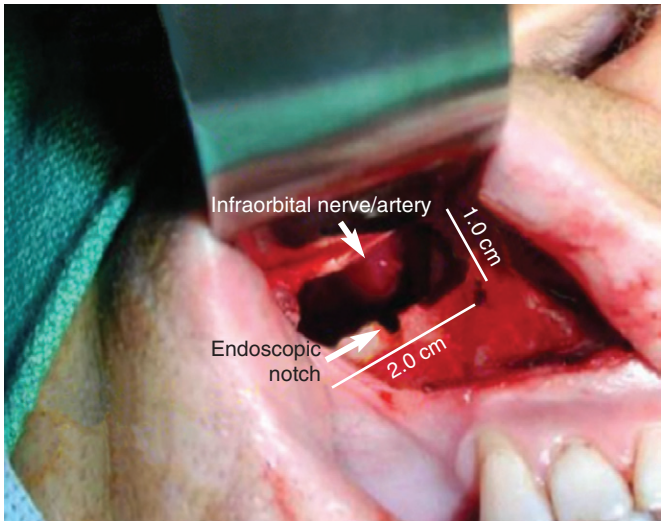


FIGURE 34-17 Caldwell-Luc osteotomy for endoscopic orbital floor access. (Courtesy R. Mueller.)

fracture repair involves access via the infraorbital, subciliary, or transconjunctival incision, with or without a lateral canthotomy with inferior cantholysis.[64,65] These approaches are relatively safe, with a 5% mean complication rate.[66-68] Possible complications associated with these incisions include visible scar formation, ectropion, entropion, lower lid retraction, prolonged lower lid edema, granuloma formation along the incision line, and iatrogenic injury to the lacrimal system and/or globe. Surgeons have developed minimally invasive techniques with endoscopy to help reduce the morbidity of orbital floor fracture repair.[69-72] An intraoral transantral approach is used, which eliminates many of the risks seen in the conventional open approach.

Indications for orbital floor fracture repair include entrapment, enophthalmos, or a large floor defect (>50%). The indications for endoscopic orbital floor fracture repair are similar, but should only be attempted by surgeons experienced in the techniques.

SURGICAL TECHNIQUE

A standard 3-cm maxillary vestibular incision is made 5 mm above the mucogingival junction and a subperiosteal dissection is performed, thus exposing the anterior sinus wall. The infraorbital nerve is identified and protected. A Caldwell-Luc osteotomy window is then made in the canine fossa region, being careful to avoid the tooth roots. The window should be 1 cm in height and 2 cm in length. This can be done with a piezo electric drill or the osteotomy should be designed to accommodate the fracture. The bony window should be removed and placed in saline. An endoscopic notch should also be created at the inferior horizontal osteotomy to allow for stability when navigating the endoscope (Fig. 34-17). A 4-mm, 30-degree Hopkins endoscope (Karl Storz, Carver City, Calif) is then used to inspect the maxillary sinus, which is the optical cavity. The maxillary sinus lining must be carefully removed, being sure to remove all of the Schneiderian membrane without injuring the ostium. Once this is accomplished, an unobstructed view of the orbital floor is possible and the location, size, and

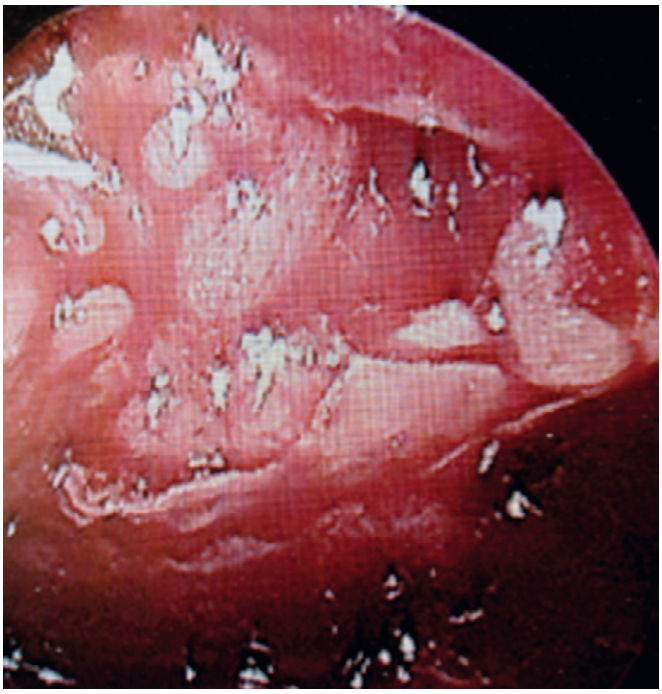


FIGURE 34-18 Endoscopic view of the orbit from below, showing comminuted bone fragments and periorbital contents. (Courtesy Michael Miloro.)

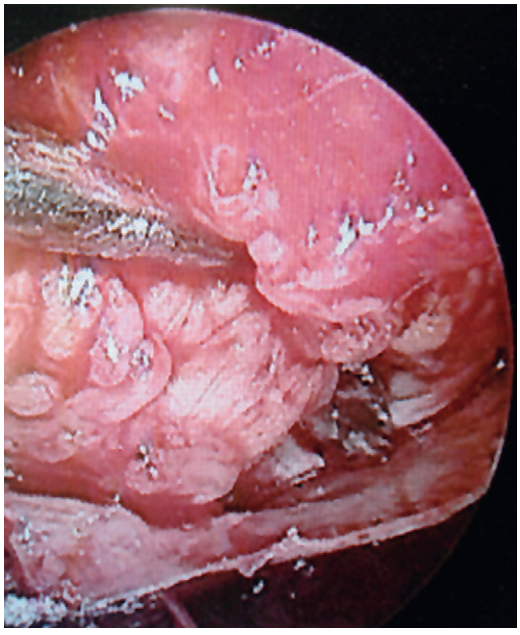


FIGURE 34-19 Endoscopic view of the orbit from below, after the bone fragments have been removed. (Courtesy Michael Miloro.)

severity of the defect can be appreciated. With a trapdoor fracture of the medial or lateral portion of the floor, it may be possible to see that all the orbital contents are reduced back into the orbit. A Medpor implant may then be used on the sinus side to reduce the floor. The fracture is often comminuted and this may not be possible (Fig. 34-18). In this case, removal of all small comminuted bony pieces must be done endoscopically (Fig. 34-19). After removal of all bony fragments, a retractor

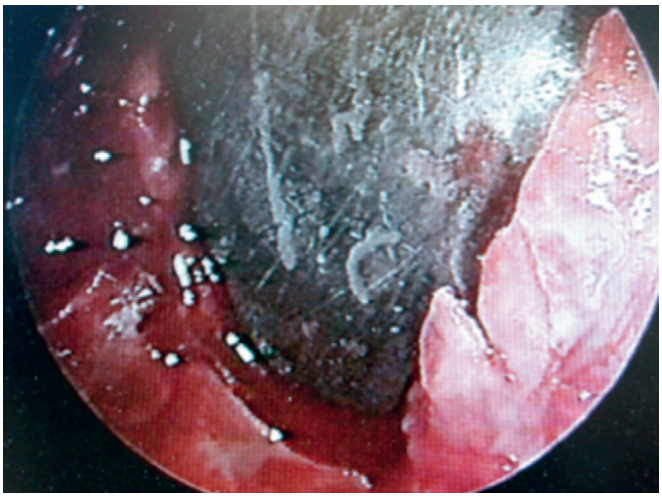


FIGURE 34-20 Endoscopic view of the orbital floor contents retracted into the orbital cavity showing the residual floor defect. (Courtesy Michael Miloro.)

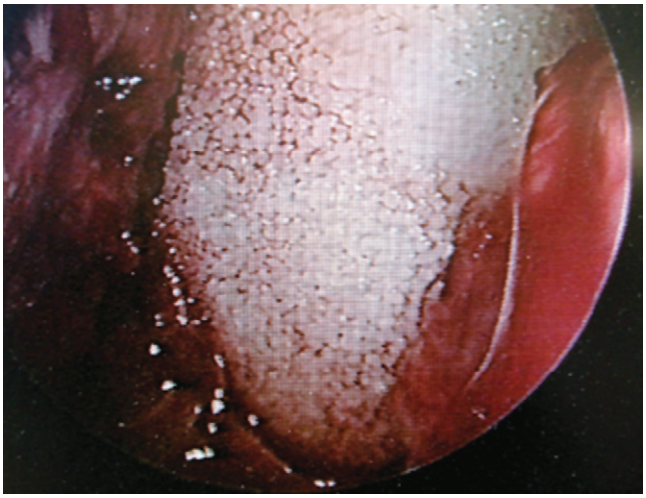


FIGURE 34-21 Endoscopic view of placement of the implant to the orbital floor defect. (Courtesy Michael Miloro.)

is used to elevate the periorbital contents into the orbital cavity (Fig. 34-20). Then, a Medpor implant can be used to replace the floor defect (Fig. 34-21). An implant of 0.85- to 1.0-mm thickness should be used to provide some flexibility in placement. Titanium mesh should be avoided because the sharp edges can often cause trauma to the surrounding soft tissue. The Medpor implant should be shaped and trimmed to be 1 to 2 mm larger in all dimensions of the floor defect so it can sit on the bony edges of the fracture. The implant is placed through the bony window (this may need to be enlarged to accommodate the implant) and placed in the defect in an exchange-like maneuver with the malleable retractor. No fixation screws are needed. A forced duction test should always be performed to rule out entrapment.

SUMMARY

Endoscopic orbital floor fracture repair has the advantages of decreased morbidity for the patient. No periorbital incision is necessary, thus eliminating associated complications, such as ectropion or scarring. Nevertheless, there is a learning curve required to master this technique. The technique is dependent on a well-trained surgeon. As more surgeons become familiar with the technique, the potential for further development and modification improves, and hopefully endoscopic repair of orbital fractures will become routine at most centers.

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